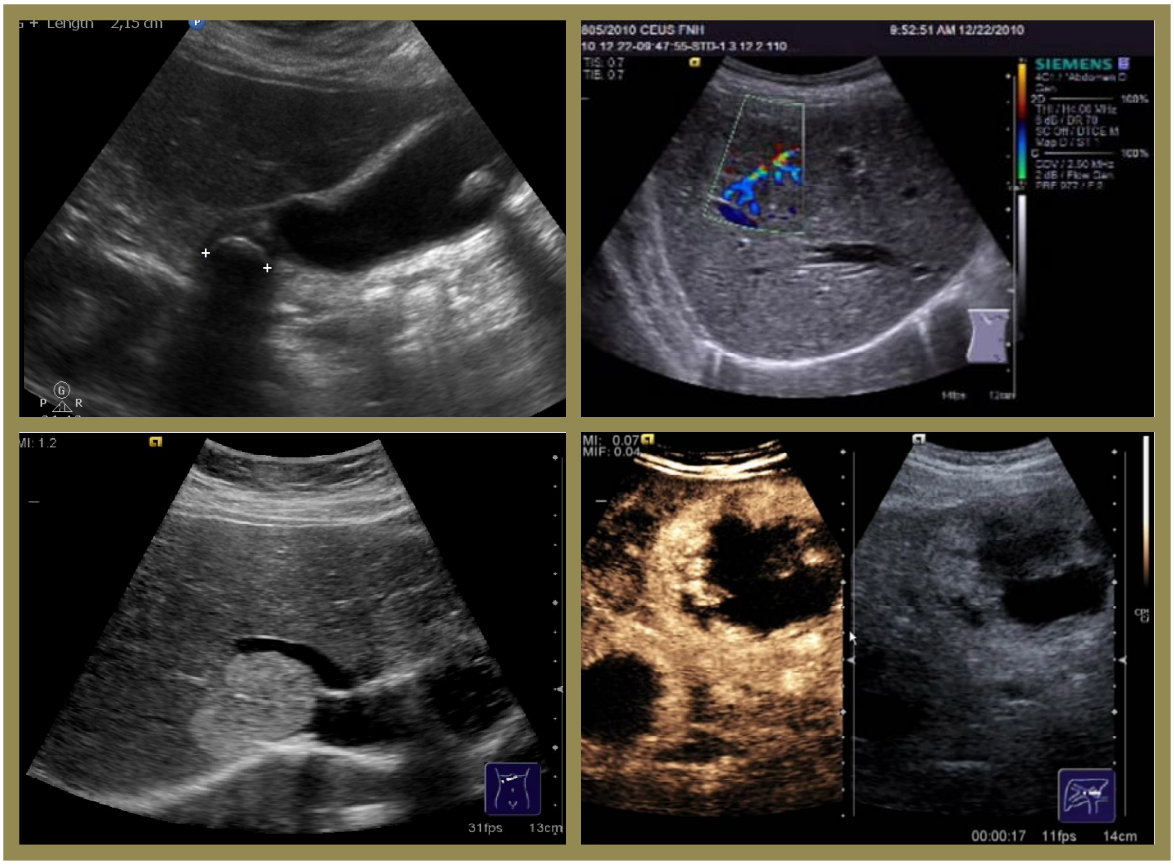
ROXANA ŞIRLI

IOAN SPOREA



COURSE OF
ABDOMINAL ULTRASOUND
FOR STUDENTS

EDITURA VICTOR BABEŞ TIMIŞOARA

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DEPARTMENT OF GASTROENTEROLOGY AND HEPATOLOGY

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COURSE OF
ABDOMINAL ULTRASOUND
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Preface

I have read with great interest and pleasure the manuscript ***"Course of Abdominal Ultrasound for students",*** written by Roxana Şirli and Ioan Sporea, two well known authors in the gastroenterology and internal medicine school from Timisoara, recognized experts in diagnostic imaging evaluation but also in interventional procedures guided by the different methods applied in the gastroenterology clinic, clinic supervised by Professor Ioan Sporea. The manuscript has all the characteristics of a real monograph, by all the aspects taken into account, in the imaging related to anatomy, physiology and pathology of abdominal organs visualized by echocardiographic imaging technique, not just the ones evaluated in gastroenterology. By all the details, the relation of imaging technique to the clinical part and the therapy of abdominal organs disease, the book represents a real manual, useful not only in medical students formation (as the title suggests), but also for a large medical group – fellows and specialists in internal medicine, Imagistics and radiology, urology, general surgery, as well as general practicians, often practicing the technique following not quite accurate and actual standards. The presentation and the language are of great clarity and efficacy, with a clear teaching, with scientific value character but also directed towards practical experience, useful, without a doubt in such an attempt.

Of course, the quality of echocardiographic imaging is exquisite, not just given by the image resolution and details, but also by the specificity of presented aspects, all resulted after large volume personal experience and performance. Actual and relevant data as well as the experience presented is certified not just by a simple "belletristic" and scientific reading of the book, but by a well certified experience in formation of many generations of practicians in abdominal echocardiography, certified by Professor Ioan Sporea and the team of Gastroenterology Clinic, that he is coordinating, a team in which Dr. Roxana Sirli is recognized as a high quality expert in that field. There isn't, under any circumstances, the case of a publishing opportunism, but an exquisite attempt in teaching valuable information of great quality and actuality, and most important, coming from the experts most authorized in a very specific field.

Taking all this into consideration, I propose to the "Victor Babes" publishing house, the publishing of the book in form submitted by the authors.

Professor LUCIAN PETRESCU

Contents

Chapter 1.

Introduction in ultrasound; Contrast enhanced Ultrasound

Abdominal ultrasound is one of the most accessible imaging methods used in daily practice. In modern medicine it should be considered an extension of the clinical examination, and is helpful in both emergency and for the initial evaluation and follow up of patients with various abdominal symptoms, in patients with chronic liver diseases, in oncology patients, in those with mild abdominal trauma etc.

In our opinion, abdominal ultrasound is the logical approach that should follow history and physical examination of patients with abdominal complaints. Imagine transducer as a flashlight that will light the way and that will allow you to view intraabdominal organs. It is a valuable method, it is accessible, non-invasive, non-irradiant, inexpensive, repetitive. But in addition it must be remembered that it is operator dependent and that the ultrasound window is not always what we want, that the examination can be difficult in obese patients, in those who cannot collaborate with a deep inspiration (that facilitates evaluation), in those who cannot be mobilized, or in patients with flatulence.

Ultrasound image is formed by the reflection of ultrasonic waves emitted by the transducer by tissues. Reflected waves are captured by the transducer and then processed electronically, the resulting ultrasound images are projected on the monitor. Reflection of ultrasound is dependent of the impedance of tissue (tissue resistance to the passage of the sound waves). The denser the tissue is, the stronger it will reflect the ultrasound at the interface between the constituent structures.

The echo-texture of the normal liver is considered to be normoechoic (gray, like a fine blend of salt and pepper). What is whiter than normal liver is considered to be hyperechoic, what is darker - hypoechoic. Fluid structures do not reflect ultrasound and appear completely black, being labeled as anechoic or transonic (gall bladder, urinary bladder, vessels, ascites, pleural effusion, etc.). Structures that reflect the majority of ultrasound waves will appear white and will generate posterior shadow (bones, stones, calcifications). Taking into account that ultrasound is almost completely reflected at the interface between air and other medium, air will appear as intense hyperechoic, similar to stones or bone.

After this technique preamble, it should be noted that to make a quality ultrasound examination we should ensure to have always optimal examination conditions: in a dark room, with sufficient time for examination, with access to complete clinical information about the patient. It is always useful to follow an examination protocol, focusing on the area of interest.

The sections (incidences) used for the ultrasound examination are: longitudinal, sagital or axial (parallel to the spine); transverse (perpendicular to the spine); and oblique sections. The examination must be dynamic, by scanning the examined organs in multiple planes, changing the incidence so that we don't "miss" lesions in areas difficult to visualize. When performing ultrasound examination we should always consider the representation of organs in space, their anatomical reports. For example, with a high transverse section through the epigastria, the following organs can be viewed (from front to back): abdominal wall, left hepatic lobe, gastric antrum, pancreas, spleno-portal axis, the large vessels (aorta and inferior vena cava), and spine.

Before one can establish a diagnosis by ultrasound one must know very well the normal aspect of various organs. Thus the first chapter of this course will address to the fundamentals of ultrasound anatomy. To visualize various organs, vessels are important anatomical landmarks. Figure 1 is meant to be a memory refresher, where 15=aorta; 32=celiac trunk; 18=hepatic artery, 19=splenic artery, ** = left gastric artery, 17=superior mesenteric artery, 24=renal arteries, 16=inferior cava vein, 10=hepatic veins, 25=renal vein, 20=splenic vein, * = superior mesenteric vein, 12=portal vein, 11=portal bifurcation, 66=main biliary duct, 14=gallbladder.

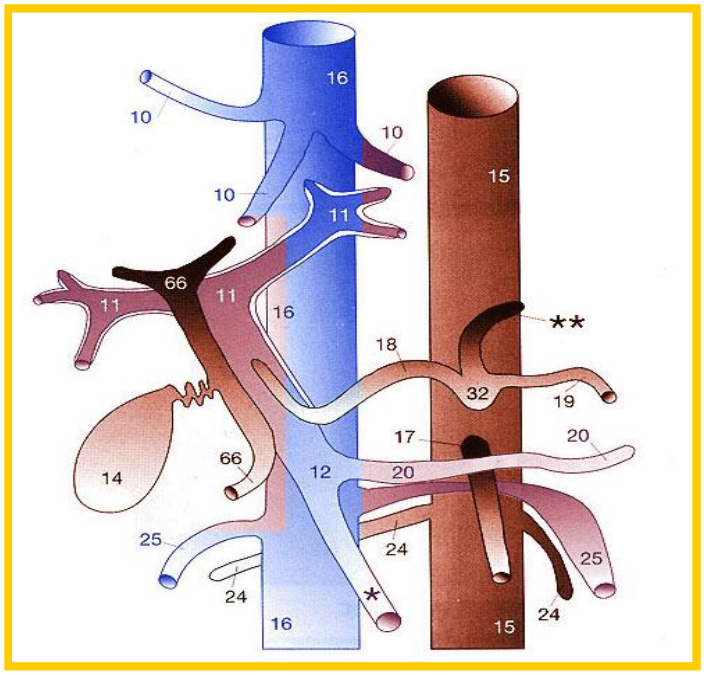


Fig. 1. Anatomical vascular landmarks (Hofer M. – Ultrasound Teaching Manual, Thieme, 1999)

CONTRAST-ENHANCED ULTRASOUND

Abdominal ultrasound is an imaging method widespread in medical practice, but unfortunately it was "handicapped" by the fact that, unlike in other imaging methods, contrast could not be used. In the recent years ultrasound has undergone a real effervescency due to the appearance and more frequent use of ultrasound contrast agents (contrast-enhanced ultrasound: CEUS).

In CEUS the ultrasound signal is amplified with the help of microbubbles, an effect discovered by a cardiologist (Joyner Claude), who observed that the ultrasonic signal was amplified in M mode, after injecting an iodine contrast agent for angiographic studies of the heart. The first clinical application of microbubbles was also performed in cardiology, when, after the intravenous injection of a mixture of saline with air, right-left intracardiac shunts were evaluated.

Then ***the first generation of ultrasound contrast agents*** soon followed (Echovist, then Levovist) consisting of disaccharide coated microbubbles containing air, mainly used to evaluate cardiac and peripheral vessels. The examination was done with ultrasound waves with high mechanical index (intensity) level, which made the microbubbles to be rapidly destroyed, thus amplifying the ultrasound signal. Levovist is taken up by the reticulo-endothelial system in the spleen and liver amplifying the ultrasound image of these organs, which lasts a few minutes, so it can be used to highlight isoechoic liver metastases (that contain no Kupffer cells), that could not be detected by standard ultrasound.

The second generation of ultrasound contrast agents (SonoVue) do not amplify the ultrasonic signal through microbubble destruction, but through microbubble resonance in the ultrasound field. The examination is made with low mechanical index (<0.4), which makes the microbubbles oscillate, not destroyed. The amplification time of the ultrasound signal is up to 4-6 minutes. Characteristic for second generation contrast agents is the microbubbles' elastic response to compression and relaxation. This oscillation will generate an asymmetric, non-linear signal. This response is different from the signal generated by the examined tissue, thus permitting its' separation by the vascular structures.

The SonoVue microbubbles are formed of a phospholipid shell that includes hexafluoride sulfur, a biologically inert gas. The microbubbles' diameter varies between 1 and 10 microns (with an average of 2.3 microns), comparable in size to red blood cells. The microbubbles cannot cross the vascular wall, thus SonoVue is strictly an intravascular contrast agent (as opposed to contrast agents in CT and MRI that diffuse into the interstitium). Five to six minutes after injection, the microbubbles are destroyed and the inert gas is released and is cleared through exhalation (not through the kidney as CT or MRI contrast agents), therefore it is not contraindicated in patients with renal failure.

SonoVue microbubbles have a specific behavior in the ultrasound field, which derives from their high compressibility, in contrast to the surrounding tissue, virtually non-compressible (molecules move with only a few Angstroms in the ultrasound field). During a normal examination, the microbubbles' diameter may vary from half to twice the original diameter.

The microbubbles have a natural frequency of oscillation (resonance) dependent on their diameter, which shows the highest energy conversion efficiency of the ultrasound in reflected signals, useful for obtaining the ultrasound image. The resonance frequency of 3-5 microns microbubbles is in the frequency range usually used for ultrasound examination (3-5 MHz).

At low intensity ultrasound examination (low mechanical index) the microbubbles response is non-linear because their diameter changes asymmetrically as compared to the equilibrium size. This is because the energy required to compress the microbubbles is greater than that consumed for their expansion (microbubbles are becoming harder the lower the volume). Consequently, the signal obtained through their oscillation will be a distorted version of the insonation wave, effect known as non-linear response which is manifested by harmonic oscillations of the insonation frequency, visible in the spectrum of signals received by the transducer.

But not only microbubbles cause the appearance of harmonics, but also the examined tissues, the effect being more obvious when the insonation signal strength (mechanical index) is higher. In conventional B mode examination, tissue harmonics are used to reduce artifacts caused by reverberations, but in contrast mode (with low mechanical index) they will only contaminate the image, appearing as "noise". Examination with lower mechanical index, used in second generation contrast ultrasound agents studies, in addition to the fact that they generate less tissue harmonics, it also has the advantage of slowly destroying microbubbles, allowing real-time examination.

The safety profile of CEUS

To be used in clinical practice, any medical product must have a good safety profile.

For SonoVue, the only ultrasound contrast product used in Europe at the moment, the most important data on the safety profile originates from an Italian multicenter retrospective study (29 centers), that included a total a number of 23188 patients during a three years period (2001-2004). In this study there were no deaths in connection with contrast ultrasound examination, and the number of reported adverse reactions was 27: 23 of them minor, three moderate and only one severe. In this study, the total rate of adverse events was 0.0086%. In the safety studies published since the marketing study, deaths have been reported after administration of SonoVue, but only in patients with severe heart disease, recent myocardial infarction, the demise probably being related to the heart disease and not to SonoVue.

Based on these data we conclude that SonoVue is a medical product with a good safety profile, which can be used in most patients requiring this investigation, except in patients with acute myocardial infarction, severe ischemic heart disease or other severe cardiac diseases.

Characterization of focal liver lesions in contrast ultrasound (CEUS)

The principle of contrast ultrasound examination in the liver is based on the double blood supply of the liver (venous – from the portal vein and arterial – from the hepatic artery). The liver lesions should be examined after bolus injection of the contrast agent in all three vascular phases (arterial, portal, and late - parenchymal), thus allowing their characterization, increasing the method's sensitivity for a correct diagnosis.

Thus, 10-20 seconds after the contrast injection into an antecubital vein, it reaches the liver via the hepatic artery, the arterial phase lasting until the start of the portal (venous) phase, 30-45 seconds after the contrast injection. In the portal phase, most of the contrast agent reaches the liver through the portal vein. The portal phase lasts up to approx. 2 minutes, when the late phase starts (the balance phase), which lasts until the disappearance of the microbubbles from the circulation, about 4-5, maximum 6 minutes (Table 1).

Table I. Vascular phases in CEUS

TIMES	**START**	**END**
ARTERIAL PHASE	10-20 s	25-35 s
VENOUS PHASE	30-45 s	120 s
PARENCHYMAL PHASE	120s	until the disappearance of the microbubbles from the tissue

Depending on their nature, focal liver lesions have a typical behavior following contrast, so it is possible to characterize them. Most important is the differentiation between malignant and benign lesions, which CEUS does very well. Characteristic for malignant lesions is the fact that the contrast does not persist in the lesion during the late phase, and the wash-out phenomenon occurs (Fig. 1). The enhancement pattern of each type of hepatic focal lesion during CEUS will be detailed in the focal liver lesions chapters.

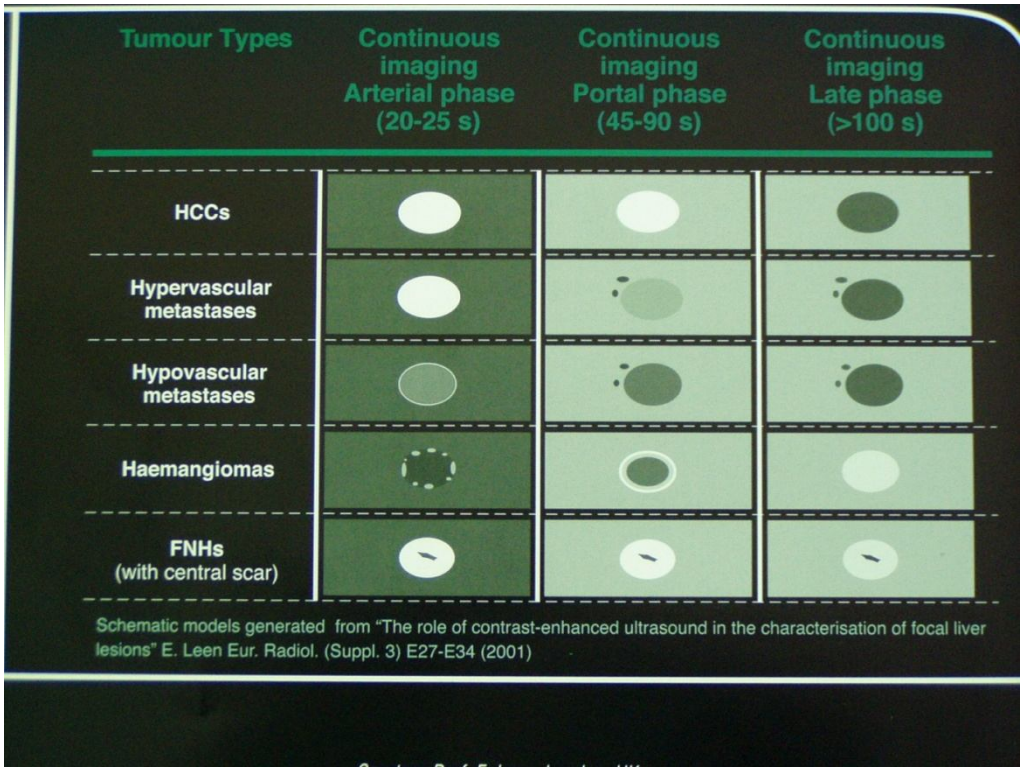


Fig. 1. The focal liver lesions enhancement pattern in CEUS

Chapter 2.

Elements of ultrasound anatomy

1. The Liver

The liver is a parenchymal organ with typical appearance, crossed by vascular structures. The normal liver is considered to be normoechoic (as a fine blend of salt and pepper) (Fig. 2). The examination begins with the patient in supine position and continues in left lateral decubitus. For a good ultrasound "window" we usually require the patient to perform a deep inspiration, to maintain it for a few seconds while the examiner scans the liver structure. A convex transducer with variable frequency of 2-5 MHz is generally used, the frequency is chosen according to the examined subject characteristics (lower frequency for better penetration). If one is interested in details of liver surface or in superficial areas of the liver, linear transducers with higher frequency (4-8 MHz) should be used.

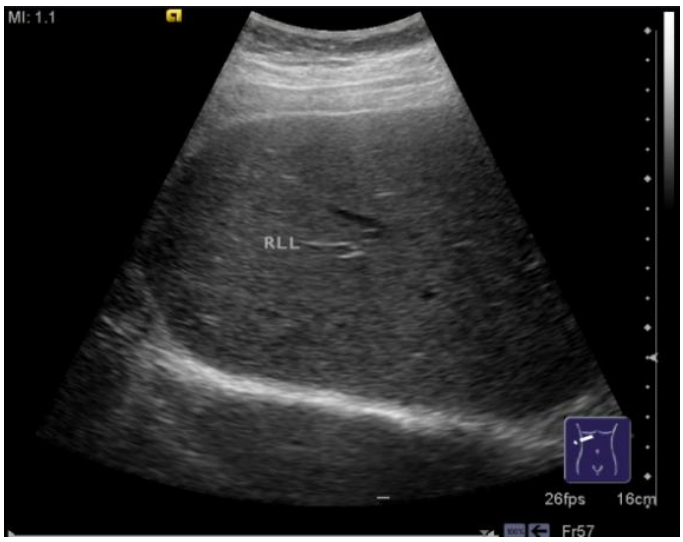


Fig. 2. Normal liver

For the examination of the liver, sagital sections, transverse and oblique sections, right sub-costal and also intercostal sections are used. The homogeneity and texture of the liver structure, the presence or absence of circumscribed lesions, the aspect of liver surface, the vessels' patency (portal vein and hepatic veins) should be noted. Liver echogenity is assessed by comparison with the right kidney cortex, to which it should be similar.

The left hepatic lobe is examined in recurrent oblique sub-costal sections, starting from the epigastria, by scanning from bottom to top, to cover all its volume. By moving the transducer to the right, by the same movements, the right hepatic lobe is examined.

Also by oblique sub-costal sections the portal bifurcation will be examined (the right and left branches of the portal vein) and, in a higher plane, the hepatic veins and their confluence with the inferior vena cava is seen.

The left hepatic lobe and the caudate lobe (located before the inferior vena cava) are examined in sagital section, starting from the epigastria. It is recommended to measure the antero-posterior diameter of caudate lobe, since it is increased in patients with cirrhosis (> 35 mm) (Fig. 3). Scanning from the epigastria to the right we evaluate the entire liver by sagital sections, finding along the way the gallbladder.

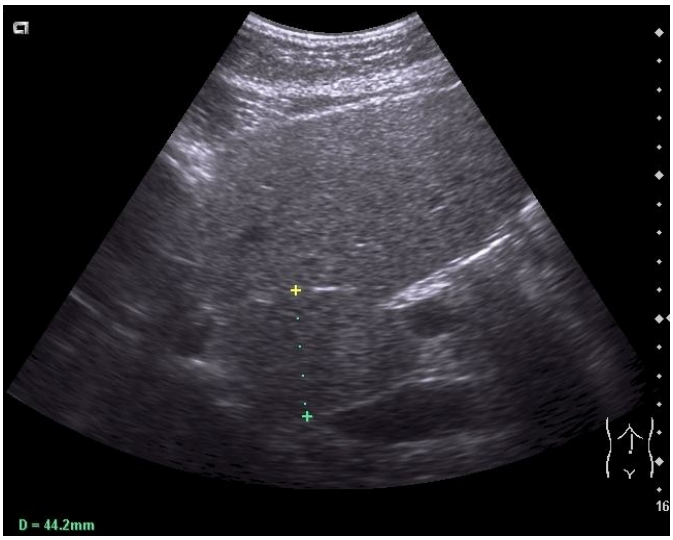


Fig. 3. Increased caudate lobe in a patient with liver cirrhosis

In addition to sub-costal oblique and sagital sections it is recommended to use intercostal incidences for the evaluation of the liver dome, especially in less cooperative patients who cannot perform a deep inspiration.

During the examination of the liver it is mandatory to evaluate the vascular structures, important elements as anatomical landmarks for segmentation of the liver, but may also present modifications suggestive for certain diseases.

The hepatic veins (HV) are anechoic structures with thin, hyperechoic wall. There are three hepatic veins: right HV, middle HV and left HV that converge to the inferior cava vein similar to the fingers that converge to the palm of the hand (Fig. 4). They are examined through high sub-costal oblique sections. When the lumen is occupied by echo-dense material without Doppler signal (intravascular thrombus), the appearance is characteristic for Budd-Chiari syndrome. When they are dilated to more than 10 mm (measurement performed at 2 cm from their convergence in the inferior vena cava), the appearance is suggestive for congestive heart failure (cardiac liver) (Fig. 5).

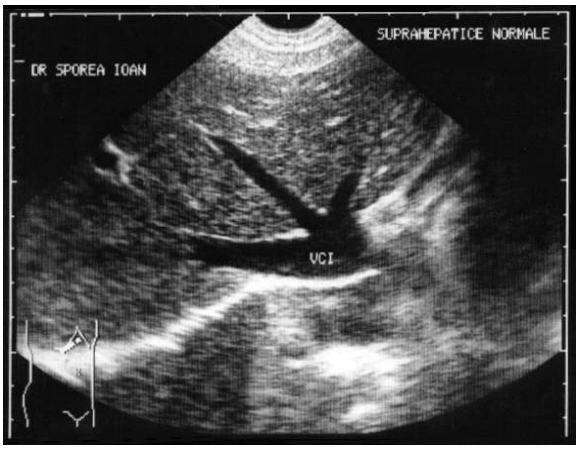


Fig. 4. Normal HV

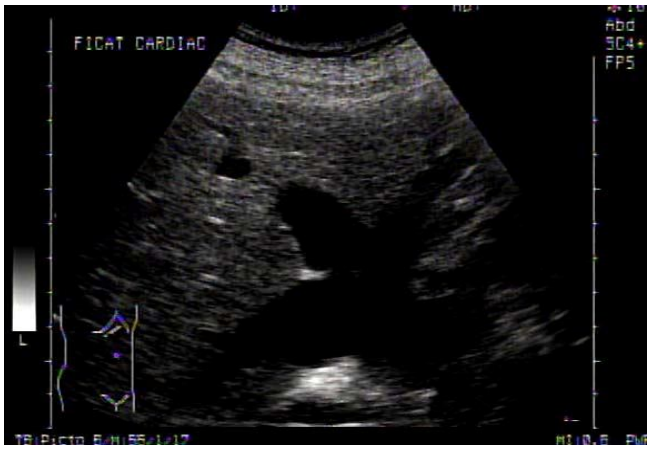


Fig. 5. Dilated HV in cardiac liver

The portal vein (PV) is examined by a perpendicular sub-costal section. It's a transonic structure with hyperechoic wall, thicker than the HV's, located posteriorly to the main biliary duct (MBD) (Fig. 6). Its maximum normal diameter is 13-14 mm, higher values being suggestive for portal hypertension. When the lumen is occupied by echodense material without Doppler signal, the aspect is suggestive for portal thrombosis, whose etiology (benign or malignant) should be established. The portal bifurcation is examined through right oblique sub-costal sections, and is located in a plane below the HV. Similar to PV, the portal branches have thicker walls than the HV (Fig. 7).

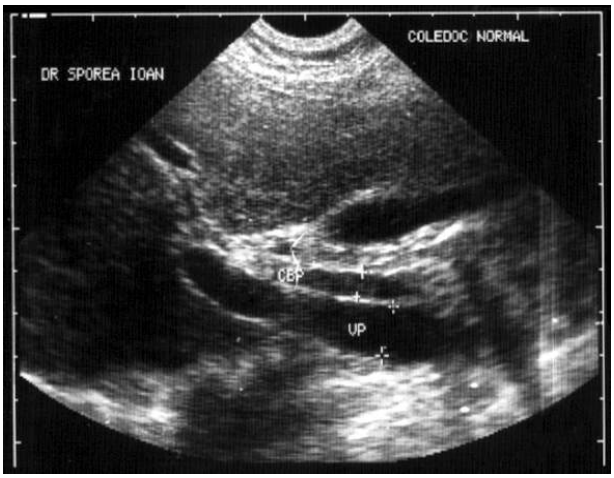


Fig. 6. Common portal vein (PV) and main biliary duct (MBD) in hepatic hilum

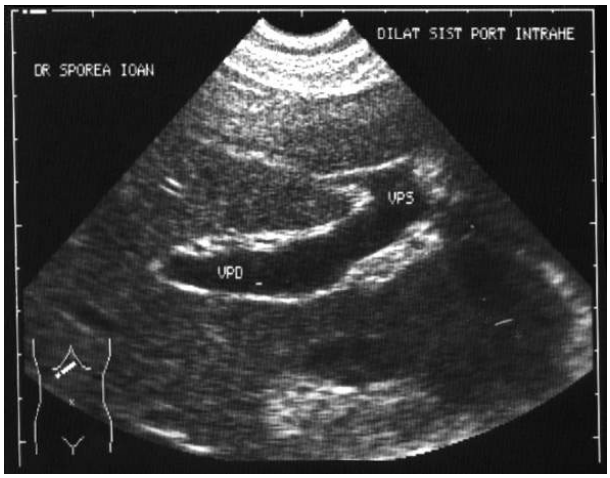


Fig. 7. Portal bifurcation

Starting from the liver vessels, a functional segmentation of the liver was imagined. Anatomically, the liver is divided into two lobes, the right and the left lobe, separated by the hepato-duodenal ligament (Fig. 8).

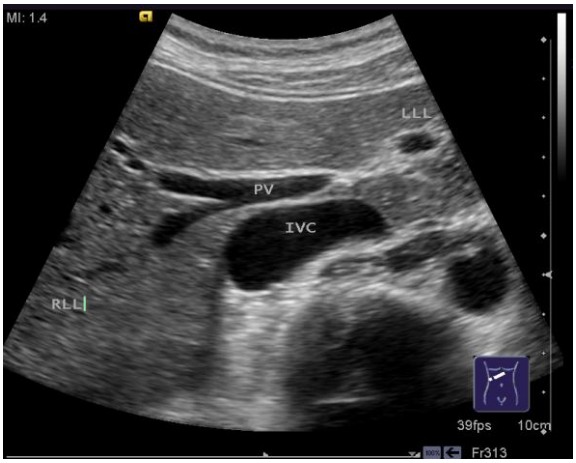


Fig. 8. Liver segmentation. LLL=Left liver lobe; RLL=Right liver lobe; PV=Portal vein; IVC= Inferior cava vein

The functional segmentation (imagined by Couinaud) allows the definition of eight liver segments considering three vertical planes passing through the three hepatic veins, and a horizontal plane passing through the portal bifurcation, which separates the upper segments of the liver form the lower ones (Fig. 9). The caudate lobe (segment I) is considered a separate structure from the two lobes, delimited posteriorly by the inferior vena cava and anteriorly by the venous ligament. The left liver lobe includes segment II (superiorly) and segment III (inferiorly) and is delimited by the right liver lobe by the plane passing through the left HV. Segment IV is located between the left HV and the middle HV. Between the middle and the right HV are segments V (inferiorly) and VIII (superiorly). Lateral to the right HV are the posterior segments of RLL, VII superiorly and VI (inferiorly).

Some practical observations:

- in the RLL, segments VII and VIII are in contact with the diaphragm;
- segment VI comes into contact with the right kidney;
- the caudate lobe is examined in sagital section;
- by cross section through the gallbladder cervix, the gallbladder bed is surrounded by the segments IV, V and VI;
- by cross section in the upper epigastria, segments IV, VII and VIII converge to the inferior vena cava.

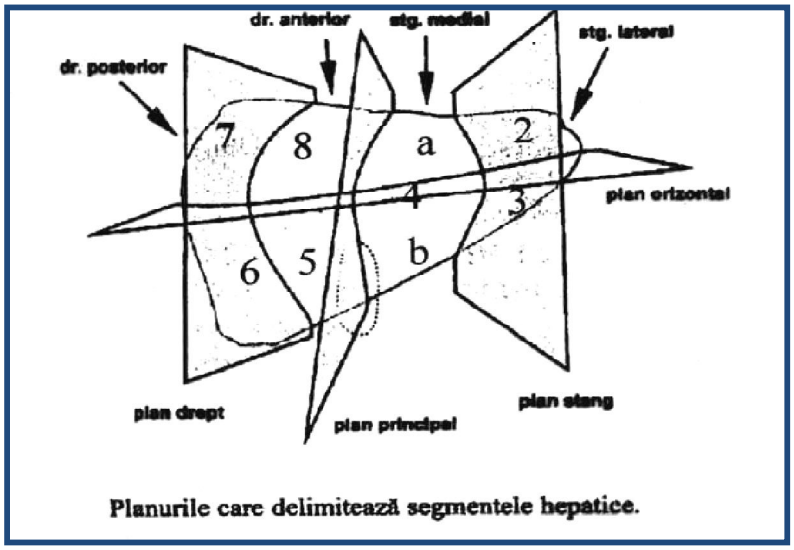


Fig. 9. Liver segmentation

The main biliary duct (MBD) is examined in a section perpendicular to the costal margin, and is located in front of the PV (Fig. 6). Its maximum normal diameter is 5-6 mm, while in patients with cholecistectomy a normal value up to 7-8 mm is accepted. MBD, PV and hepatic artery (HA) are the constitutive elements of the hepatic hilum. HA intersects at a point the MBD and the PV, passing between them. The MBD and PV have the same trajectory, appearing as a "double-barreled shotgun", but the “barrels” are unequal, the thinner one, situated anteriorly, is the MBD. When the ratio between the diameter of the PV and the MBD is inverted, the aspect is diagnostic for obstructive jaundice.

The intrahepatic bile ducts normally aren’t seen in standard ultrasound, since they have a very fine caliber. They will become evident when there is an obstacle downstream, appearing as transonic structures parallel with the branches of the portal vein, realizing the appearance of "double duct". If dilatations are important in the incidence that visualizes the portal bifurcation the aspect will be of a "spider" (Fig. 10).

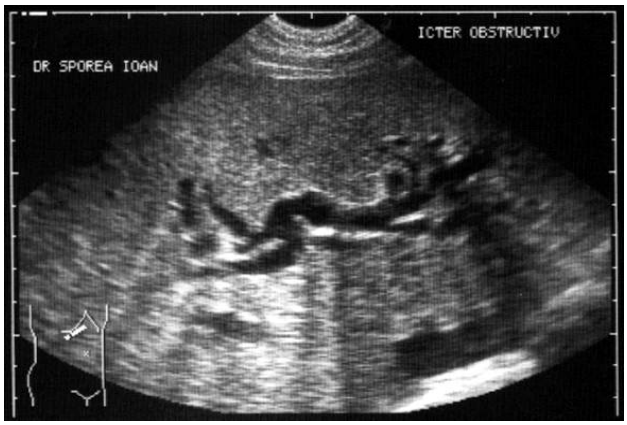


Fig. 10. Dilatations of intrahepatic bile ducts –"spider" aspect

2. Gallbladder

The gall bladder is the source of many abdominal complaints, and is an organ which can easily be examined by ultrasound. Examination is done by right sub-costal recurrent oblique sections; through sagital right sub-costal or by intercostal sections; in supine and mandatory in left lateral decubitus position. The examination must be made carefully, with full view of gallbladder, with special attention to the infundiblar area, where gallstones can hide. By turning the patient in the left lateral decubitus, the infundiblar area will become more accessible and possible stones can mobilize, falling by gravity to the bottom of the gallbladder, where they are better visualized.

The normal appearance of the gallbladder is of a pear shaped, anechoic structure with well-defined hyperechoic wall (Fig. 11). The normal diameters are generally smaller than 8/3 cm, the maximum accepted ones are 10/4 cm, and higher values are suggestive for hydrops. The normal gallbladder wall thickness is maximum 4 mm. Following food ingestion, the gallbladder wall appears duplicated, due to smooth muscle contraction (Fig. 12).

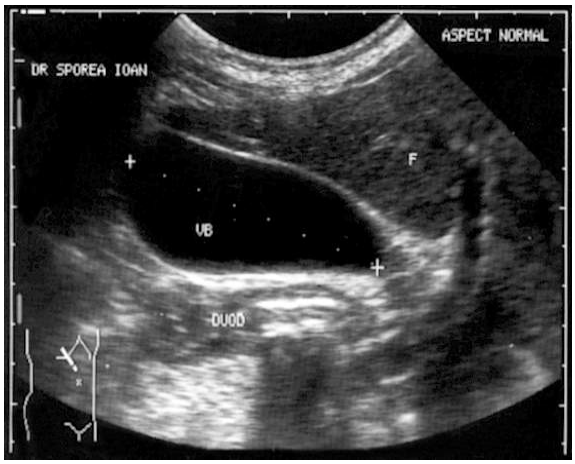


Fig. 11. Normal gallblader

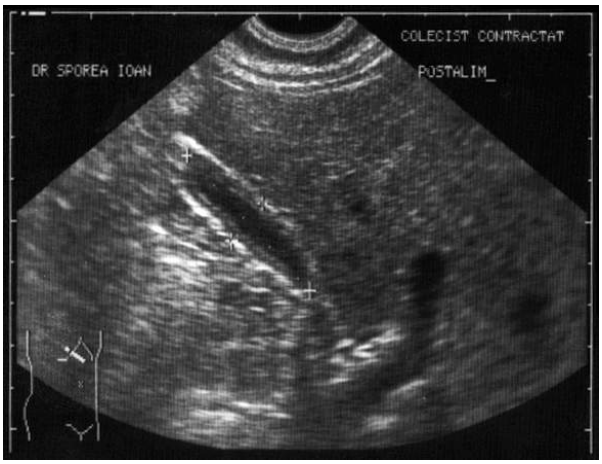


Fig. 12. Gallblader contracted postprandially

3. Pancreas

Pancreatic ultrasound is “the corner-stone” of ultrasound examination, especially for a beginner in ultrasound. But patience and perseverance will lead to increasingly easier visualization of this organ. The examination difficulties come from the fact that the pancreas is a retroperitoneal deeply located organ, partially masked by the bowel loops, the gas contained in the intestinal loops working as a screen that prevents the penetration of ultrasound. Examination begins from the epigastria, with mild, progressive compression that can mobilize and remove of the intestinal content, thus optimizing the acoustic window.

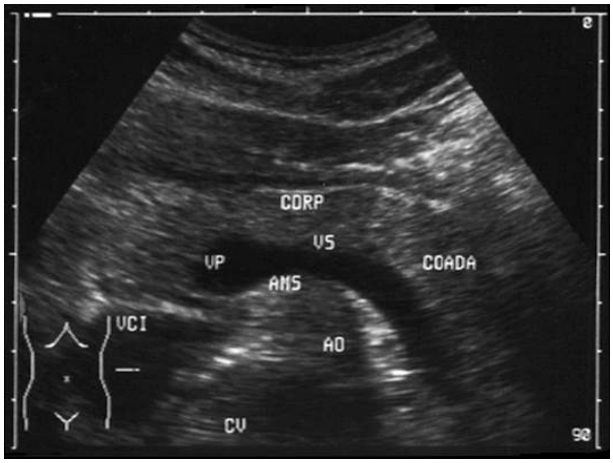
Before the examination, the ultrasonographist must be well acquainted with the local anatomy, with the vascular landmarks that will help him delineate the pancreas. The examination is done mainly through epigastric sections. The 3.5 MHz convex transducer is preferred. Rarely, in thin (or cachectic) people, a 5 MHz linear transducer is needed.

It is mandatory to examine the pancreas in a fasting patient. The presence of food in the stomach may prevent a correct examination or may create false pancreatic tumor images. The fasting period is 7-8 hours. Liquid consumption is allowed, but carbonated drinks are contraindicated (air in the stomach will make pancreatic examination difficult).

The pancreas will be examined through the gastric antrum or, if the transducer is placed at a high level in the epigastria, using the ultrasound window of the liver, or more rarely, below the antrum (the position of the transducer is midway between the xiphoid appendix and the umbilicus). The best ultrasound window for pancreatic examination will be obtained by high sections (avoiding the colon), through the left hepatic lobe or trans-gastric. For trans-gastric pancreatic examination, the antrum should contain no air.

The presence of liquid in the stomach plays the role of an ultrasound window for the examination of the pancreas. Hence the practical approach used in cases of difficult visualization of the pancreas, when the patient will be administered 500-700 ml plain water or apple juice that will form an ultrasound window in the stomach. After ingestion, 10-15 minutes are required for the debubbling of the ingested liquid. If examination is performed immediately after water ingestion, a hypoechoic (not anechoic) appearance of the stomach will be seen, which might be a surprise. This appearance is the consequence of the bubbling air in the water during deglutition. After 10-15 minutes, the stomach will be filled with transonic liquid that will act as an acoustic window for a better visualization of the pancreas. Sometimes it is possible to find no water in the stomach if the patient is in dorsal decubitus. In this case, the patient will be placed in a sitting position, so that water accumulates in the antrum, which is the ideal anterior landmark of the pancreas.

In order to examine the pancreas through a transverse epigastric section, firstly the spleno-portal axis (the portal vein and the splenic vein) should be identified. It delimits the pancreas posteriorly and appears like a transonic image, comma-shaped, situated anteriorly of the spine, aorta and inferior vena cava. The pancreas is delimited anteriorly by the gastric antrum or the left hepatic lobe (depending on the level at which the transverse section is performed) (Fig 14). Another important vascular reference is the celiac trunk, specifically the pancreatic and hepatic arteries: at their emergence from the celiac trunk they lie on the top of the pancreas. For this reason, when you see the emergence of celiac trunk of the aorta (the appearance of "fountain") (Fig. 15), the transducer must be angled slightly down and the pancreas will appear in the examination plan.



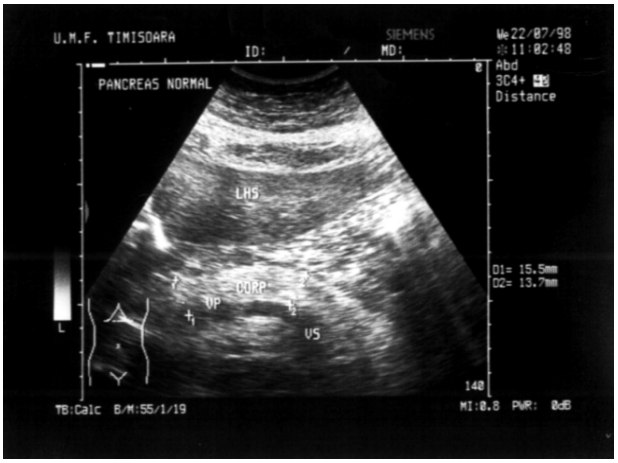


Fig. 13. Normal pancreas

Fig. 14. Normal pancreas, slightly hyperechoic

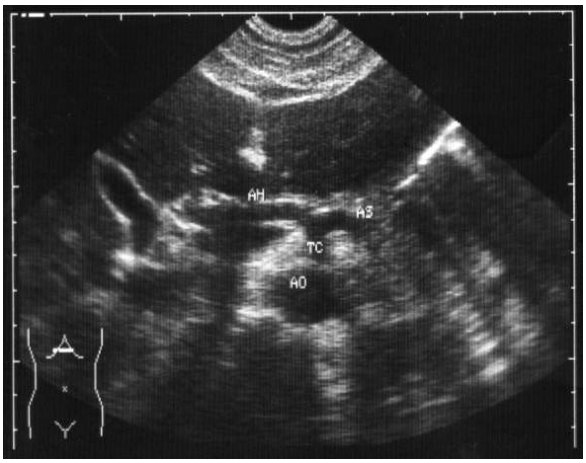


Fig. 15. The emergence of the celiac trunk (TC) from the aorta (AO), with the hepatic artery (HA) and splenic artery (SA)

Between the posterior landmark (the spleno-portal axis) and the anterior landmark (the gastric antrum and the left hepatic lobe) the parenchymal structure of the pancreas is found. The echogenity of the normal pancreatic parenchyma is similar to that of the liver (possibly slightly hypoechoic). In obese patients (due to fatty infiltration) or in elderly patients (fibrosis), the pancreas can by hyperechoic. All these appearances are normal, provided that the structure of the pancreatic parenchyma is fine, homogeneous (Fig. 14). A normal Wirsung duct can be visualized particularly in young persons, with a diameter of up to 2 mm. It is usually seen only along a portion, rarely in its entire length (Fig. 16).

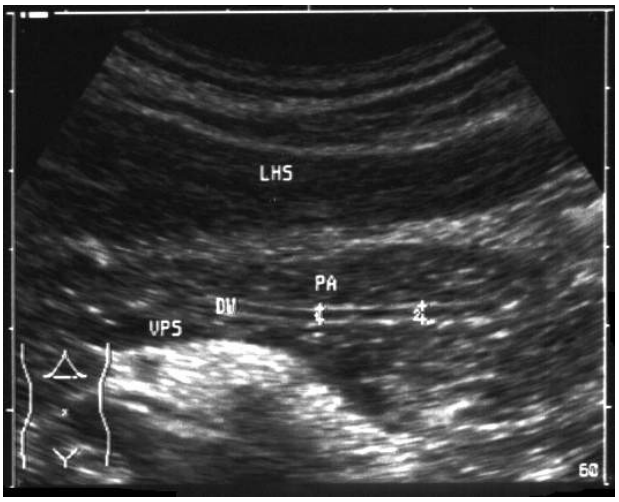


Fig. 16. Normal pancreas with visible, normal Wirsung duct (WD)

Pancreatic examination in transverse section will visualize most of the pancreas, but the entire pancreas is almost never seen in a single section due to its slightly ascending trajectory. Pancreatic tail is harder to examine due to the interposition of the gastric body and is sometimes better visualized in left sub-costal oblique section (Fig. 17).

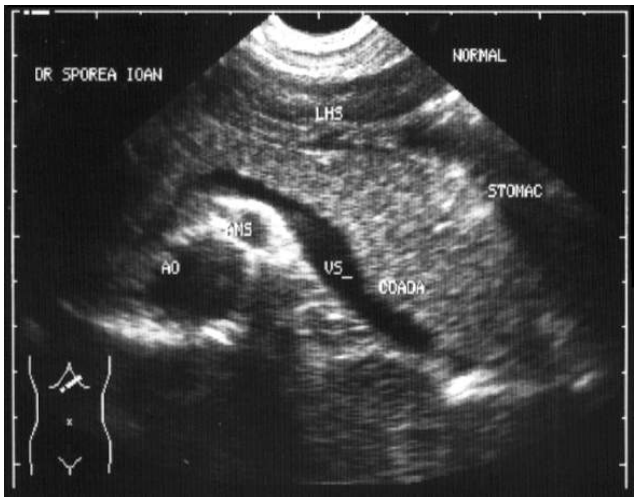


Fig. 17. Pancreatic tail

Regarding the normal size of the pancreas opinions are divided. We do not consider pancreatic size as very important because of its wide individual variability. The easiest to measure is the body of the pancreas, by antero-posterior measurement in transverse epigastric section. Usually, the antero-posterior diameter of the pancreatic body is 10-20 mm, the head of the pancreas is considered normal up to 30 mm and the tail of the pancreas up to 20-25 mm.

4. The Spleen

The spleen is an organ with a parenchymatous structure, with echogenity similar to that of the liver. The ultrasound evaluation of the spleen is performed through left intercostal sections or by oblique sections under the left costal margin in supine position or in right lateral decubitus. The normal spleen is shaped as a crescent, less than 12/6 cm in size (Fig. 18). The entire spleen is relatively difficult to visualize, especially by beginner ultrasonographists. The examination of the spleen will be conducted so as to include both splenic poles in the ultrasound plane, allowing an accurate measurement of the long axis (the most important), as well as of the short axis in globular spleen (more than 6 cm thick). In tall individuals (over 180-190 cm), a larger size, up to 13/7 cm is acceptable. A spleen with larger diameters than those mentioned should raise the suspicion of a liver disease or a blood disorder, but ultrasound cannot differentiate between the two. Sometimes an accessory spleen may be seen. It appears as a round parenchymal structure, 1-2 cm in diameter, located near the splenic hilum (Fig. 19).

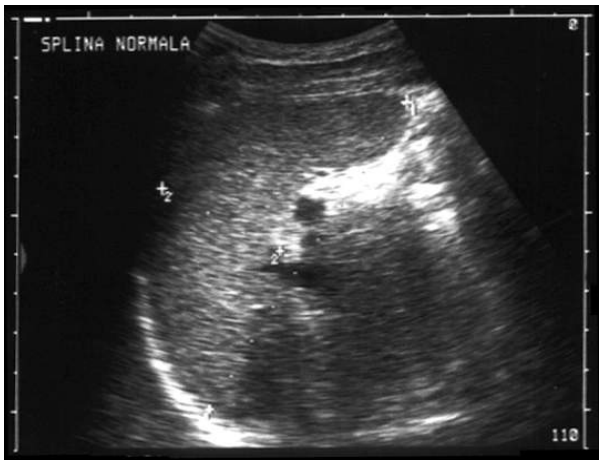


Fig. 18. Normal spleen

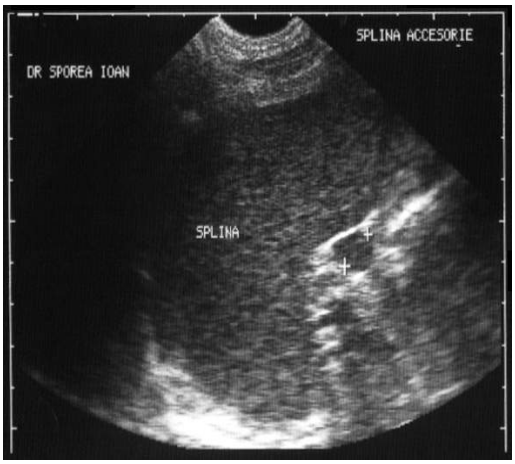


Fig. 19. Accessory spleen

5. The Kidneys

The kidneys are retroperitoneal organs with diameters of 10-12/5-6/3 cm. Renal ultrasound examination is performed with standard 3.5 MHz, preferably convex transducers. The renal ultrasound approach can be through the loins (with the patient in ventral decubitus), by lateral approach (right lateral decubitus for the examination of the left kidney, and scanning is performed through the left lateral abdominal region and left lateral decubitus for the right kidney), or through sagital sections in a patient in dorsal decubitus. Generally, the right kidney is easier to visualize in lateral sections or with the patient in dorsal decubitus, using the liver as an acoustic window. For the left kidney, examination is easier in lateral or dorsal sections. Additional intercostal sections are often used for renal ultrasound examination. For better

visualization, kidneys must be scanned both by longitudinal and transverse sections, until the largest diameter is visualized, considered as the real size of the kidney, which may be suggestive of renal pathology: small kidneys are suggestive of chronic renal failure. In transverse section, approximately in the middle of the kidney, the renal hilum with the renal artery and vein can be seen. Knowing the anatomy of this region is required for the evaluation of vascular structures if a venous (tumor) thrombosis or a renal artery stenosis is suspected.

The ultrasound anatomy of the kidney includes a peripheral hypoechoic area – parenchyma; and a central hyperechoic area - the renal sinus (Fig 20). The ultrasound differentiation between the cortex and the medulla is possible only in children and in thin persons. In current ultrasound practice, this distinction is not possible, so that the renal sinus and the parenchyma will be discussed in relation to the kidney. Normally, the thickness of parenchymal area is 15-20 mm. Its narrowing and poor differentiation between the parenchyma and the renal sinus are suggestive for chronic renal failure.

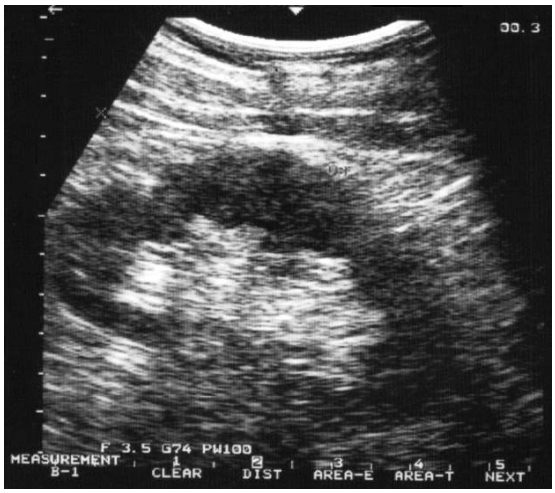


Fig. 20. Normal kidney

6. Large vessels

The large vessels, aorta (AO) and the inferior vena cava (IVC) are retroperitoneal organs that are examined in axial midline section. Scan with the transducer along their length to examine them as long as possible. Slight, progressive compression facilitates the examination by pushing aside the bowel gases that are interposed between the transducer and the vessels. Doppler examination shows blood flow at this level and can expose areas of stenosis or thrombosis.

The ultrasound appearance of the aorta is of a pulsating anechoic formation with hyperechoic wall, located anteriorly to the spine. During the examination, from top to down, the following structures are visualized: the emergence of celiac trunk, the superior mesenteric artery, and sometimes the inferior mesenteric artery (Fig. 21). In transverse section, the aorta

appears as a round structure and we can detect the emergence of the celiac trunk with the hepatic and splenic artery which are "lying" on the upper margin of the pancreas. Approximately 1.5 cm below the celiac truck, the superior mesenteric artery is seen in front of aorta and the spleno-portal axis. A little lower the emergence of renal arteries (right renal artery in front of the ICV) can be seen.

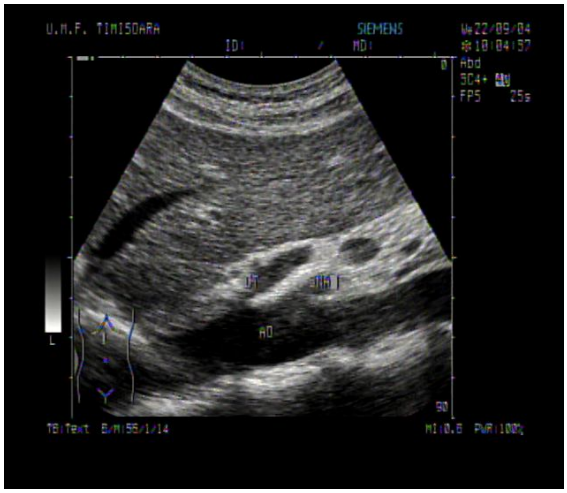


Fig. 21. Aorta (AO), celiac trunk (CT) and superior mesenteric artery (SMA)

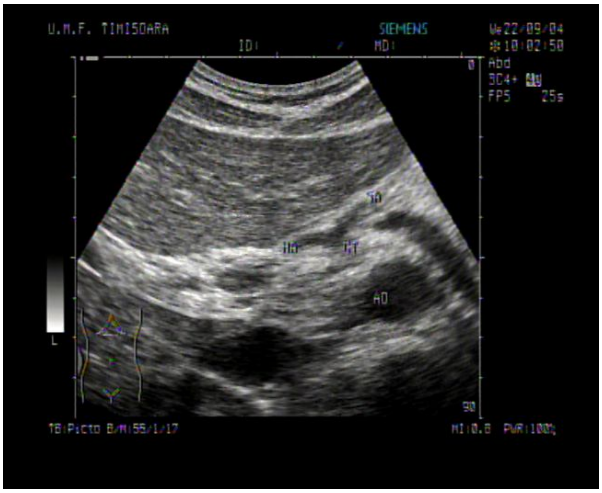


Fig. 22. Aorta (AO), celiac trunk (CT) with hepatic artery (HA) and splenic artery (SA)

IVC is located slightly right of the spine, its ultrasound appearance is of a transonic formation with hyperechoic wall, with slow pulsations, related to respiratory movements (if aorta is pulsating, ICV is fluttering) (Fig.23). Its maximum normal diameter is also 20 mm, higher values are suggestive for heart failure.

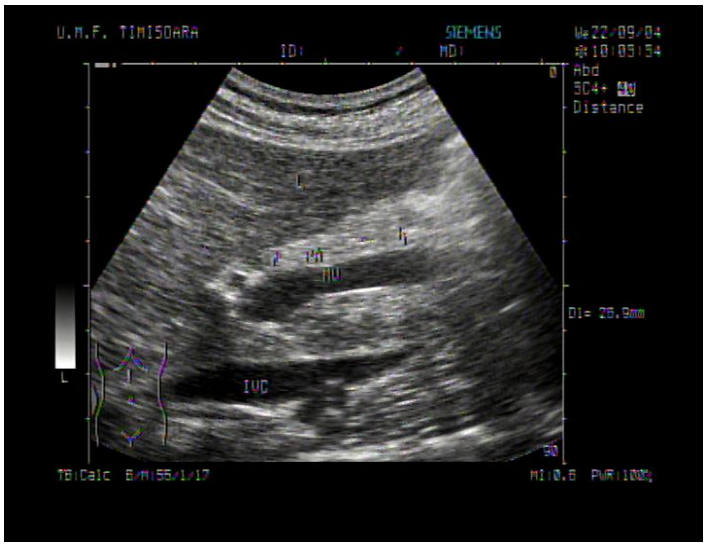


Fig. 23. Inferior vena cava (IVC), superior mesenteric vein (SMV), head of the pancreas (PA).

7. The pelvis

Pelvic organs are examined in transverse and longitudinal sections above the pubes with the transducer angled to the legs. The urinary bladder will be seen as a round anechoic structure with hyperechoic wall, the size is variable depending of the post-urinary time. Normal bladder wall thickness is maximum 4 mm. In elderly men with prostate adenoma, the bladder wall may be thicker - "fighting bladder."

In men, the urinary bladder neck is surrounded by the prostate - a parenchymal structure with the maximum normal diameter 3/4 cm. Larger diameters are suggestive for prostate adenoma (Fig. 24).

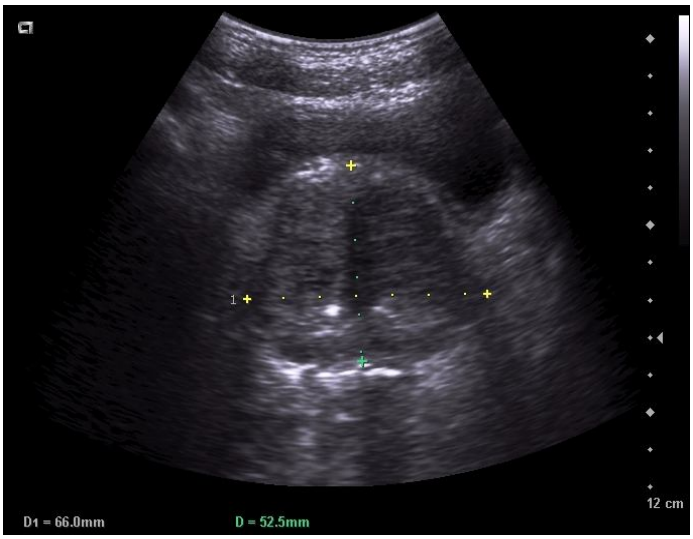


Fig. 24. Prostate adenoma

In women the uterus is visualized posteriorly of the urinary bladder, as an echoic structure, pear shaped in longitudinal section. In adult fertile woman its maximum size is 9/5 cm. Normally the uterus is bent forward, an angle of 60° is formed between the cervix and uterus (Fig. 25). When the uterine fundus is found in the Douglas space (the uterus is bent backwards), it defines the uterus in retroversion (Fig. 26). The myometrium appears as a thick, hypoechoic area located peripherally, which encloses the endometrium, a hyperechoic area whose size varies according to the phases of the menstrual cycle. In cross section the uterus appears like a round structure, located at the base of the urinary bladder (Fig. 27). By scanning to the left and to the right we can visualize the ovaries, slightly hypoechoic, round or oval structures, maximum 3/2 cm diameter.

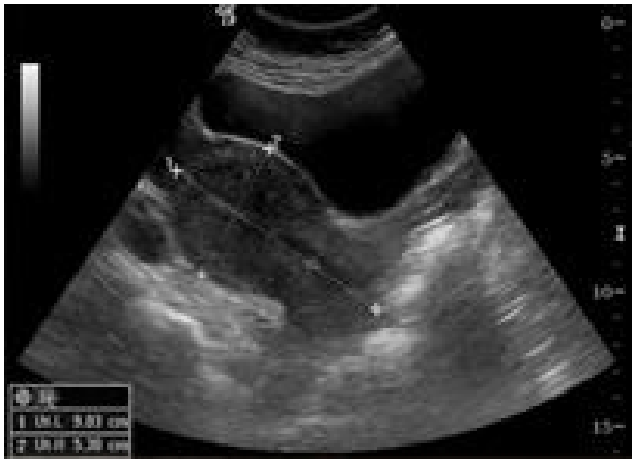


Fig. 25. Uterus in anteversion – longitudinal section

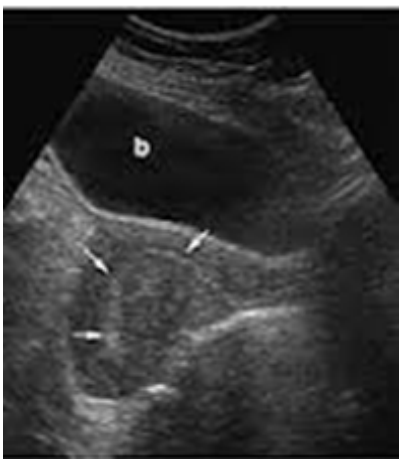


Fig. 26. Uterus in retroversion – longitudinal section

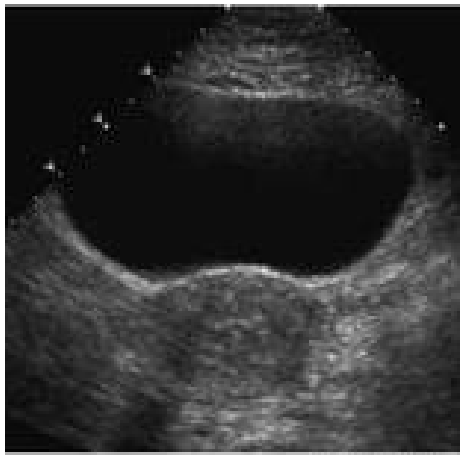


Fig. 27. Uterus – cross section

Chapter 3.

Abdominal ultrasound in the diagnosis of diffuse liver diseases

The liver is the organ in which ultrasound evaluation has maximum value. In experienced hands, standard ultrasound, especially with contrast, can establish difficult diagnoses, without requiring other expensive imaging investigations required. Liver ultrasound should only be done in a clinical context, after knowing the patients' history, after a brief physical examination, during which palpation of the liver is mandatory. Thru palpation we can appreciate the size of the liver (more accurately than by means of imaging) and its consistency – a useful element for the diagnosis of chronic liver diseases.

In the following we will refer to the value of ultrasonography for the diagnosis of acute hepatitis, of chronic hepatitis, of liver steatosis (diffuse or focal), for the diagnosis of liver cirrhosis and cardiac cirrhosis.

1. Acute hepatitis

Acute hepatitis is an acute illness of the liver, characterized by the increase of aminotransferases, especially GPT, values typically more than 10 times the upper limit of normal. Acute viral hepatitis can be caused by some typical hepatotropic viruses (hepatitis A, B, C and E virus) or by other viruses (herpes virus, Epstein-Barr virus or cytomegalus virus), by alcohol abuse, or rare causes: toxic drugs (Paracetamol, Halotan, etc), acute autoimmune hepatitis.

The diagnosis of acute hepatitis is made in an epidemiological context (contact with a hepatitis virus infected person; with possible infected blood or blood products; hepatotoxic drugs intake; alcohol abuse, etc.); in a biological and clinical context (asthenia, dyspepsia, often fever, with or without jaundice). Among blood tests, high elevated transaminase values, with or without increased bilirubin, with or without positive markers for infection with hepatitis viruses: HAV - IgM antiHAV; HBV - HBsAg and IgM anti HBV; HCV - RNA PCR HCV viral load, are needed for a positive diagnosis.

The **ultrasound** appearance of acute hepatitis is *not characteristic*. Liver ultrasound is frequently completely normal. Sometimes, some ultrasonographic signs can suggest this diagnosis.

Thickened and doubled gallbladder wall occurs in up to 80% of acute hepatitis, particularly in viral hepatitis (Fig. 1). It is due to hypoalbuminemia that generates gallbladder wall edema, and it's a suggestive sign for acute viral hepatitis in a young person with jaundice and dyspeptic syndrome.

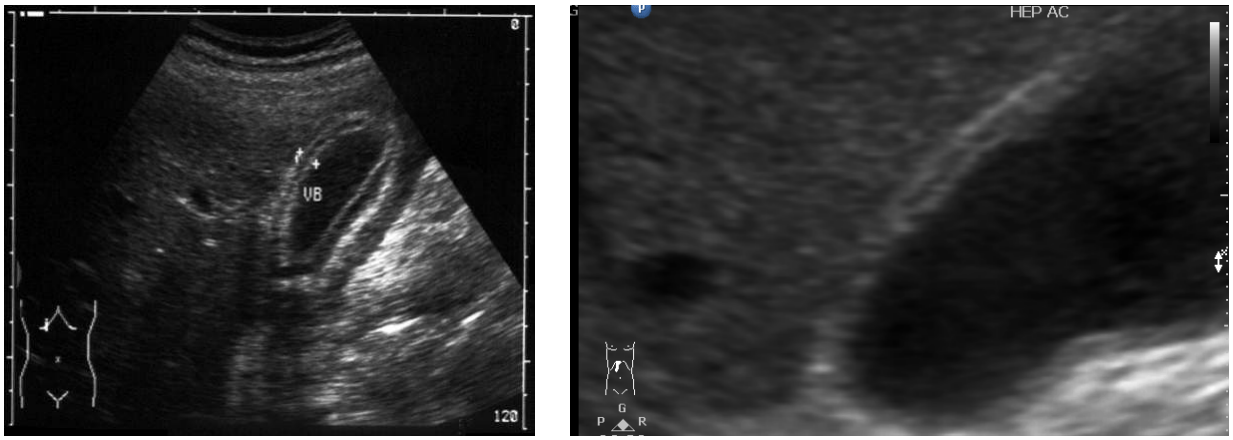


Fig. 1. Thickened and doubled wall in acute hepatitis

Other ultrasonographic signs, but with poor specificity are: *diffuse liver hypoechogenity* (difficult to evidence by ultrasound in the absence of a landmark structure), due to the hepatic edema; and possibly *mild splenomegaly* (slightly enlarged spleen – considering a spleen < 12 cm in its long axis as normal).

In acute alcoholic hepatitis, the background can be of hepatic steatosis ("bright liver" with posterior attenuation on ultrasound), consequence of prolonged alcohol abuse, not of acute hepatitis.

2. Chronic hepatitis

Chronic hepatitis is a chronic inflammatory disease of the liver, of various etiologies, with an evolution of minimum 6 months, without a tendency to healing, with necrotic and fibrotic lesions as a pathological substrate. The biological expression of chronic hepatitis is usually a moderate cytolysis syndrome. To state the diagnosis of chronic liver disease, the cytolysis syndrome must last at least six months, because in some cases the detection of the moderately increased transaminase levels could be in the context of a previously undiagnosed acute hepatitis, which will heal spontaneously in several weeks.

Chronic hepatitis is most frequently caused by hepatitis B, C or B+D viruses. Hepatitis A does not become chronic. A common cause of chronic hepatitis is alcohol abuse which causes fatty liver (alcoholic steatohepatitis). Other causes of chronic hepatitis are: nonalcoholic steatohepatitis, autoimmune hepatitis, toxic drug hepatitis, cholestatic hepatitis or abnormal storage of metals in the liver – hemochromatosis (iron) and Wilson disease (copper).

Ultrasound examination in chronic hepatitis does no evidence typical signs. Most frequently (in approx. 50% of cases), a mild *splenomegaly* is detected (up to 13-14 cm). The majority of authors consider as the upper limit of normal 12 cm as the length of the long axis of the spleen. The width and the thickness of the spleen are not equally important, but a globulous spleen can be a sign of activation of the reticuloendothelial system. Larger splenomegaly (>15 cm) suggests liver cirrhosis (Fig. 2) in a clinical context.

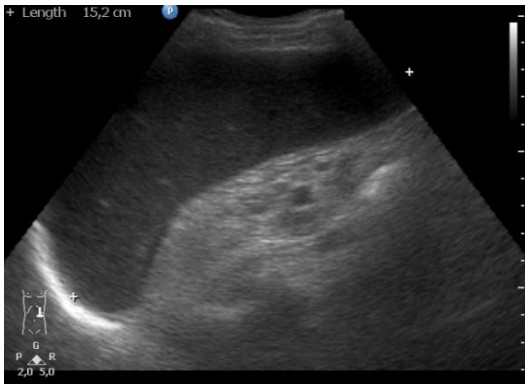


Fig. 2. Splenomegaly in a patient with chronic hepatitis C (longitudinal diameter 15 cm)

A frequent ultrasound sign, especially in chronic hepatitis C (up to 70% of the cases), but also in autoimmune hepatitis or chronic hepatitis B, is finding lymph nodes in the hepatic hilum (adenopathies of the hepato-duodenal ligament) (Fig. 3). The lymph nodes of the hepato-duodenal ligament are usually oval, 5-10/10-20 mm in size. They are best visualized along the hepatic artery or the portal vein. They must be differentiated from malignant adenopathies, which are generally round and hypoechoic.

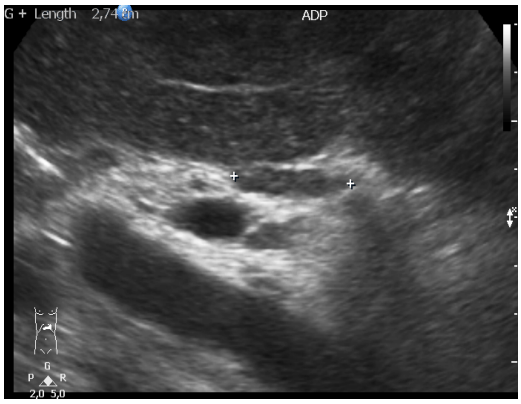


Fig. 3. Hepatic hilar adenopathy

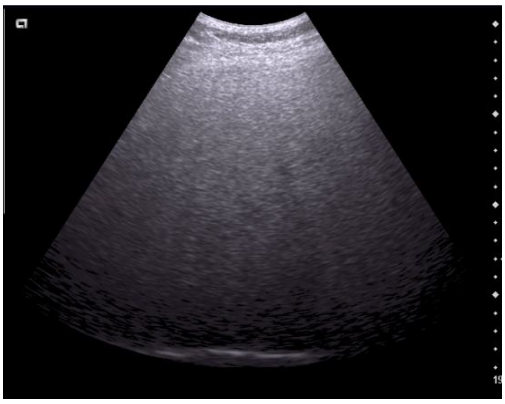


Fig. 4. Moderate hepatic steatosis

In patients with alcoholic or nonalcoholic steatohepatitis, and in some of those with chronic hepatitis C, the ultrasound appearance will be of *hepatic steatosis* (bright hyperechoic liver, with posterior attenuation (Fig. 4).

Although abdominal ultrasound does not give decisive evidence for the diagnostic of chronic hepatitis, it can be a useful tool for diagnostic and for a correct evaluation. Specifically ultrasound is used to choose the place where liver biopsy will be performed (ultrasound guided or assisted hepatic biopsy) and for follow-up assessment (every 6 months) in patients with severe fibrosis and cirrhosis for HCC screening.

In conclusion, ultrasound examination in chronic hepatitis has a *limited value*, only splenomegaly and hepato-duodenal ligament adenopathies are relatively constant elements (good sensitivity, but lower specificity).

3. Hepatic steatosis

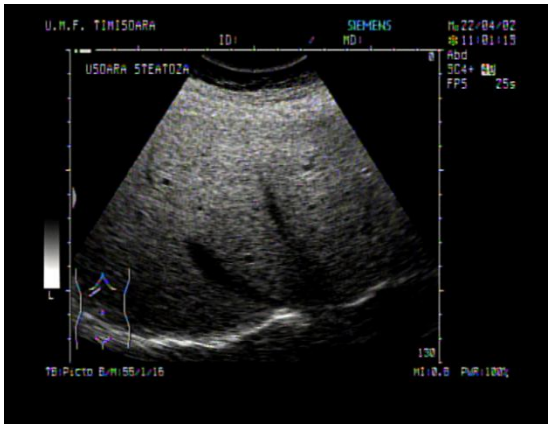
Hepatic steatosis is defined as fatty loading of the liver higher than 10%. The main causes of hepatic steatosis are: chronic alcohol intake (alcoholic steatohepatitis – ASH syndrome), obesity, diabetes, dyslipidemia (non-alcoholic steatohepatitis – NASH syndrome). Another cause of fatty liver chronic infection with hepatitis C virus (up to half of the chronic C virus patients have mild fatty loading).

Before the development of modern imaging methods (CT, ultrasound) it was believed that hepatic steatosis is always diffuse. Later, in the early 80s, this imaging methods showed that hepatic steatosis can also affect the liver unequally, therefore areas that have less fat (fatty-free areas) can appear on o fatty liver, and fatty loaded areas (focal fatty infiltration) can appear in a normal liver. The reason for this different fat load is not clear, it is likely due to changes in the arterial-portal-venous vasculature, well vascularized areas being less fatty.

Hepatic steatosis can be simple (asymptomatic) or it can be associated with inflammation, manifested through cytolysis syndrome (steatohepatitis). Ultrasound cannot differentiate between the two, therefore, in patients with steatosis the cytolysis syndrome should always be evaluated (an increased De Rittis GOT/GPT ratio can be suggestive for ethanolic etiology), and also the presence of HCV antibodies (association of liver steatosis with chronic hepatitis C virus).

Hepatic steatosis is easily and precisely diagnosed by ultrasound (sensitivity 90 %). The ultrasound appearance is of a hyperechoic liver as compared with the renal parenchyma, "bright liver", often accompanied by "*posterior attenuation*" due to partial absorption of ultrasound waves by the fatty tissue. There is a direct correlation between liver fat load and the severity of posterior attenuation. Thus, depending on the intensity of posterior attenuation, a subjective and

semi-quantitative assessment of the steatosis can be made: mild steatosis (discrete attenuation - Fig.5, Fig. 6), moderate steatosis (obvious attenuation – Fig.7, Fig. 8), and severe steatosis (difficult or impossible to visualize posterior diaphragm – Fig.9, Fig.10) Special attention should be given to patients with severe steatosis, in whom deep lesions are difficult to visualize due to posterior attenuation. In these cases, CT is recommended for solving unclear cases.



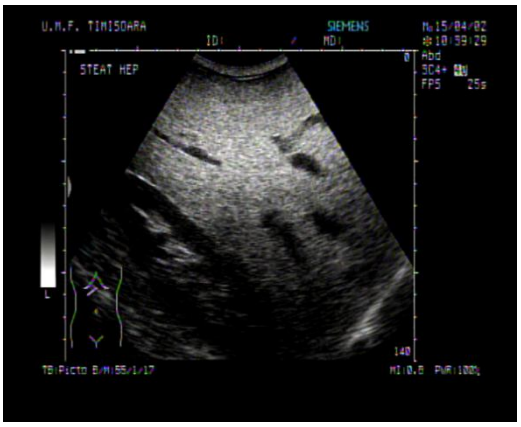
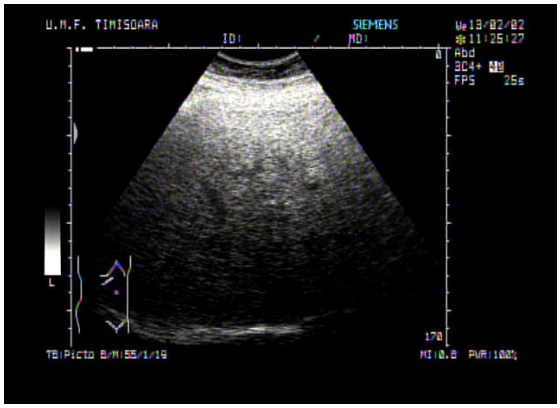


Fig. 5; Fig. 6. Mild hepatic steatosis



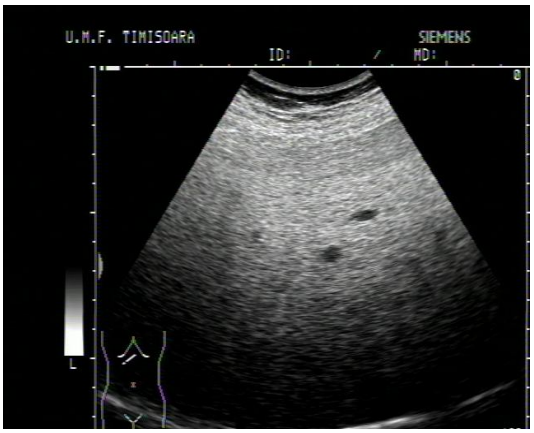
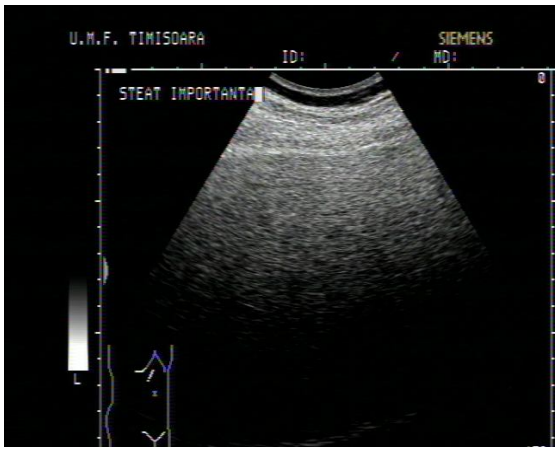


Fig.7; Fig. 8. Moderate hepatic steatosis



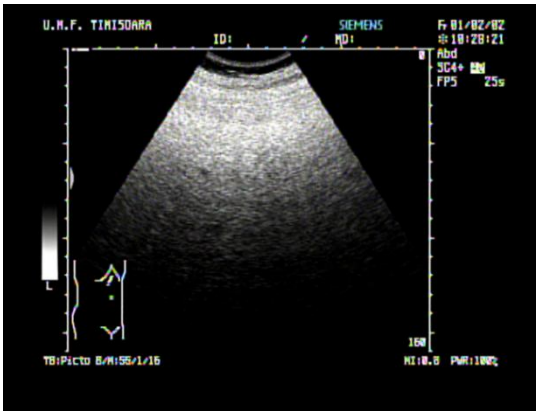


Fig. 9; Fig. 10. Severe hepatic steatosis – the diaphragm cannot be visualized

Focal steatosis and fatty-free areas are particular situations quite frequently encountered. The ultrasound appearance is the juxtaposition of liver tissue with different echogenity: fatty-free areas are hypoechoic areas in a hyperechoic liver (Fig. 11, Fig. 12), focal steatosis are hyperechoic areas in a liver with normal echogenity (Fig.13). The delimitation of these areas is clear, they have often a geographical contour and variable size. They never alter the hepatic surface or infiltrate and invade vascular structures.

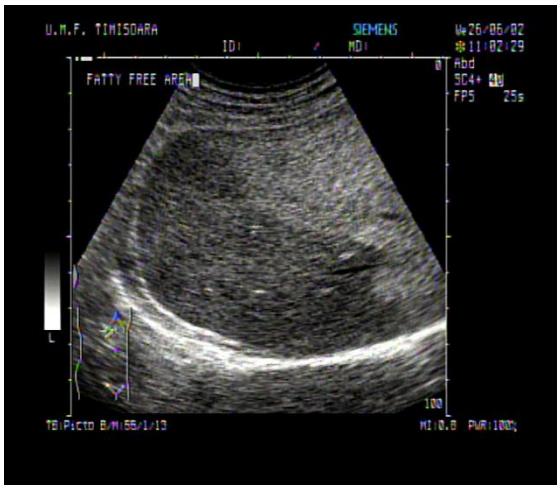


Fig. 11. Fatty-free area in the right lobe

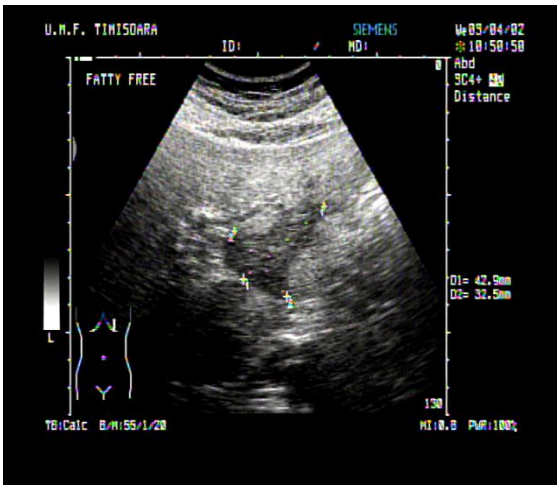


Fig. 12. Fatty-free are in the left lobe

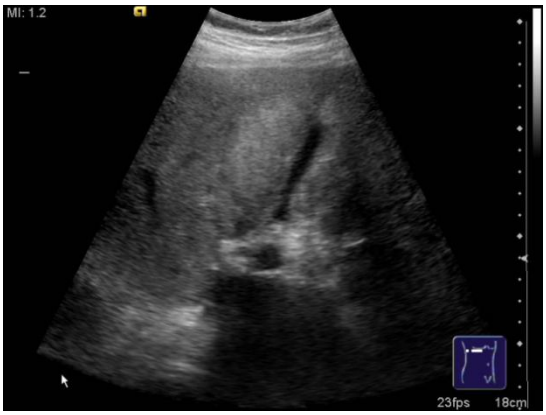


Fig. 13. Focal steatosis, surrounding the gallbladder

A common case of focal hepatic steatosis is the fatty hilum. This involves excess fat storage in a typical hepatic area, which is situated at the portal bifurcation. It is an oval shaped area, usually 3-4/2-3 cm in size, situated at the bifurcation of the portal vein, between its right and left branch. Differential diagnosis should exclude a hemangioma or a hepatic tumor.

The differential diagnosis of fatty-free areas is difficult, we have to exclude primitive or secondary liver tumors that can appear in fatty liver. This cannot be done only by standard ultrasound, contrast imaging methods are necessary, CEUS, CT or MRI.

In conclusion to the chapter on hepatic steatosis, it can be said that ultrasound is a good method for assessing hepatic steatosis (a non-invasive technique), and also a method for the semi-quantitative evaluation of steatosis (relatively well correlated with the histological fat loading of the liver). In the case of focal hepatic steatosis or fatty free areas, the positive ultrasound diagnosis is easy, while differential diagnosis will require an experienced ultrasonographist and sometimes, evaluation by contrast-enhanced ultrasound (CEUS).

4. Liver cirrhosis

Liver cirrhosis is the final stage of the majority of chronic liver diseases, in which necrosis, regenerative phenomena and fibrous changes coexist, resulting in the nodular transformation of the liver.

The etiology of liver cirrhosis is multiple, but alcohol and hepatitis viruses B and C are responsible in 90% of the cases. Rare causes are autoimmune hepatitis, Wilson's disease (ceruloplasmin deficiency), hemochromatosis, alpha-1-antitrypsin deficiency, primary biliary cirrhosis, drug-induced cirrhosis, and cryptogenic cirrhosis (a rare condition).

Though advanced cirrhosis has a typical ultrasound appearance: ascites, irregular hepatic surface, heterogeneous structure of the liver, splenomegaly (90% specificity for the diagnostic); in early cirrhosis the ultrasound aspect can be perfectly normal (up to 20% of the cases).

The typical elements that can be found in liver cirrhosis (which are not necessarily present) are: caudate lobe hypertrophy, heterogeneous liver structure, splenomegaly, ascites, signs of portal hypertension, and changes in the gallbladder wall.

a) Caudate lobe hypertrophy

The caudate lobe (segment 1 of the liver) undergoes hypertrophy during the evolution of liver cirrhosis, so that it will be frequently enlarged in this pathological condition. In practice we measure the anterior-posterior diameter of the caudate lobe. An antero-posterior diameter greater than 35 mm is suggestive for the diagnostic of liver cirrhosis (Fig. 14, Fig. 15), since in about 2/3 of the cases the caudate lobe is hypertrophied. The antero-posterior diameter of the caudate lobe will be measured in a sagital section at epigastric level. The inferior vena cava (IVC) is seen and anterior to it, the ovoid structure of the caudate lobe will appear.

Subsequently, the maximum antero-posterior diameter of the caudate lobe will be measured. Certain measurement problems may occur in the case of marked steatosis (ultrasound waves are strongly absorbed by fat tissue) or, more rarely, in case of ascites.

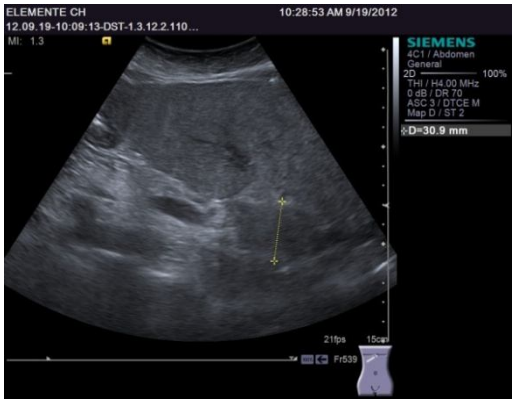


Fig. 14. Normal caudate lobe (31 mm) in liver cirrhosis

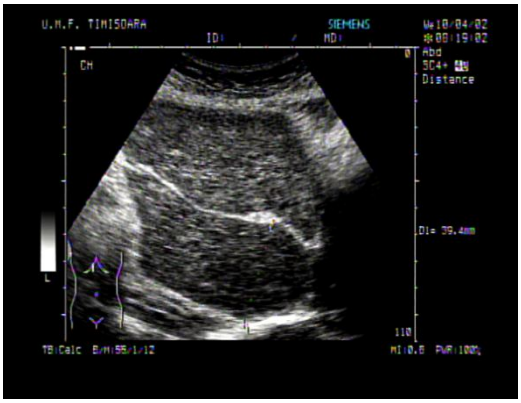


Fig. 15. Enlarged caudate lobe (39 mm) in liver cirrhosis

b) Heterogeneous hepatic structure

The aspect of hepatic heterogeneity occurs in approximately half of the liver cirrhosis cases, as the consequence of fibrous changes, and of regeneration nodules. Practically, instead of the appearance of fine mixed salt and pepper, like in normal liver (Fig. 17), in the case of heterogeneous liver structure "salt and pepper" will appear coarsely milled (Figure 18). Special attention should be paid in patients with known liver cirrhosis, in which the liver structure is highly heterogeneous, particularly if this aspect is limited to certain areas. In these conditions the presence of a diffuse HCC should be suspected. It is recommended to measure the alpha-fetoprotein and to perform a contrast imaging method (ultrasound, CT or MRI) to elucidate the diagnostic.

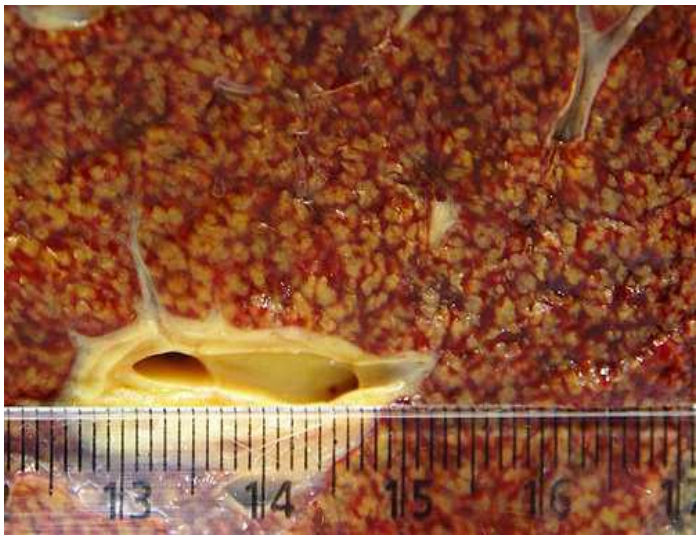


Fig. 16. Macroscopic appearance of micronodular cirrhotic liver section

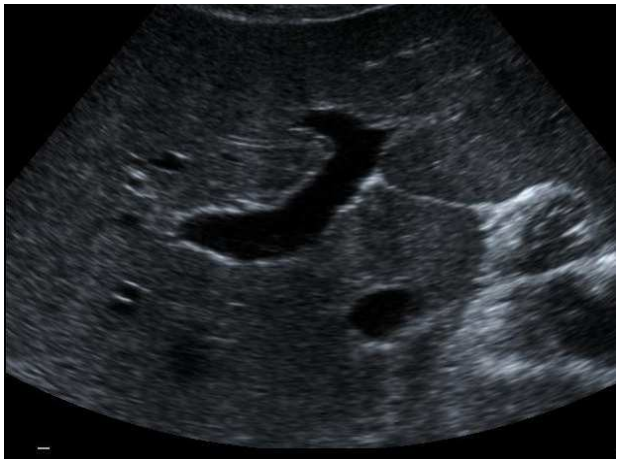


Fig. 17. Normal liver echostructure in liver cirrhosis

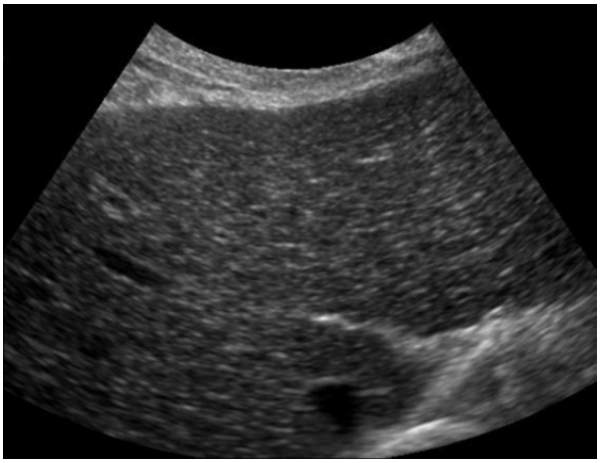


Fig. 18. Heterogenous structure in liver cirrhosis

c) Irregular hepatic surface

Irregular liver surface is the consequence of histological micronodular liver (which cannot be demonstrated by ultrasound inside the liver parenchyma, although the term is incorrectly used in everyday practice). Micronodular liver is a histological reality in liver cirrhosis, but ultrasound cannot highlight these parenchymal nodules.

Irregular liver surface is easily evidenced in the presence of ascites (Fig.19). In its absence the liver surface is difficult to assess. The examination can be facilitated by using high frequency transducer (5-9 MHz) (Fig. 20).

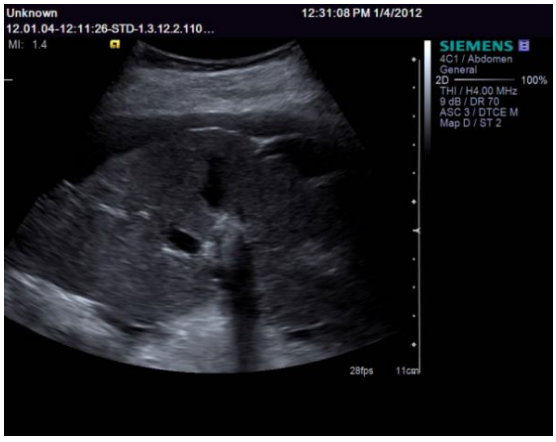


Fig. 19. Irregular liver surface liver cirrhosis and ascites

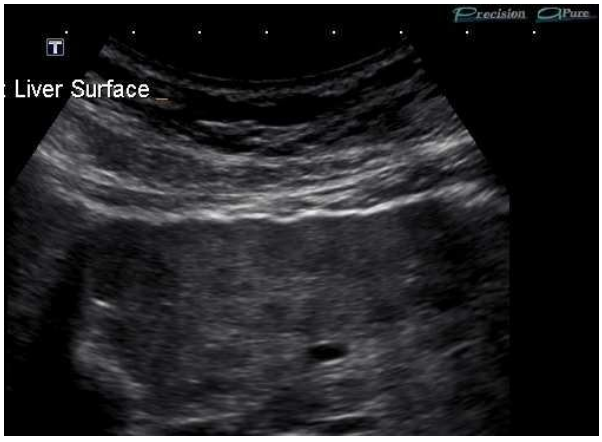


Fig. 20. Irregular liver surface (5MHz transducer)

We have to mention that when we find through ultrasound a heterogeneous liver structure and an irregular liver surface in an asymptomatic patient, without any history of liver pathology, we need to think of a possible chronic liver disease. Clinical examination, biological evaluation, FibroScan, and endoscopic evaluation in these cases can discover unknown liver cirrhosis.

d) Splenomegaly

An enlargement of the spleen exceeding 12 cm along its long axis is frequent in the case of liver cirrhosis, in approximately 80% of the cases (Fig. 21). In these patients splenomegaly is more severe than in chronic hepatitis, frequently exceeding 15 cm. Splenomegaly larger than 18 or even 20 cm is often accompanied by hematological hypersplenism (thrombocytopenia<100,000/mm^3, and/or leucopenia < 3000/mm^3, and/or anemia). In other situations, the increase in the long axis of the spleen is not necessarily very important, but the spleen has a globulous appearance, through the increase of its width and thickness.

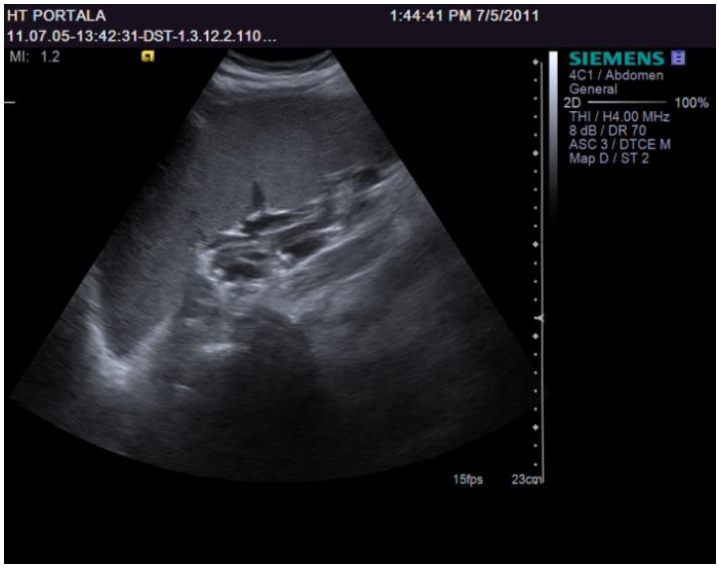


Fig. 21. Splenomegaly

e) Ascites

Ultrasound is a very sensitive method to evidence ascites, frequently encountered in patients with decompensated cirrhosis. It is also useful for assessing the volume of ascites and its evolution during diuretic therapy. We subjectively appreciate the ascites volume (minimal, small, moderate and large), based on the amount of liquid in the Douglas space and in the perihepatic space. We consider that in a minimal ascites the amount of peritoneal liquid is about 1-2 kg (Fig. 22), in a mild ascites 3-4 kg (Fig. 23), in a moderate ascites about 7-8 kg (Fig. 24) and in a voluminous ascites 10-15 kg (Fig. 25, Fig. 26).

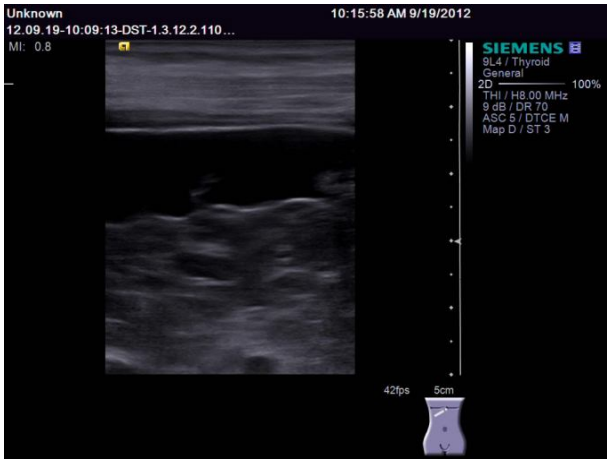


Fig. 22. Minimal perihepatic ascites (8 MHz transducer)

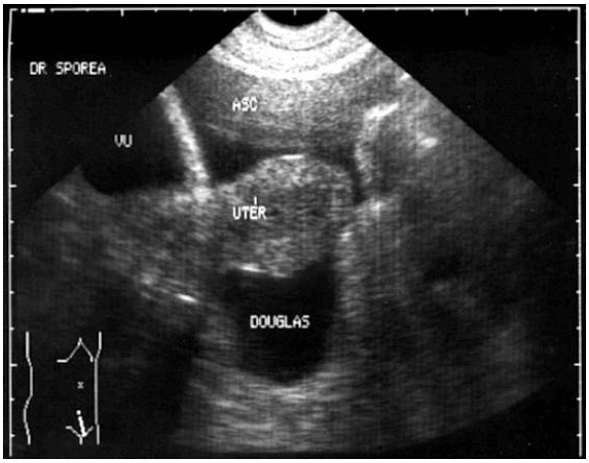


Fig. 23. Small ascites in Douglas space

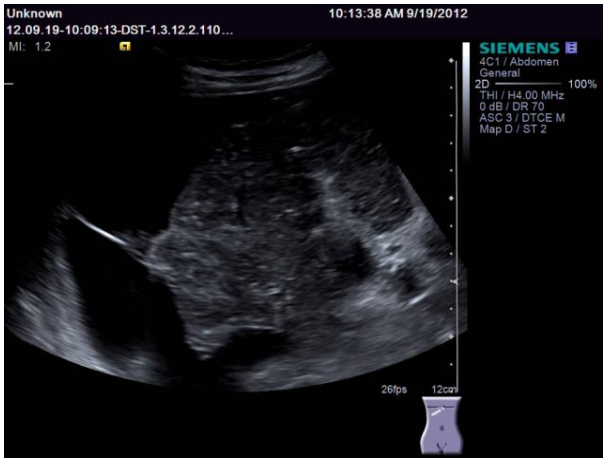


Fig. 24. Moderate perihepatic ascites Intestinal loops "float" in the ascites

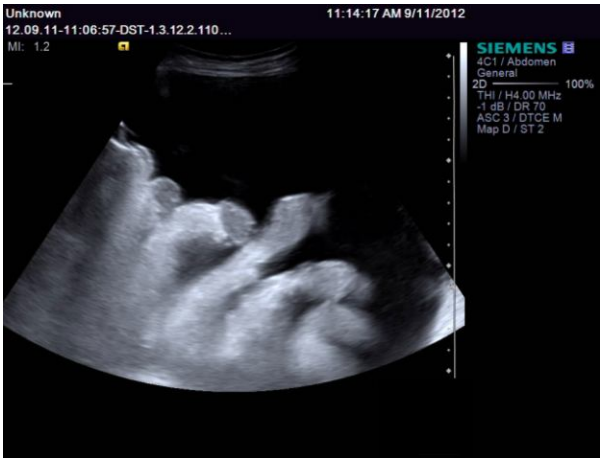
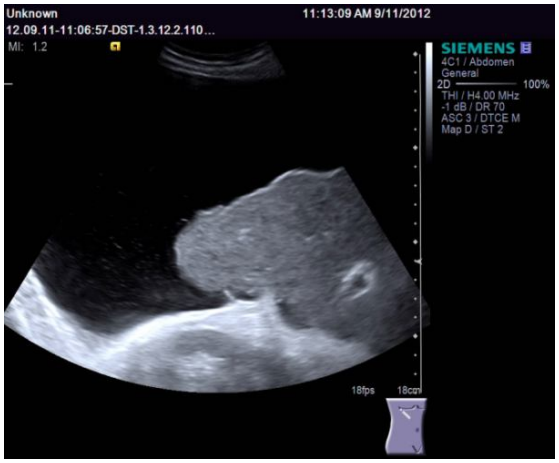


Fig. 25. Large ascites in the Douglas space

The ultrasound appearance of ascites is of an anechoic image that changes form with changes in the patient's position. We search for ascites in Douglas space, in Morrison space (interhepatorenal), perihepatic and perisplenic. In a patient with old ascites, the ascites may not be completely anechoic, it can be slightly hypoechoic and contain small echogenic particles in Brownian motion, aspect of "dense" ascites (Fig. 26, Fig. 27). Excepting the patient with old ascites, "dense" ascites can also occur if the ascites is infected (spontaneous bacterial peritonitis), in hemoperitoneum or chylous ascites. A diagnostic paracentesis is recommended if "dense" ascites is found in a patient with liver cirrhosis.



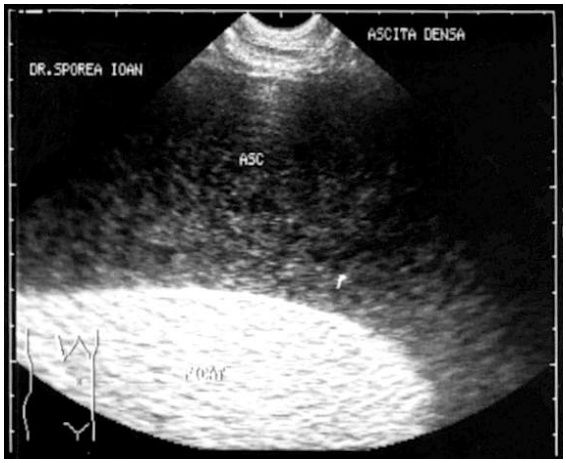
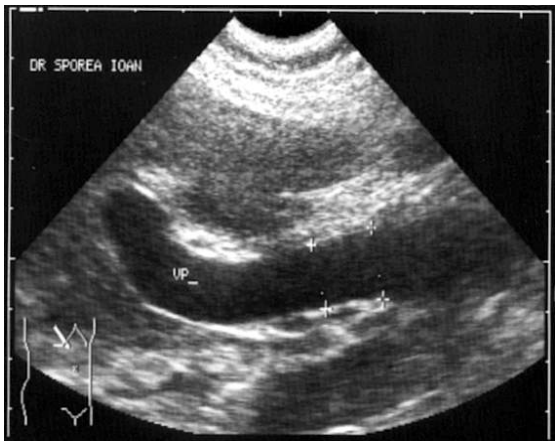


Fig. 26. Fig. 27. Large, „dense" perihepatic ascites

f) Signs of portal hypertension (PHT)

One of the consequences of fibrosis in liver cirrhosis is the increase of portal circulation resistance. The consequences of portal hypertension include collateral abdominal, peritoneal circulation, the opening of vascular shunts and the appearance of varices, most frequently located in the esophagus.

One of the first signs of PHT in ultrasound is the increased diameter of the portal vein in the hilum to 13-14 mm (Fig. 28), and its lack of variability in inhale/exhale (Bolondi sign). Dilatation of intrahepatic portal system may also occur in case of PHT in liver cirrhosis (Fig.29), but its assessment is somewhat subjective, since an upper limit size was not defined as for the portal vein in the hilum.



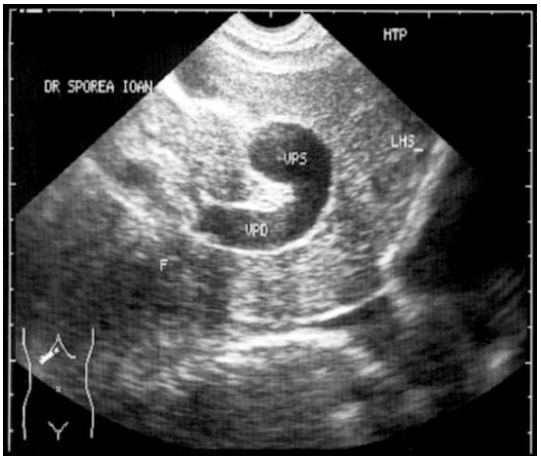


Fig. 28. Dilatation of the portal vein in the hilum

Fig. 29. Dilatation of the portal bifurcation

Measurement of the splenic vein in front of the aorta and in the splenic hilum can provide some elements of PHT. Thus, a splenic vein larger than 10 mm measured or in front of the aorta or larger than 8 mm measured in the hilum (Fig. 30) and (Fig. 31), respectively, can be a sign of portal hypertension.

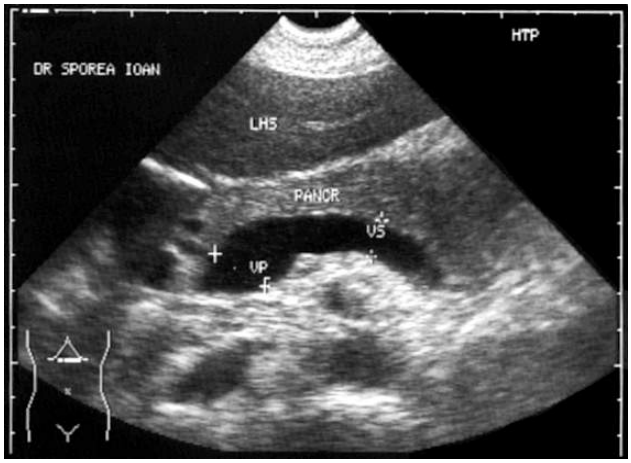


Fig. 30 Dilatation of the splenic-portal axis in the hilum

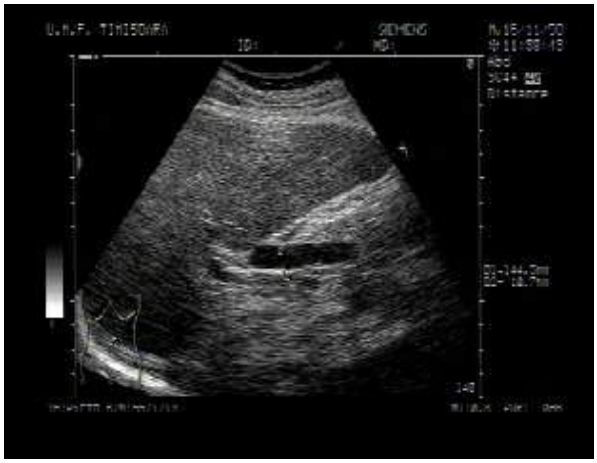


Fig. 31. Splenomegaly and dilatation of the splenic vein

Other signs of HTP are the dilation of visceral veins and the appearance of venous shunts. The detection of collateral epigastric circulation (dilation of the gastric coronary vein), of spontaneous splenic-renal shunts or of splenic varices (Fig. 32) are typical signs of portal hypertension. In ultrasound they appear as multiple anechoic images that communicate between them and the Doppler examination shows present flow (Fig. 33).

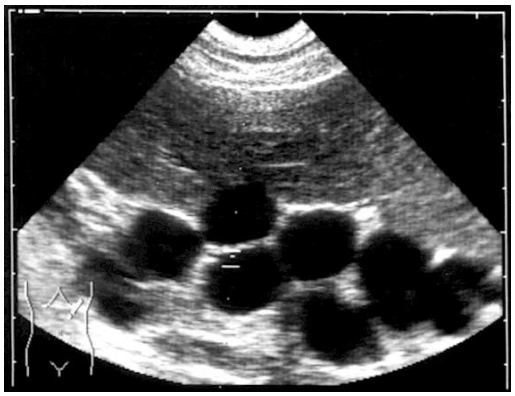


Fig. 32. Splenic varices

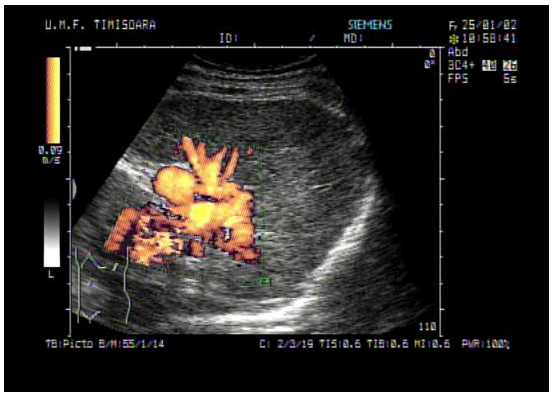


Fig. 33. Splenic varices – Doppler examination

Repermeabilization of the umbilical vein is a severe sign of PHT that can be found in 10-20% of advanced cirrhosis cases. The repermeabilization of the umbilical vein will be checked starting from the left branch of the portal vein (Fig. 34), where a vascular (venous) cord starts, continuing to the lower side of the liver and then, posteriorly to the abdominal wall, towards the umbilicus (Fig. 35).

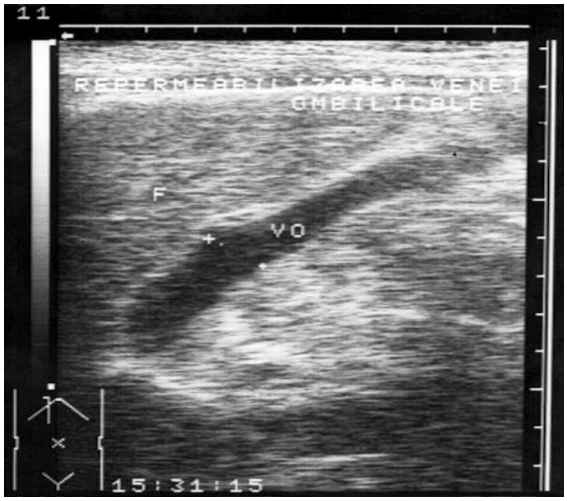


Fig. 34. Repermeabilization of the umbilical vein intrahepatic aspect

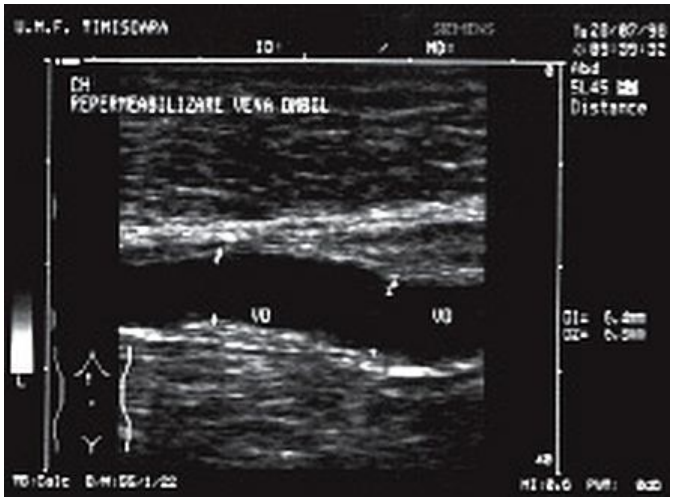


Fig. 35. Repermeabilization of the umbilical vein - abdominal wall

Portal thrombosis (the presence of echo dense material in the lumen of the portal bifurcation or the common PV) (Fig. 36) in a patient known with liver cirrhosis should raise the suspicion of HCC. In malignant PV thrombosis due to HCC, the portal thrombus is actually neoformation tissue, vascularized, which will enhance following contrast bolus in CEUS, CT or MRI with contrast. Portal thrombosis may also be benign, in this case the blood clots form as a result of slowdown of the portal blood flow in PHT. Since it is avascular, the benign portal thrombus will not enhance following contrast bolus in contrast imaging.

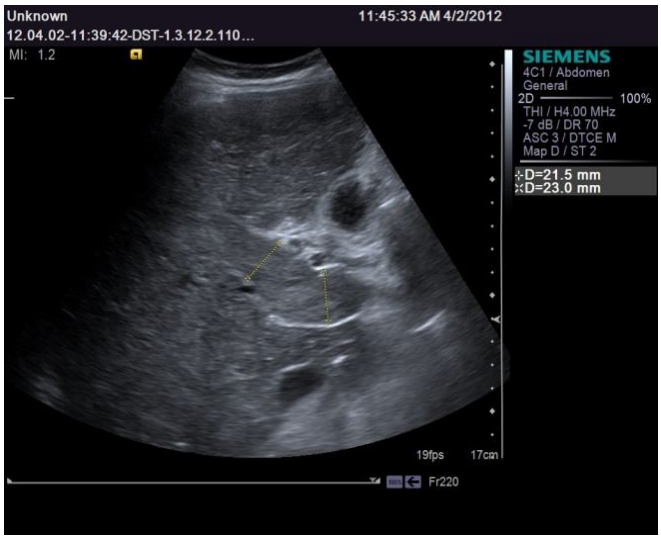


Fig. 36. Portal thrombosis (Common portal vein)

g) Changes in the gallbladder wall

A frequent aspect in liver cirrhosis is the thickening and duplication of the gallbladder wall due to edema secondary to hypoalbuminemia, portal hypertension and lymphatic stasis. The diameter of the normal vesicular wall is less or equal to 4 mm. Given that in liver cirrhosis gallstones are found often enough, special attention must be paid to the differential diagnosis with acute cholecystitis, in which an ultrasound diagnostic criteria is also the thickening of the gallbladder wall. But acute cholecystitis is also accompanied by a suggestive clinical aspect and the ultrasound Murphy's sign (intense pain at pressure with the transducer in the gallbladder area).

In liver cirrhosis, the gallbladder wall can be thickened, reaching 6-8 or even 10 mm (Fig. 37, Fig. 38), most frequently doubled (with a "sandwich" appearance). In ascites with unknown etiology, by measuring the gallbladder wall we can differentiate ascites due to peritoneal carcinomatosis and due to tuberculosis, in which the vesicular walls are normal, from of the one in liver cirrhosis were the gallbladder has thickened, doubled wall.

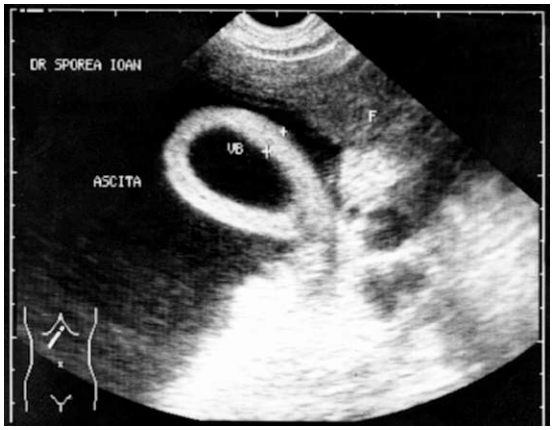


Fig. 37. Gallbladder with thickened, doubled wall - liver cirrhosis

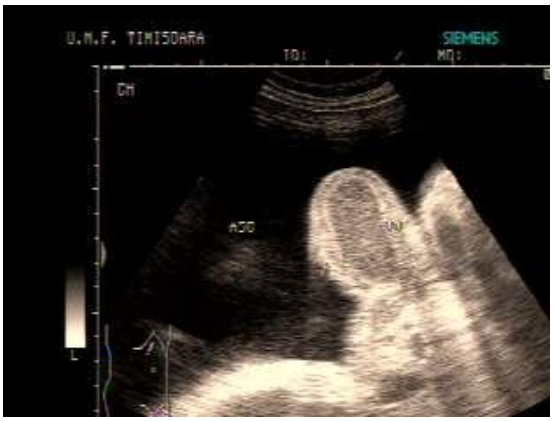


Fig. 38. Gallbladder with thickened wall, filled with sludge in liver cirrhosis

.

Approximately one third of the patients with liver cirrhosis have gallstones and/or biliary sludge (Fig.39), but most often they are asymptomatic gallstones, satellite to the background disease, and they do not require surgical intervention.

In PHT we can also notice varices near the gallbladder that appear as anechoic, confluent structures (Fig.39), with Doppler signal (Fig.40), in the immediate vicinity of the gallbladder.

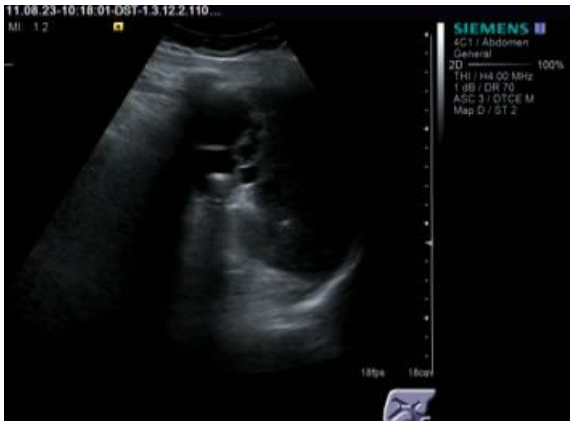


Fig. 39. Gallstones and gallbladder varices

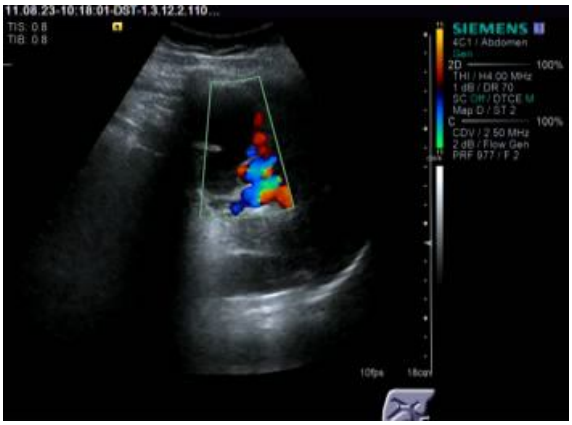


Fig. 40. Gallbladder varices - Doppler

5. Cardiac liver

Cardiac liver includes changes due to vascular alterations and venous stasis, secondary to right heart failure. The ultrasound aspect of cardiac liver appears in a clinical context, with signs of right or global heart failure, in a patient with a known long history of cardiac disease or with an old broncho-pulmonary disease (cordus pulmonale), with dyspnoea, hard cyanotic edema of the inferior limbs and frequently ascites.

The ultrasound signs of cardiac liver are dilation of the hepatic veins and of the inferior vena cava (IVC). *Dilation of the hepatic veins* (SHV) is typical (Fig. 41) and can be quantified by measuring their diameter 2 cm from the junction with the inferior vena cava (IVC). In right or global heart failure SHV will appear larger than 10 mm, very well visible up to the periphery (Fig. 42). The normal respiratory variability of the hepatic veins disappears. Another ultrasound sign of heart failure is *the dilation of the inferior vena cava*, to more than 20 mm in diameter (Fig. 43), but especially the disappearance of the physiological inspiration/expiration variability.

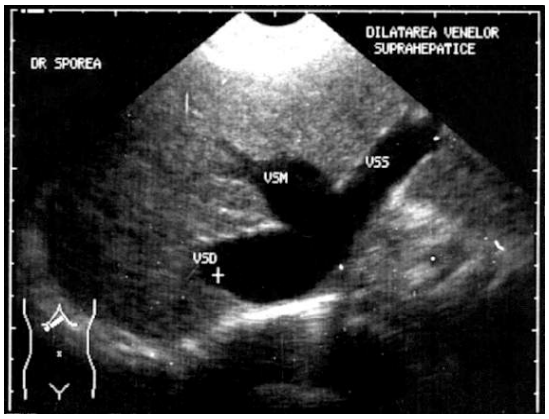


Fig. 41. Dilation of SHV in cardiac liver

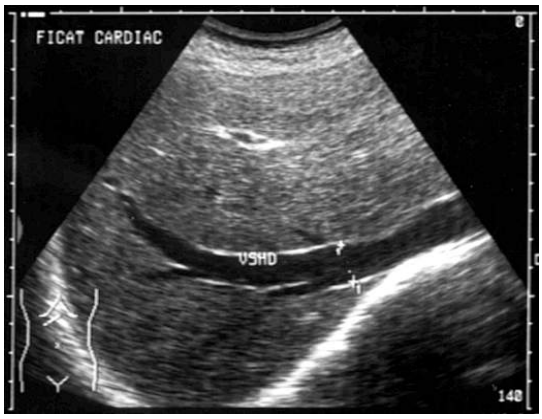


Fig. 42. Right SHV visible in the periphery in cardiac liver

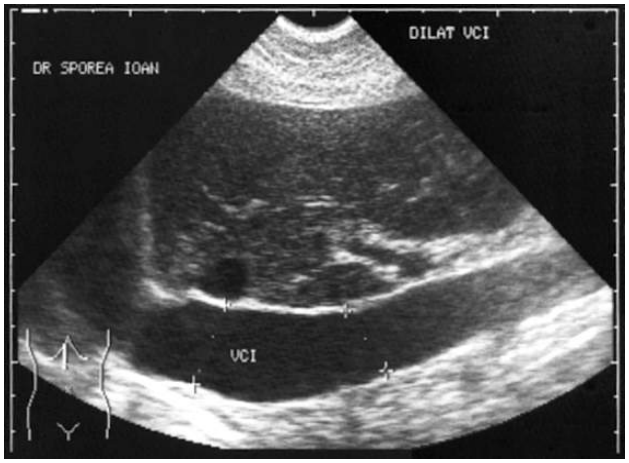


Fig. 43. Dilation of IVC in heart failure

In heart failure sometimes we can detect *peritoneal effusion* (ascites) in variable amount, particularly in the Douglas space or in the perihepatic area. The presence of *pleural effusion* is relatively frequent. It appears as an anechoic crescent situated outside the diaphragm (Fig. 44), which allows the differentiation from the peritoneal effusion (fluid bellow the diaphragm) (Fig. 45). The volume of pleural effusion (small or large) can also be correctly assessed by ultrasound. The diagnosis of pleural effusion is easier to make on the right side (where the ultrasound window of the liver is used) than on the left side. The *pericardial effusion* appears as an anechoic area surrounding the heart (Fig. 46) and has a variable volume. We recommend in all cases of suspected pericardial effusion to request echocardiographic examination, by which the cardiologist will confirm the diagnosis (there is a possibility of confusion between pericardial effusion and highly hypoechoic pericardial fat). Unlike pericardial fat, pericardial effusion changes with the movements of the patient.

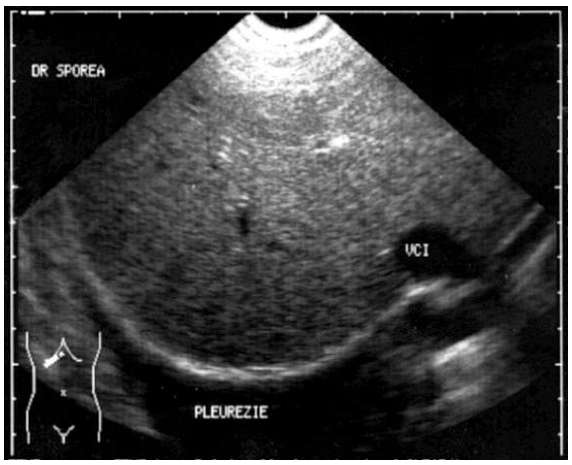


Fig. 44. Fluid in the right pleura

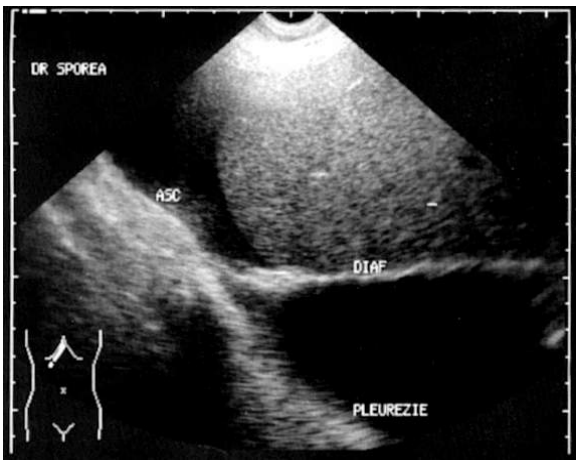


Fig. 45. Fluid in the right pleura and ascites

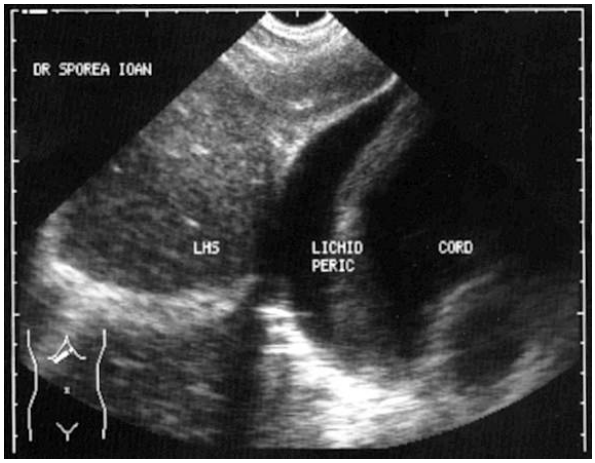


Fig. 46. Pericardial effusion

6. Budd-Chiari syndrome

Budd-Chiari syndrome is a clinical condition characterized by the thrombosis of the hepatic veins. It can be idiopathic, but it may a consequence of coagulopathies, myeloproliferative diseases, and neoplastic conditions. The diagnosis can be suspected in the presence of edema, ascites and hepatalgia with sudden onset.

In ultrasound the SHV cannot be viewed, partially or totally, due to the presence in their lumen of an echo dense material – thrombosis. In case of doubt we will use Doppler examination, which will show the absence of venous flow in the SHV.

More rarely, ultrasound will detect partial thrombosis, a solid like structure in the vascular lumen. of a hepatic vein. Also, the presence of thrombosis in the inferior vena cava can be detected by accident or in a clinical context. This thrombosis is more frequently found in renal or hepatic cancers.

Chapter 4.

Abdominal ultrasound in the diagnosis focal liver lesions – fluid lesions

Abdominal ultrasound is often the first imaging method performed in a patient for various complaints, whether about abdominal symptoms; or for the evaluation of a patient with a suspected or established chronic liver disease; for the follow-up of an oncology patient; or for the assessment of an individual with minimal abdominal trauma. In these circumstances often focal liver lesions are found, which we expect to find or not. Some of them have a typical aspect in standard ultrasound (biliary cysts, hydatid cysts, small hemangiomas), but more often the appearance of the lesion does not allow its definite diagnosis based only in gray scale ultrasound. In the latter case additional imaging investigations with contrast should be performed and when those are inconclusive, liver biopsy.

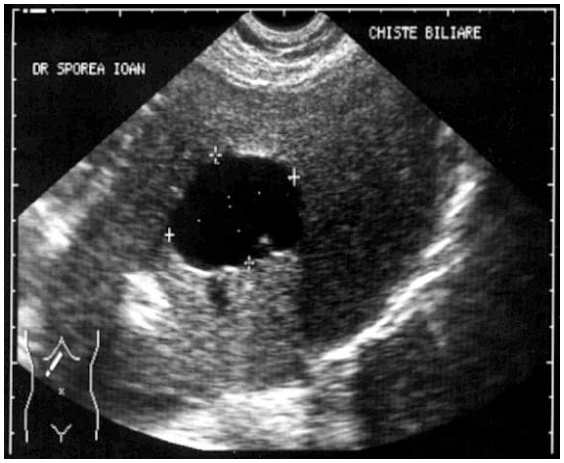
Below we will discuss the focal liver fluid lesions: simple hepatic cyst, polycystic liver, hydatid cyst, liver hematoma and liver abscess.

1. Simple liver cysts (or biliary cysts)

Liver simple cysts are non-parasitic, relatively common in clinical practice (1-3% of the ultrasound examinations performed). It is most frequently an incidental ultrasound finding, an "incidentaloma", its cause is the lack of communication of the bile ducts with the biliary tree.

The clinical signs in simple liver cysts are generally absent, very rarely they can generate symptoms, such as discomfort or pain in the right hypochondrium (large cysts or with intracystic hemorrhage).

The ultrasound appearance of hepatic cysts is usually typical, as an *anechoic lesion* with a very thin wall (sometimes not visualized by US), because they are lined by a single epithelial layer (Fig. 1, Fig. 2, Fig. 3, Fig. 4). The posterior enhancement is typical of all liquid structures and is due to the acceleration of ultrasound speed when passing from a solid environment (the liver) to the liquid environment of the cyst.



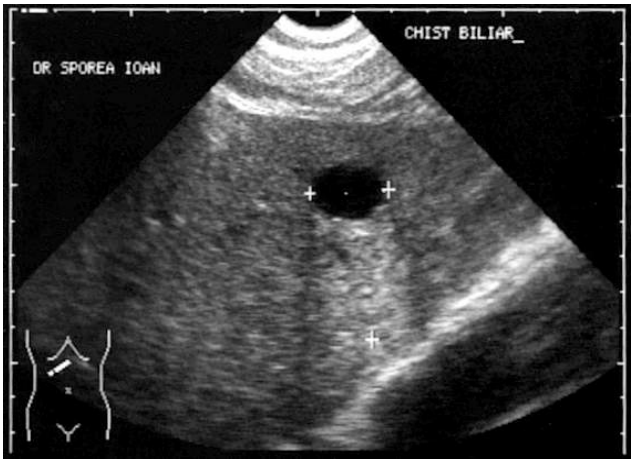


Fig. 1 and Fig. 2. Simple liver cyst: anechoic lesion with thin wall and posterior enhancement

The cysts' shape is round or oval, most commonly ranging in size from 1-5 cm, but may exceed this size sometimes reaching up to 15 cm. Biliary cysts may be single or multiple. The contour of the cyst is sometimes neat, often being irregular or "geographic" (Fig. 3). The appearance is most commonly perfect anechoic and fine septa may be present (Fig. 4). When the content is hypoechoic artifacts must be excluded and a differential diagnosis with tumors should be made.

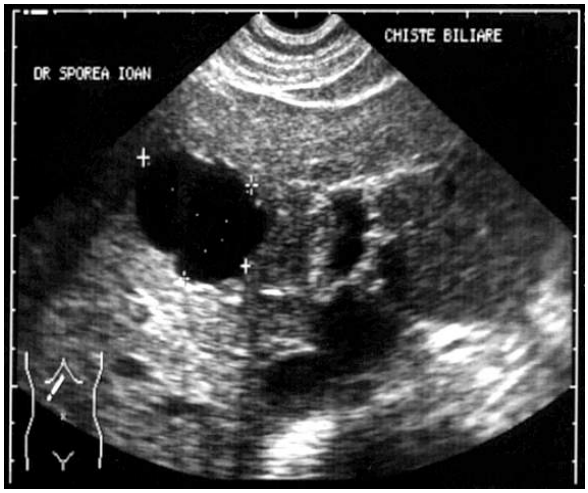


Fig. 3. Biliary cyst: anechoic lesion, with thin wall, geographic contour and posterior enhancement

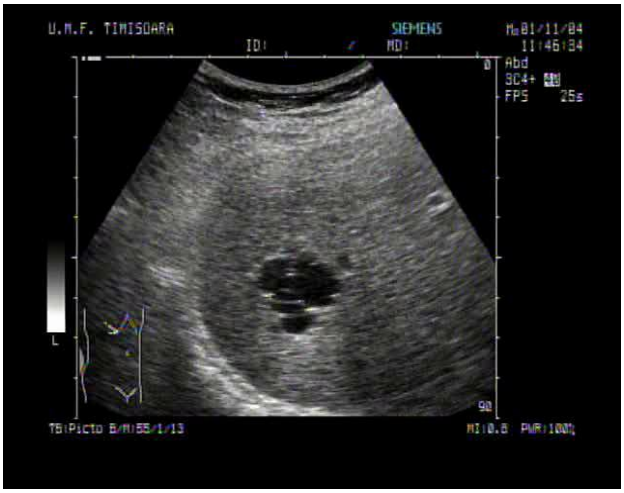


Fig. 4. Biliary cyst: anechoic lesion, with thin wall, geographic contour and septa

The differential diagnosis of bile cyst is made with*:*

- young hepatic hydatid cyst (a relatively frequent condition in Romania, which is an endemic area for this disease). In hydatid cysts the wall is thick, well defined, the contour is neat, and the lesion is round or oval, looking as a ping-pong ball (Fig. 5). Testing for Echinococcus antibodies in a good laboratory allows the differentiation of the two entities.

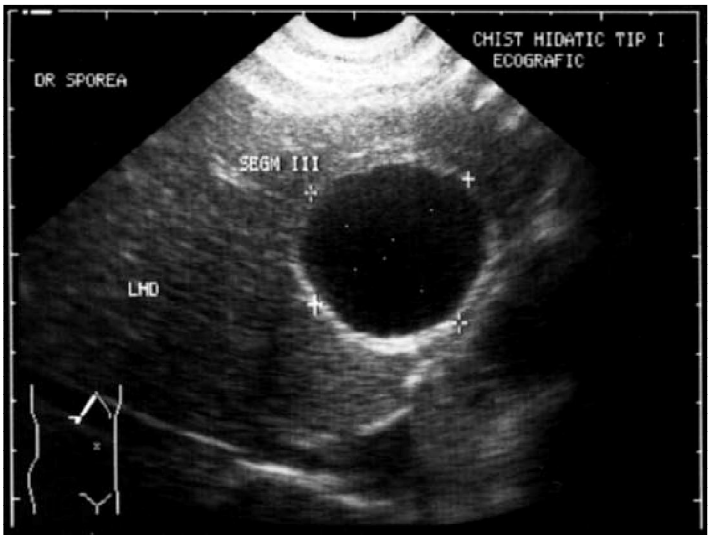


Fig. 5. Youg hydatid cyst: thick, well defined wall

- polycystic liver, the oligo-cystic form. In polycystic liver cysts characters are similar to those of simple biliary cysts, the only difference being the number and possible association with polycystic kidney (Fig. 6).

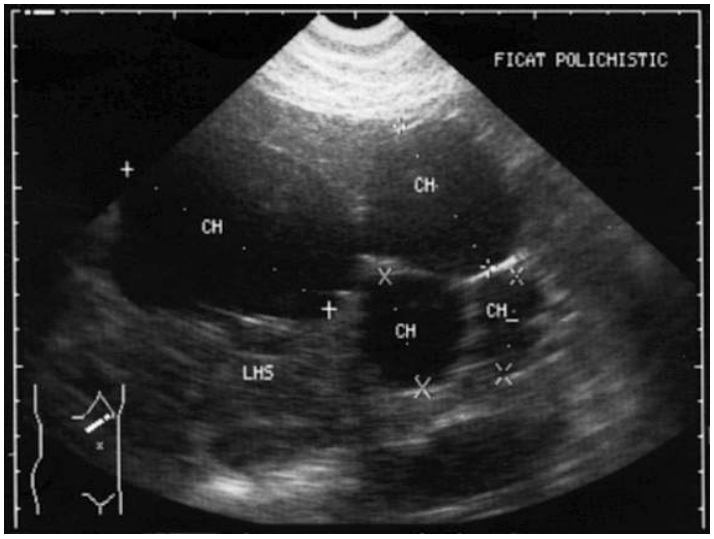


Fig. 6. Polycystic liver

- liver hematoma - it is found in a specific clinical context (post traumatic or after liver biopsy), sometimes accompanied by hemoperitoneum. The contour of is not so well defined and can be hypoechoic.

- liver abscess - it also occurs in a clinical context (patient with sepsis, fever, and leucocytosis). The outline is ill-defined, the content is often hypoechoic and the shape and size of the lesion changes from day to day (Fig. 7).

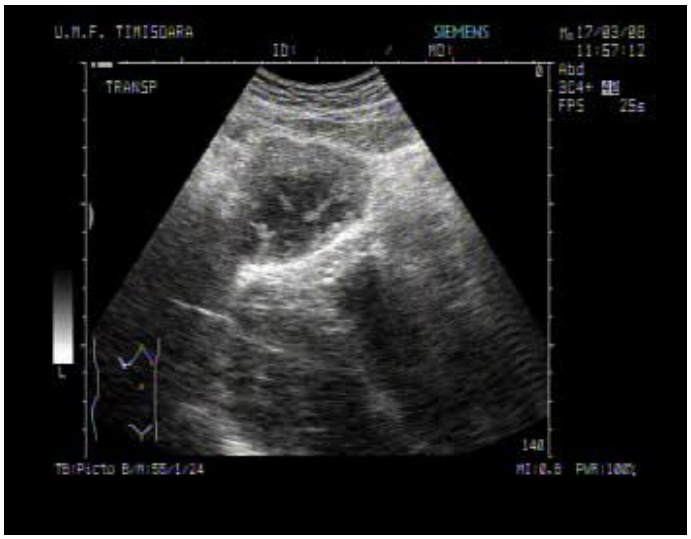


Fig. 7. Liver abscess

- gall-bladder or liver vessels caught in an incidence perpendicular to the lumen (Fig. 8). Ultrasound beginners are most likely to make such confusion. Careful examination which "follows" the structure, possibly Doppler examination that reveals blood flow in the vascular structure shall settle the diagnosis.

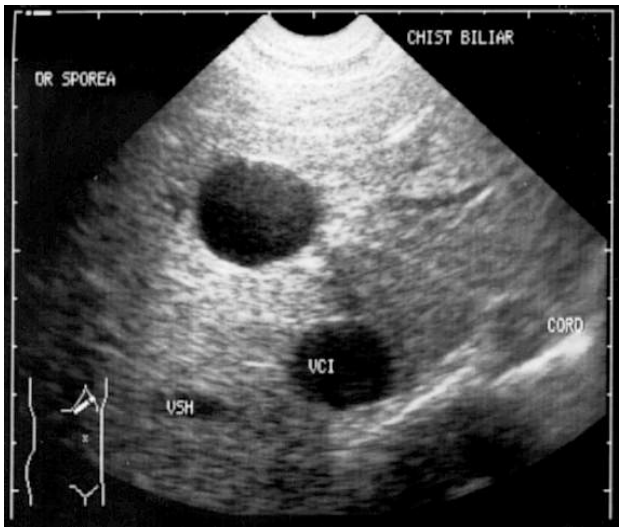


Fig. 8. Bile cyst, inferior vena cava and the right hepatic vein

- cystadenoma, cystadenocarcinoma and cystic metastases. The wall is thick, with protrusions that may exist inside (Fig. 9). In CEUS, both, wall and protrusion, will enhance during the arterial phase. In the late phases the cystadenoma wall remains enhancing, while in chistadenocarcinoma and cystic metastases wash-out will occur.

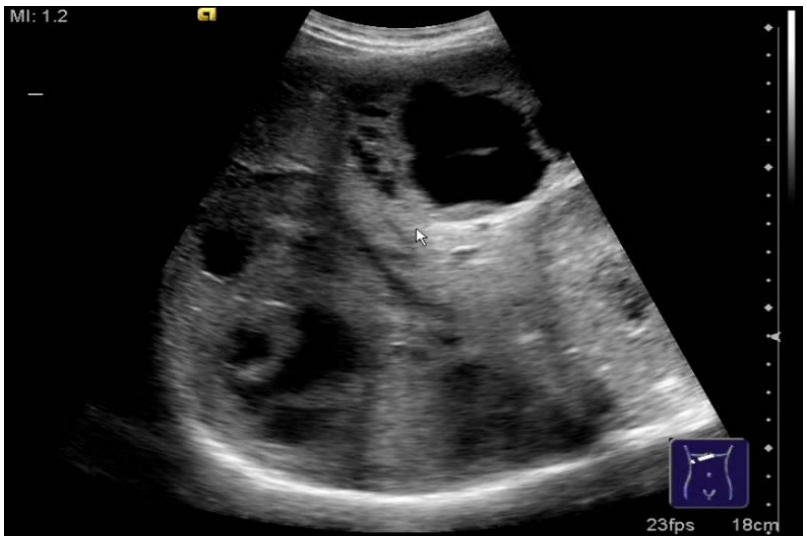


Fig. 9. Cystic metastases

- superior pole right kidney cyst - a careful examination will show the lesion's affiliation to the kidney, even if initially it seems to belong to the liver.

Rarely biliary cysts may be complicated by intracystic hemorrhage (post-traumatic or spontaneous), when the cyst, from anechoic can become hypoechoic. An exceptionally rare complication is cyst infection clinically translated by fever, chills and pain in the right upper quadrant. In ultrasound, the anechoic image of the cyst will have detritus and it can become hypoechoic or inhomogeneous.

Since biliary cysts are asymptomatic and, in most cases, with no risk of complications, they do not require therapeutic intervention, especially surgery. They will be followed periodically by ultrasound to see any increase (at the beginning biannual and then annually, or more rarely).

2. Polycystic liver

Polycystic liver is a congenital disease characterized by the presence of many cysts in the liver. Polycystic liver is frequently accompanied by polycystic kidneys, a congenital disease with dominant autosomal transmission. Usually the disease is detected by routine ultrasound, the patients being usually asymptomatic.

The ultrasound appearance of polycystic liver is typical, translating into multiple round or oval anechoic images (Fig. 10, Fig. 11), of variable sizes (Fig. 12), from 1 to 5-10 cm. There are cases of polycystic liver with a smaller number of cysts (5-20), which can even be counted - the oligocystic form. In other cases, an impressive number of cysts are present, which almost completely replace the normal liver structure.

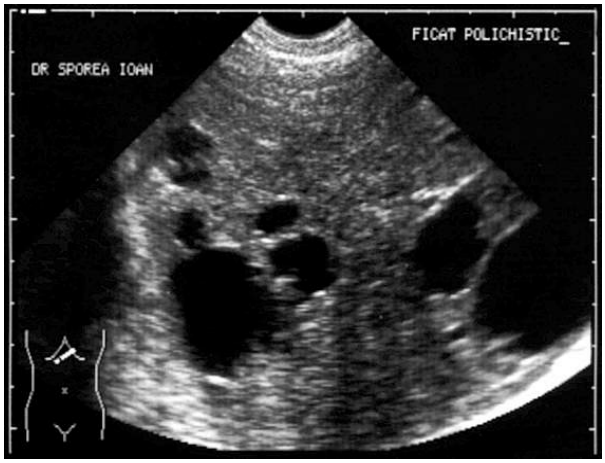


Fig. 10. Polycystic liver

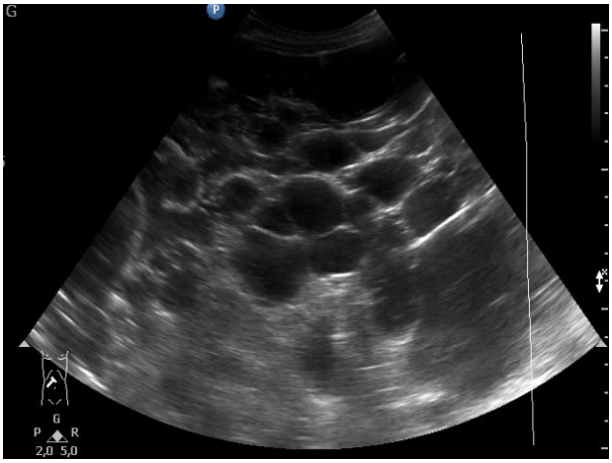


Fig. 11. Polycystic liver

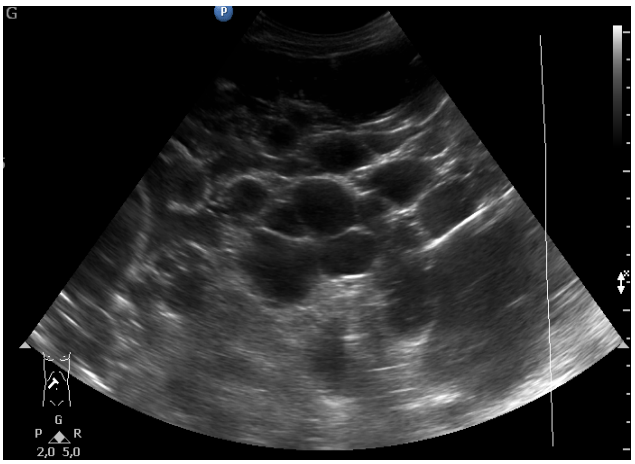


Fig. 12. Polycystic liver

On standard ultrasound, the cysts will have a completely anechoic (transonic) appearance, but sometimes inner septa can be found. The transonic appearance of the cysts will be replaced by hypoechogenity in cases with intracystic hemorrhage (which is sometimes possible) or in infected cysts, especially in a significant clinical context (pain, fever, leucocytosis, sepsis). The ultrasound aspect of the free liver parenchyma is perfectly normal.

The ultrasound aspect of the polycystic liver is typical, differential diagnosis being made rather in theory with giant septated hydatid cyst or with liver abscesses, with Caroli's disease, or, in rare cases, with multiple necrotic liver metastases.

The evolution of polycystic liver, unlike of polycystic kidneys, is completely "benign". In time, no signs of liver failure occur, and complications are exceptional (intracystic hemorrhage). The polycystic liver does not require treatment. In the case of symptoms generated by the increase of pressure in some cysts or intracystic hemorrhage, cyst decompression can be performed by ultrasound guided fine needle aspiration (0.6-0.7 mm needles).

3. Hydatid hepatic cysts

Hydatid hepatic cyst is a parasitic cyst generated by Echinococcus granulosus. Romania is an endemic area of hydatid cyst, which is why this disease is frequently found in current medical practice.

The modality of infection: parasite eggs are involuntarily swallowed (dirty hands, incompletely washed vegetables), after which they penetrate the intestinal wall and through the portal blood flow, the parasite reaches the liver. The most frequent location of hydatid cyst is the liver (in approximately 60% of the cases), followed by the lungs (approximately 20%).

The clinical signs are frequently absent, often being an accidental discovery, but complications can be severe (anaphylactic shock, cyst rupture in the biliary ducts with biliary obstructive jaundice and angiocholitis, secondary dissemination to multiple organs, etc).

The ultrasound appearance of hydatid cysts differs depending on the age of the cyst. Its main characteristic is the thick, well delimited wall (Fig. 13), often with thick septa inside (Fig. 14). The thick cyst wall is formed by the proligerous membrane, to which the layer formed by the compressed hepatic tissue is added. The germinal layer (protoscolexes) can be sometimes identified as a polypoid endomembrane of 0.5-1 cm. The daughter vesicles determine the septated aspect of the cyst.

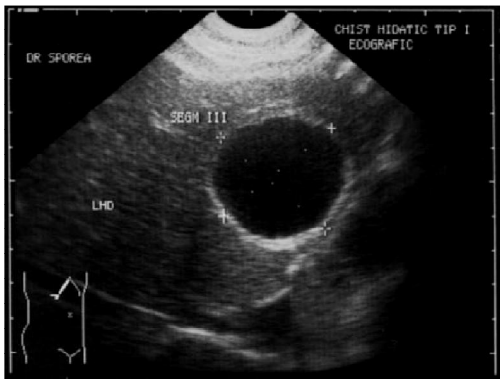


Fig. 13. Hydatid cyst – anechoic image with thick, well defined wall

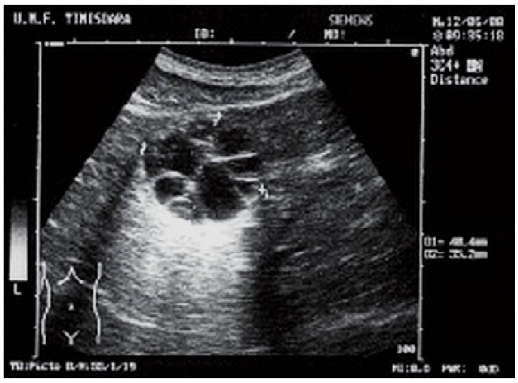


Fig. 14. Hydatid cyst – anechoic image with thick wall and daughter vesicles

Two main ***classifications*** of hydatid cyst are used, one by *Lewall and McCorkell,* which is simpler and divides hepatic hydatid cysts into 3 types depending on their aspect. The other classification, more complex, divides the ultrasound appearance of the hydatid cyst into 5 types and belongs to *Gharbi*. The two classifications have common elements, as shown in Table I. When formulating the diagnosis it is useful to clarify which classification was used.

Table I: Ultrasound classification of hydatid cysts

Ultrasound aspect	**Type according to the classification**	
	Lewall and McCorkell	**Gharbi**
completely anechoic hydatid cyst, with a well defined wall, without echoes inside (Fig. 13)	I	I
hydatid cyst with a detached proligerous membrane (echogenic band inside the cyst) (it can occur spontaneously or after therapy) (Fig. 15)	Subtype IR	II
– hydatid cyst with daughter vesicles, multiseptated cyst appearance (Fig. 14)	II	III
the hydatid matrix (magma) - a gelatinous structure resulting from dehydration of the hydatid fluid - hypoechoic or solid like appearance, cyst wall delimitation is obvious (Fig. 16, Fig. 17)		IV
old hydatid calcified cyst, highly hyperechoic hydatid wall, possibly with ”posterior shadow” or, sometimes ”the shell sign” – a highly echoic band that generates a marked posterior shadow (Fig. 18).	III	V

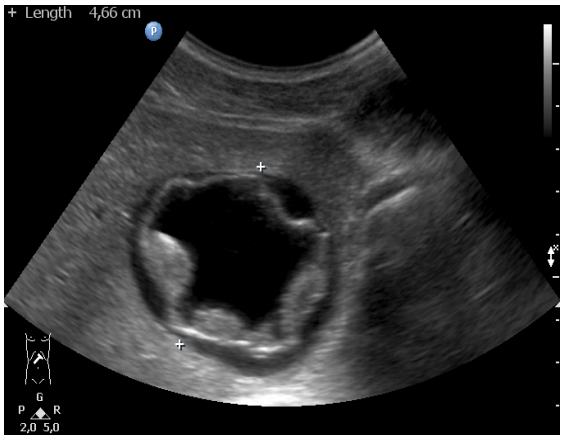


Fig. 15. Hydatid cyst with detached proligerous membrane

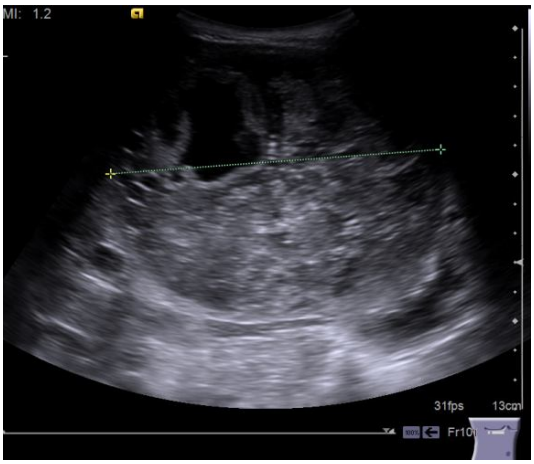


Fig. 16.Hydatid matrix

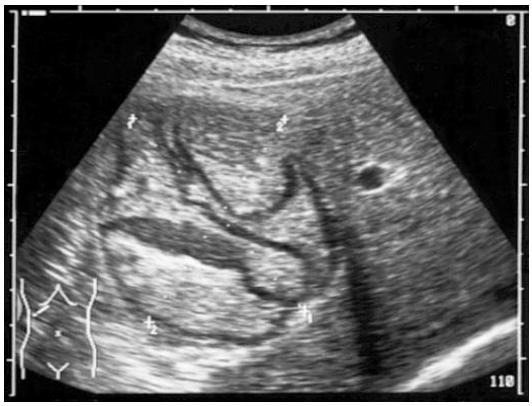


Fig. 17.Hydatid matrix

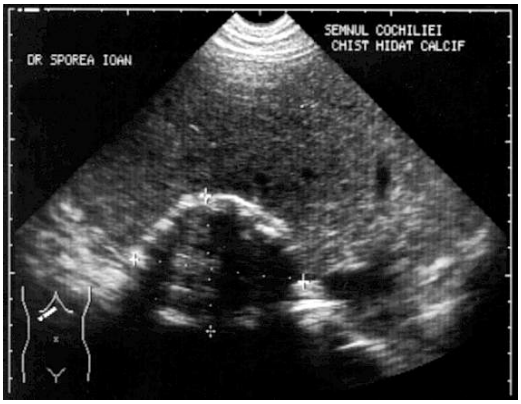


Fig. 18. Old hydatid cyst with calcifications

Differential diagnosis

A completely anechoic, *type I, hydatid cyst* must be differentiated from a simple hepatic cyst. Unlike a simple biliary cyst, the hydatid cyst has a thick wall (1-2 mm). Thus, the sensation of “inside strain” in hydatid cysts, while simple biliary cysts have a less clear-cut outline (”geographical outline”). The hydatid cyst wall frequently has a lamellar appearance (proligerous membrane + hepatic tissue displaced by the cyst growth), while the hepatic cyst wall seems to be absent. In cases requiring differentiation, anti-Echinococcus granulosus antibodies will be evaluated; the sensitivity is about 90%. In ultimate cases, ultrasound guided aspiration under protection with Hydrocortisone Hemisuccinate and Albendazole with a 0.6-0.7 mm needle will be made. The cyst will be always be punctured passing through normal hepatic parenchyma. An amount of fluid will be extracted and examined for the presence of scolexes and of the specific Echinococcus antigen. In hydatid cyst the extracted fluid is crystal-clear.

Hydatid cyst aspiration can be followed by complications that include rash, allergodermia and, very rarely, Quincke's edema.

Subtype IR hydatid cyst, with detached membrane, is a typical appearance for the hydatid cyst, and poses no problems for the differential diagnosis, as well as the *hydatid cyst with daughter vesicles.*

The *hydatid cyst with hydatid matrix* raises the most important problems regarding the differential diagnosis, since it should be differentiated from a solid hypoechoic tumor. The thick, visible cyst wall allows the differentiation and is the most useful element of ultrasound diagnosis. Other possible differential diagnoses are: cystadenoma or hepatic cystadenocarcinoma (both very rare), Echinococcus multilocularis infection, necrotic malignant primary or secondary tumors (metastases). Of real help is the contrast ultrasound (CEUS). Hydatid cysts will not enhance following contrast bolus, while tumors will enhance, with or without wash-out in late phases, according to histology type.

Type III hydatid cyst, with highly hyperechoic wall, with a posterior shadow or "shell sign", should be differentiated from a hepatic calcification (usually of small size, 1-3 cm), or from a scleroatrophic lithiasis gallbladder, but the "shell sign" is within the gallbladder projection area. Demonstration of parietal calcification is important for prognosis and treatment, these signifying that the parasite had died, thus there is no need for any treatment. The most sensitive method for highlighting calcifications is computer tomography (CT).

Treatment is addressed to viable hydatid cysts and can be medical, percutaneous or surgical. *Medical treatment* consists of Albendazole treatment in a dose of 800 mg/day for 30 days. Three courses of treatment of 30 days each, separated by a month pause, are recommended. The efficiency of therapy is checked by ultrasound, by monitoring cyst size (which decreases or remains unchanged), by membrane detachment, by cyst membrane disruption, by "aging" cyst appearance. Medical treatment is intended for young cysts, recently detected by ultrasound, or for postoperative recurrences.

Percutaneous treatment is addressed to young, perfect anechoic hydatid cysts and consists of the injection of sclerosing agents into the cyst, using 23 gauge (0.6 mm) needles. The patient is under conscious sedation with Midazolam. The ultrasound guided approach to the cyst is always through unaffected liver (in order to prevent the peritoneal rupture of the cyst). The cyst content will be completely aspirated, then hypertonic saline solution (50% or 20%) or, more frequently, 96° or 70° alcohol will be injected (the use of alcohol causes the sclerosis and consequently the effective destruction of the cyst). The solution injected into the cyst is left in place for approximately 10 minutes (even 20 minutes for saline solutions), after which the content is completely aspirated (under ultrasound guidance) (Pavia protocol). The complete sclerosis of the cyst wall is thus performed, which prevents fluid recurrence. This injection-

aspiration technique is termed PAIR (percutaneous aspiration-injection-reaspiration) and is an effective alternative to the surgery of hydatid cyst. The adverse reactions of this technique include: allergic reactions, fever, rarely liver abscess, biliary lesions, and vascular thrombosis.

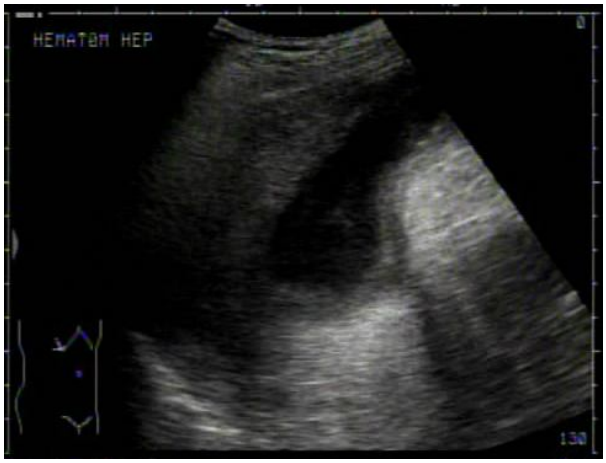
The percutaneous treatment of hepatic hydatid cyst must be performed under Albendazole protection (which is administered before aspiration, but also after percutaneous therapy, in 1-2 treatment courses). Generally, after 24 hours, the detached cyst membrane is seen, and follow-up at 1-2 months will reveal the disappearance of the cyst or its change into a hyperechoic lesion. The persistence or reappearance of a fluid lesion is considered as failure, due to the incomplete or insufficient treatment of hydatid cyst.

Surgical treatment is now rare and addresses to complicated cysts (ruptured, infected) and must be done under the Albendazole protection. Postsurgical ultrasound is important for the early diagnosis of relapse.

4. Liver hematoma

Liver hematoma is a collection of blood, intrahepatic or under the Glisson's capsule, as a consequence of trauma (a blow, fall, or road traffic accident) or after liver biopsy. It can remain strictly localized or it can open into the peritoneal cavity, with the appearance of hemoperitoneum. The clinical signs of liver hematoma are extremely variable, from asymptomatic, to pain in the right hypochondrium and hemorrhagic shock.

The ultrasound appearance of hepatic subcapsular hematoma is most commonly as a crescent shaped hypoechoic area (rarely anechoic or almost anechoic), located between the liver parenchyma and the Glisson's capsule. Intrahepatic hematoma is generally hypoechoic (sometimes almost anechoic) and can have different shapes and irregular margins (Fig. 19, Fig. 20). Sometimes liquid in the Douglas space is seen as a "dense" peritoneal effusion, which may be another argument to support the diagnosis. Not in all cases the anamnesis reveals a serious trauma, because sometimes, on the background of coagulopathy or in a pathological liver, a mild trauma can cause injury.



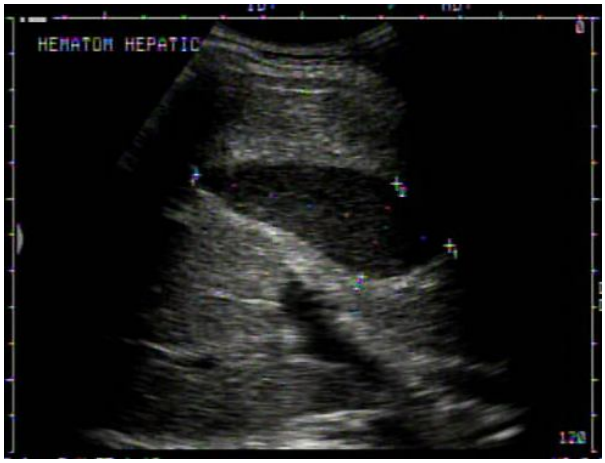


Fig. 19. Liver hematoma

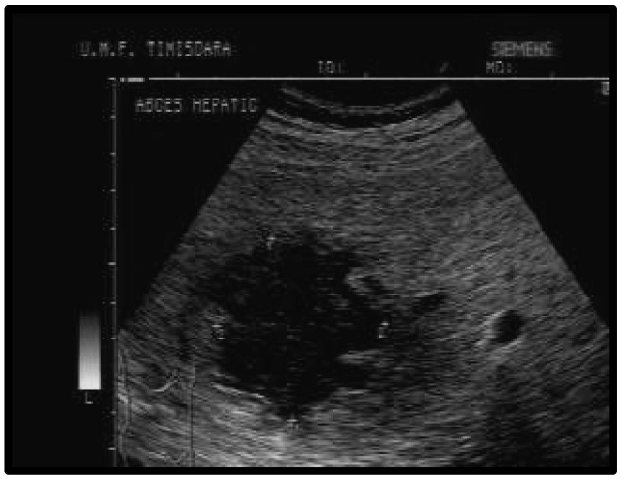
Fig. 20. Liver hematoma

Ultrasound differential diagnosis in intrahepatic hematomas is made with hypoechoic hepatic tumors, with liver abscess, with hepatic infarction, with hepatic lymphoma infiltration. The differentiation of subcapsular hematoma is made with perihepatic fluid collection (generally with ”dense” ascites). Ultrasound contrast is useful for diagnosis, highlighting the lack of enhancement in the hematoma following contrast. The visualization of even a few microbubbles in the hematoma or in the peritoneal fluid signifies active bleeding and requires reconsideration of treatment.

5. Liver abscess

Liver abscess is an intrahepatic pus collection. *The clinical signs* of liver abscess are mostly typical, with an altered general state, fever, chills, and a septic state, less common are mild symptoms, such as sub fever. Anamnesis may show an invasive procedure (ERCP) or surgery.

Standard ultrasound visualizes a hypoechoic mass, which is most frequently poorly delimited, inhomogeneous (Fig. 20, Fig. 21). It may show moderate posterior enhancement. Liver abscess is frequently highly inhomogeneous. In cases in which gas bubbles are formed, they appear as echoic structures moving with the patient’s position. Liver abscesses can be multiple, communicating or not.



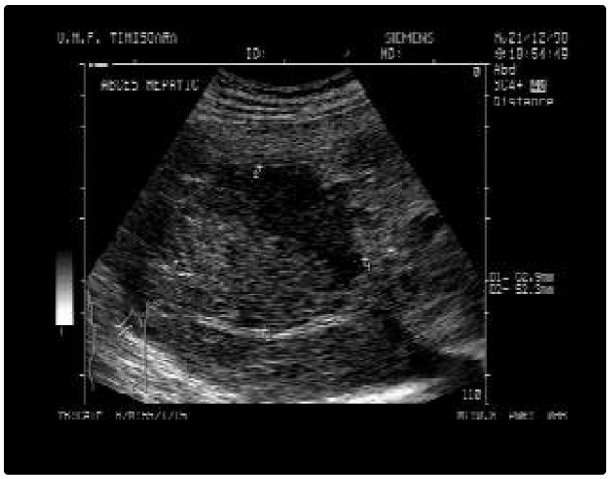


Fig. 20. Liver abscess

Fig. 21. Liver abscess

A lesion suspected to be an abscess can be evaluated using CEUS, the contrast agent will be captured in the inflammatory periphery of the lesion, but not inside it, which is avascular (SonoVue is an ultrasound contrast agent with strictly intravascular distribution). CT can help diagnosis by clarifying some aspects such as the density of the collection, the presence of air in the abscess.

Ultrasound differential diagnosis is required particularly in cases without obvious clinical symptoms when differentiation will be made with liver hematoma, liver tumors, hemorrhagic hepatic cysts, type II hepatic hydatid cyst.

In cases with an uncertain ultrasound diagnosis, the diagnostic and therapeutic method of choice is ultrasound guided puncture. Needle will be guided into the collection and the content will be aspirated. An antibiogram should be performed on the extracted pus or direct slide examination is possible.

The diagnosis of liver abscess must be followed by therapy, ideally by percutaneous drainage with pigtail drain tubes 10-15 F (3-5 mm) that will be placed under ultrasound guidance into the lesion, followed by continuous or discontinuous aspiration of the puss collection.

Monitoring of the residual cavity is also performed by ultrasound, possibly complemented by CEUS or CT. A diminution of the collection up to its disappearance will be seen, with a possible hyperechoic scar at the site of the resolved abscess.

6. Complex cysts

This category includes: bleeding biliary cysts; hydatid cysts with solid content, cyst-adenomas with solid content, cystic adenocarcinomas and metastases. The complex character of these cystic lesions results from the combination of anechoic areas, better or poorly delimited, with hypo or hyperechoic areas (either septa or protrusions, or both) in a liver lesion. Standard ultrasound alone usually cannot differentiate these lesions, so that a contrast imaging method is needed for the differential diagnosis (CEUS, CT or MRI).

Bleeding biliary cysts and hydatid cysts with solid content will not enhance following contrast in either vascular phase (Fig. 22 a, b, c, d). Also no change in the enhancement pattern of the lesion's wall will be seen in late phases.

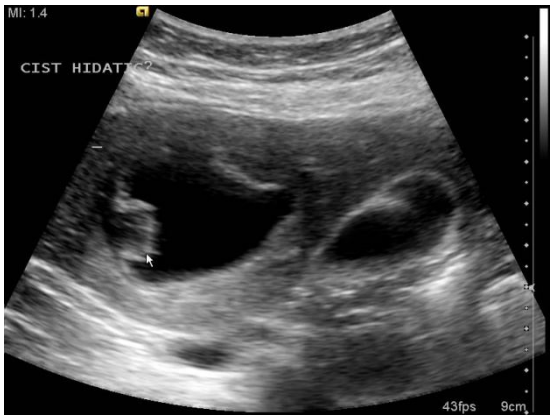


Fig. 22a

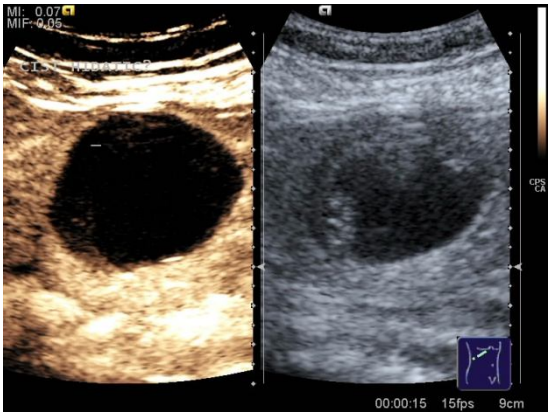


Fig. 22b

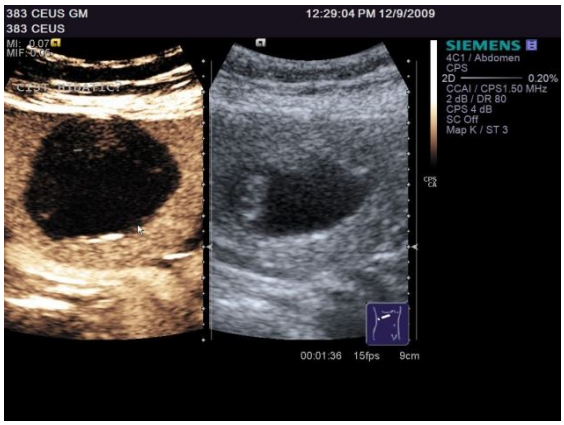


Fig. 22c

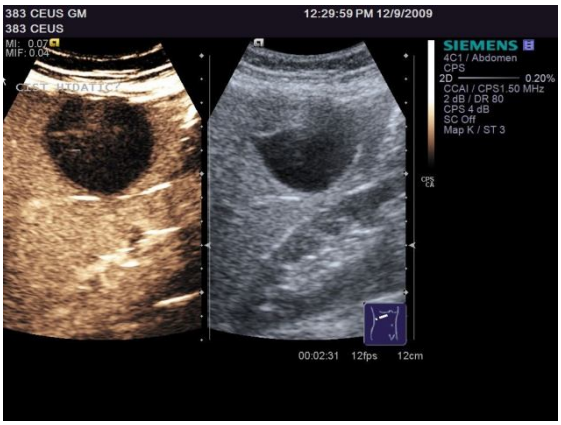


Fig. 22d

Fig. 22: a. Anechoic lesion, quite well defined, with obvious wall, with echoic protrusions inside. Following contrast bolus, the structure is clearly defined, and the protrusions do not enhance in the arterial (b), portal (c) or late phase (d). Final diagnosis: Hepatic hydatid cyst.

In **cystadenomas**, arterial enhancement of the capsule, septa and/or protrusions will be observed following contrast, which will remain hyperenhancing in the portal and late phases (Fig. 23. a, b, c). Cystadenomas are very rare benign liver tumors with high rate of recurrence. Due to the risk of malignancy (10%), they have surgical indication. In large tumors, unresectable, liver transplantation is indicated.

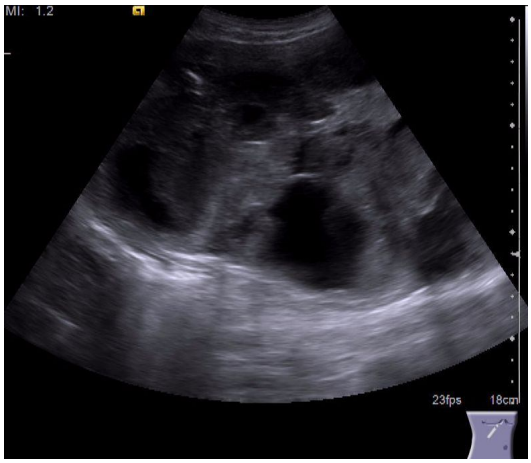


Fig. 23a

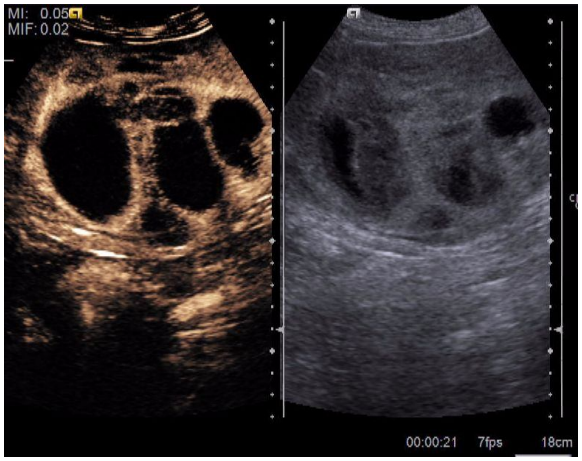


Fig. 23b

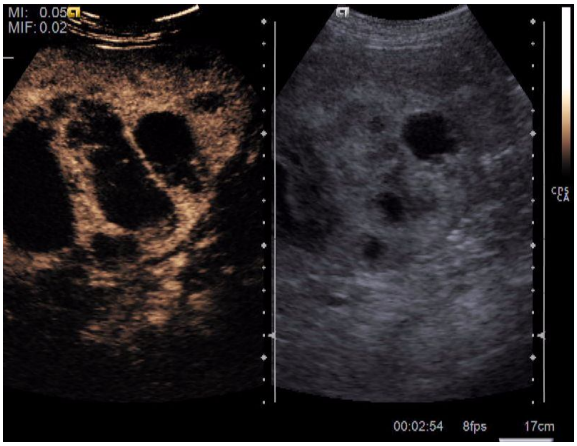


Fig. 23c

Fig. 23: a. Multiple anechoic, poorly defined lesions, with thick septa inside. Following contrast bolus, arterial enhancement of the septa and capsule is observed (b), which is maintained during the late phase (c).

In CEUS, cystic cystadenocarcinomas and metastasis may enhance during the arterial phase, but wash-out will occur in the portal and late phases, certifying the diagnosis of malignancy (Fig. 24 a, b, c). A possible starting point should be searched for (most commonly ovarian) and a diagnostic biopsy should be made.

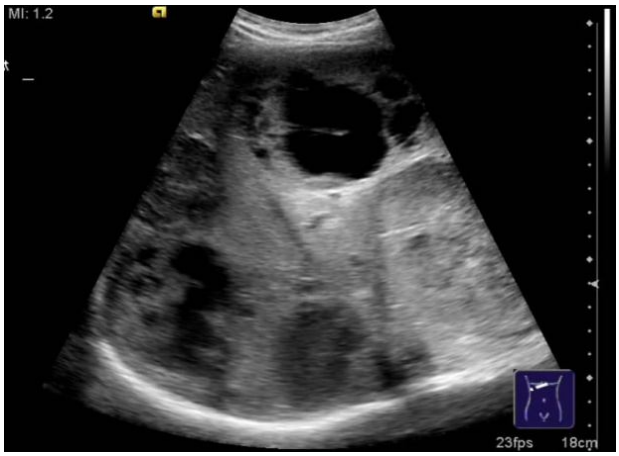


Fig. 24a

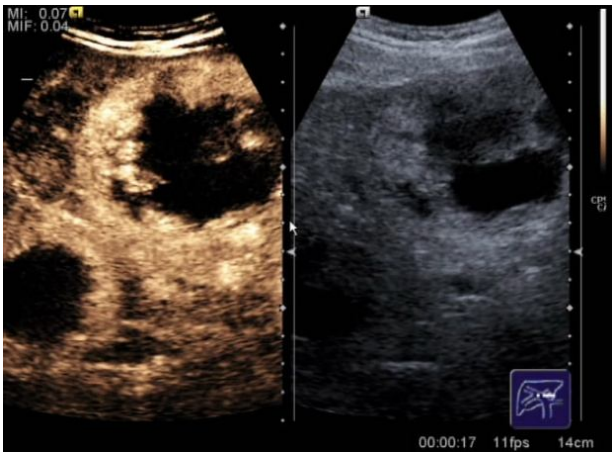


Fig. 24b

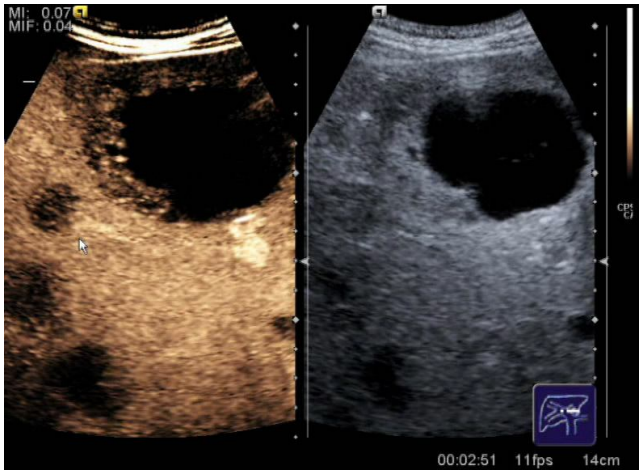


Fig. 24c

Fig. 24: a. Multiple anechoic lesions with thick septa inside and with thick, poorly defined wall. Following contrast bolus, arterial enhancement of the septa and capsule is observed (a), which is not maintained during the late phase and wash-out phenomenon occurs (c). The final diagnosis: Cystic metastasis after operated ovarian cancer.

Chapter 5.

Abdominal ultrasound in the diagnosis of focal liver lesions – solid lesions

As said before, abdominal ultrasound is often the first imaging investigation performed in a patient, during which often liver lesions are found, expected or not. After discussing cysts (or predominantly liquid) lesions, which are usually not very difficult to diagnose by an experienced ultrasonographist, we will discuss about the solid liver lesions in which the real problems of differential diagnosis occur. Firstly, we will study *benign* masses and will continue with *malignant* ones. The differential imaging diagnosis (by ultrasound, CEUS, CT or/ and MRI) is not always easy or possible. Sometimes ultrasound guided biopsy is needed for a final diagnosis.

1. Benign hepatic tumors

a. Hepatic hemangioma

Hepatic hemangioma is a benign vascular tumor, formed of capillary clusters and fibrous septa, and is considered to be a vascular malformation. It is the most common benign liver tumor, it can be single or multiple. Published data suggest a prevalence ranging between 0.4 - 7.3% (depending on how the diagnosis was established), more common in women than in men (ratio 5:1), and may be single or multiple. Most commonly hemangiomas are completely asymptomatic, being discovered incidentally on a routine ultrasound exam. In clinical practice they can be classified in: “normal” hemangiomas - diameter up to 5 cm, relatively common in daily practice and cavernous angiomas (more than 5 cm in diameter and with atypical ultrasound appearance).

The ultrasound appearance of hepatic hemangiomas is typical in 90% of the cases. The typical aspect is of a homogeneous, hyperechoic, well delimited mass (Fig. 1, Fig. 2). They often show posterior enhancement (due to the liquid blood content) (Fig. 3). The connective stroma that supports the vascular cluster generates the hyperechoic ultrasound appearance, despite the rich capillary fluid content (according to which we would expect for at least a hypoechoic if not anechoic appearance). Hemangiomas can imprint neighboring vascular structures without invading them.

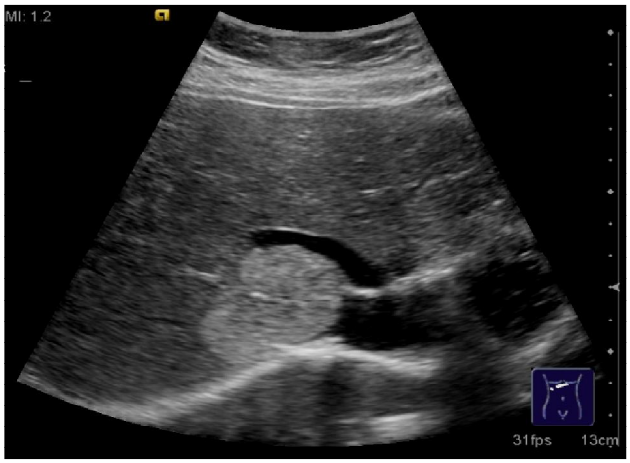


Fig. 1. Typical hemangioma with imprinting of PV

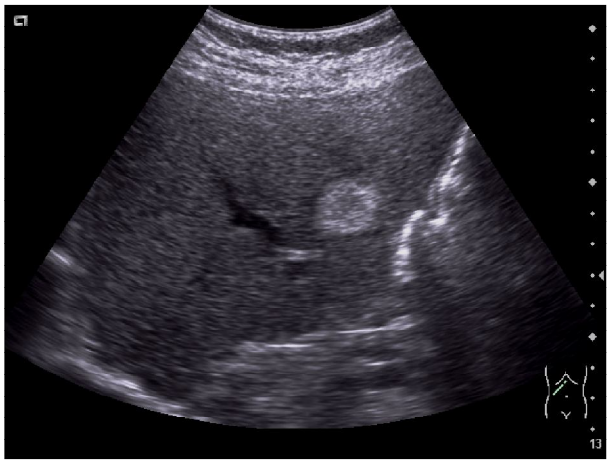


Fig. 2. Typical hemangioma

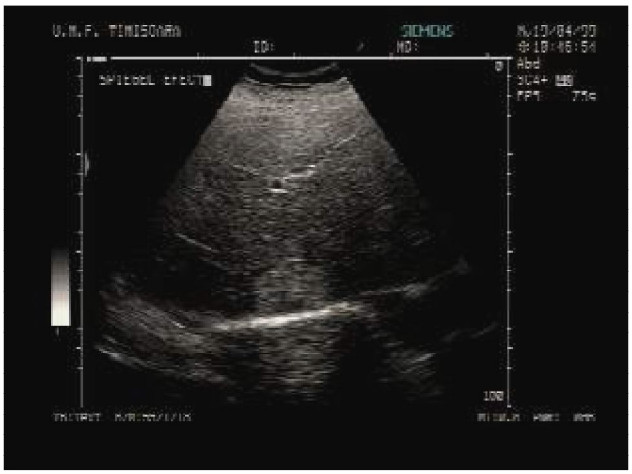


Fig. 3. Liver hemangioma with posterior enhancement

In up to 10% of cases, hemangiomas have *atypical appearance*, being hypoechoic or isoechoic (Fig. 4, Fig. 5), most commonly in hemangiomas larger than 3 cm. Another example is the occurrence of hemangiomas on a fatty liver, when they may appear as hypoechoic masses in a hyperechoic liver due to fatty overload. These cases are difficult to distinguish from other liver tumors. Elements suggestive for atypical hemangiomas are a clear delimitation, possibly as a fine hyperechoic margin (Fig. 4), vessel compression but not vascular invasion (Fig. 6).

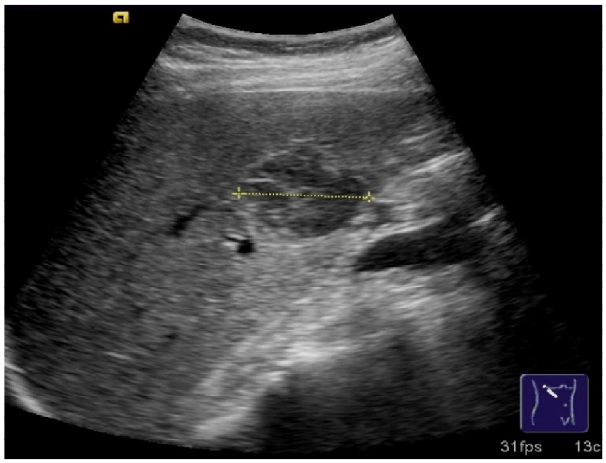


Fig. 4. Liver hemangioma - atypical aspect hypoechoic with a hyperechoic margin)

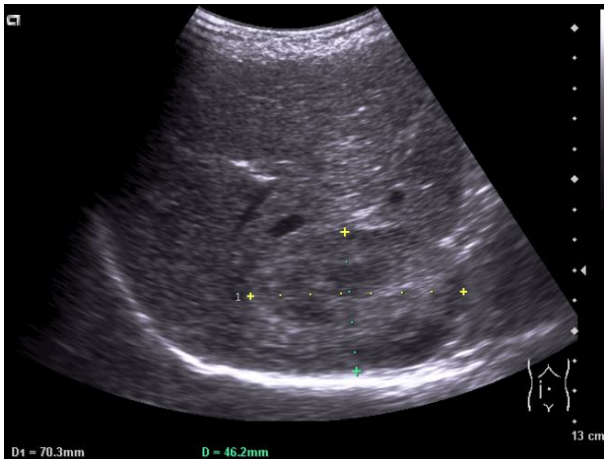


Fig. 5. Atypical hemangioma (inhomogeneous, isoechoic)

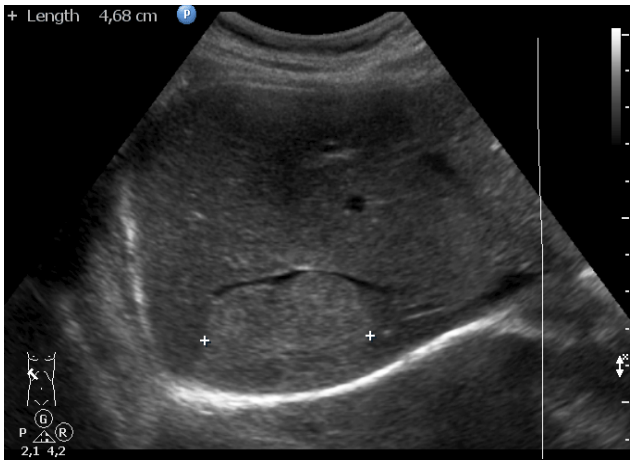


Fig. 6. Atypical hemangioma, isoechoic, compressing the right HV

With the introduction of CEUS, atypical hemangiomas have become easy to diagnose. The typical appearance of hemangioma in contrast is arterial nodular enhancement starting from the periphery to the center, that continues in the portal and late phases, with late homogenization of the mass (in cavernous angiomas areas of vascular thrombosis may exist in which the contrast agent will not penetrate) (Fig 7a,d,c,d). CEUS is diagnostic in approximately 90% of cavernous angiomas or atypical hemangiomas.

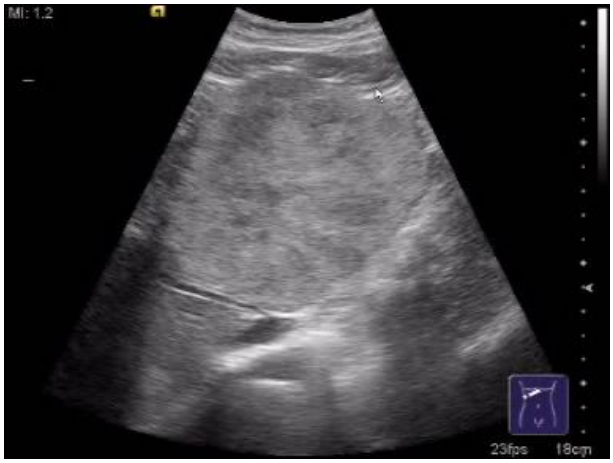


Fig. 7a

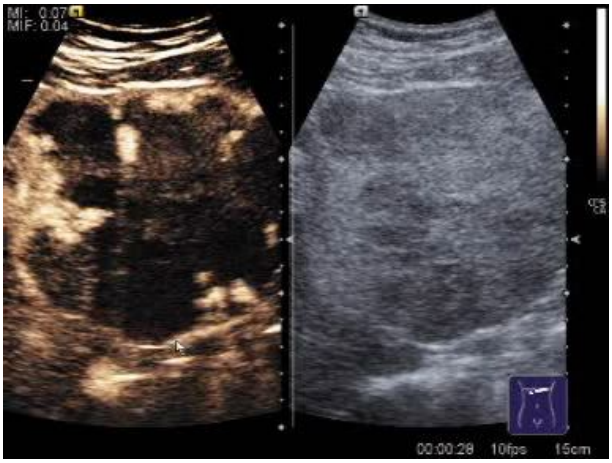


Fig. 7b

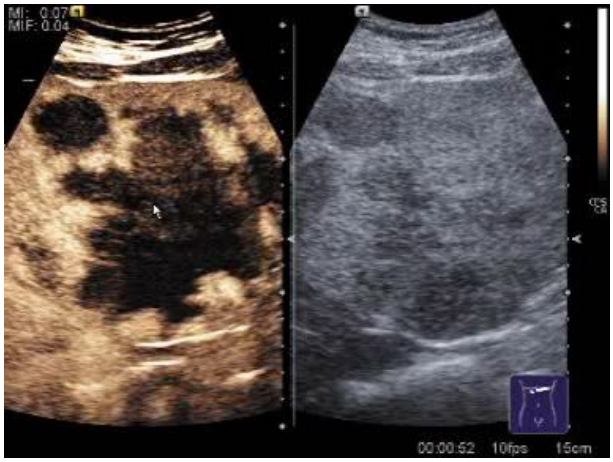


Fig. 7c

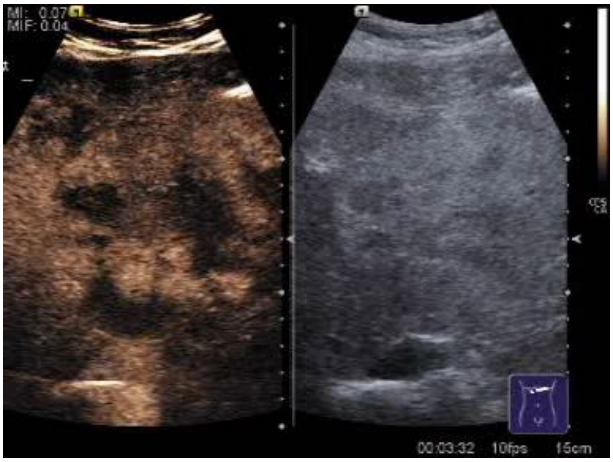


Fig. 7d

Fig. 7: a. Standard US – hyperechoic, inhomogeneous, well delimited mass in RLL.
b. CEUS arterial phase – arterial, nodular peripheral enhancement.
c. CEUS portal phase - peripheral centripetal enhancement continues.
d. CEUS late phase - mass still enhancing, with some areas that are unenhanced in all vascular phases.
Conclusion: Cavernous angioma.

On contrast enhanced CT, hemangiomas appear as hypodense images which enhance following contrast starting from the periphery to the center, becoming isoenhancing or hyperenhancing in late phases. This may compete with malignant tumors. Because of this, the ideal method for the diagnosis of hemangiomas is MRI, where they appear white and homogeneous in T2 sequence. An older method of diagnosis intended for masses larger than 5 cm (cavernous angiomas) is scintigraphy with labeled red blood cells with technetium-99 (SPECT scintigraphy), but probably today CEUS and MRI have replaced this method.

Once diagnosed, hemangiomas do not require a specific therapeutic intervention, they have to be just monitored. If ultrasound surveillance shows obvious growth trend, the diagnosis should be reconsidered (there is a small risk of confusion between hemangioma and malignant tumor). If non-invasive imaging methods are not sufficient, biopsy can be performed, always passing through healthy liver tissue to decrease the risk of bleeding.

Ultrasound differential diagnosis of hemangioma is made with: liver metastases (sometimes hyperechoic appearance in case of digestive adenocarcinoma), hepatocarcinoma, hepatic adenoma, focal nodular hyperplasia (FNH), patchy areas of steatosis or conversely, lack of fat load (“fatty free area") in a fatty liver.

b. Hepatic adenoma

Hepatic adenoma is a relatively rare benign hepatic tumor. Its origin is either in hepatocytes or in bile ducts. It is found in a normal liver, in 90% of cases in women, linked to the administration of oral contraceptives, and can often complicate with necrosis, intra-tumoral bleeding or spontaneous rupture, intrahepatic bleeding or hemoperitoneum.

The ultrasound appearance of hepatic adenomas is not typical, as it is a slightly hyperechoic liver tumor (Fig. 8), often inhomogeneous. In other cases it can be hypoechoic (Fig. 9), especially in patients with liver steatosis, or isoechoic (Fig. 10), in the latter case the diagnosis is suggested by the "bulge sign" (deformation of liver outline) or vascular impingement.

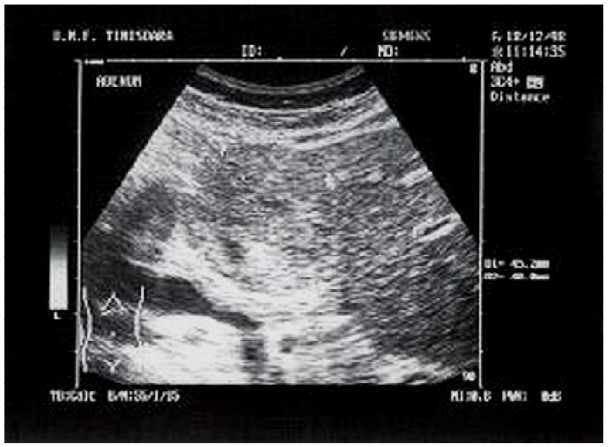


Fig. 8. Slightly hyperechoic hepatic adenoma

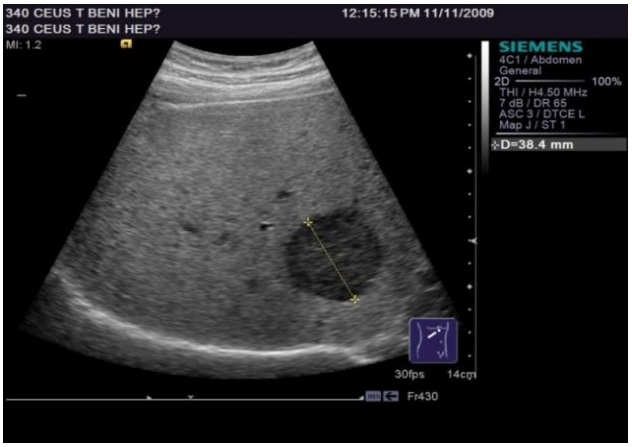


Fig. 9. Hypoechoic hepatic adenoma

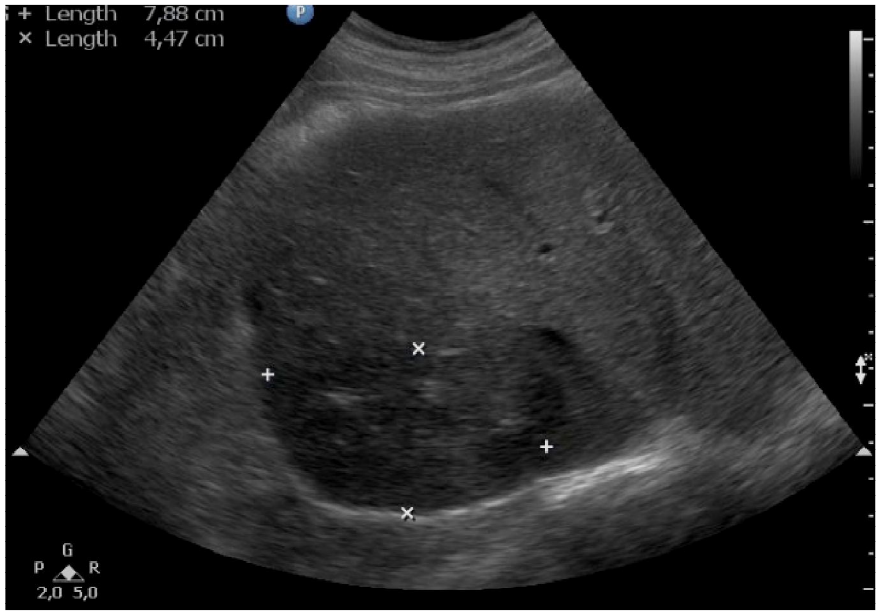


Fig. 10. Almost isoechoic hepatic adenoma

Only standard ultrasound cannot establish a definitive diagnosis of hepatic adenoma, additional diagnostic methods: CEUS, CT with contrast, MRI with contrast. Often imaging methods are not enough for diagnosis and ultrasound guided biopsy is needed, which will reveal normal liver cells.

CEUS enhancement has a sensitivity of 50-70% for the diagnosis of hepatic adenomas, the typical appearance is rapid, homogeneous, arterial enhancement, with the persistence of the contrast in the lesion in the portal and late phases (certifying its benign character) (Fig. 11 a, b, c, d). In cases with an unclear diagnostic, all efforts will be made for a definite diagnosis, considering the risk of spontaneous rupture and bleeding (30%) of hepatic adenomas, and the possibility of malignant degeneration. Once the diagnosis of adenoma is established, especially if it is a large lesion, the tumor should be surgically removed.

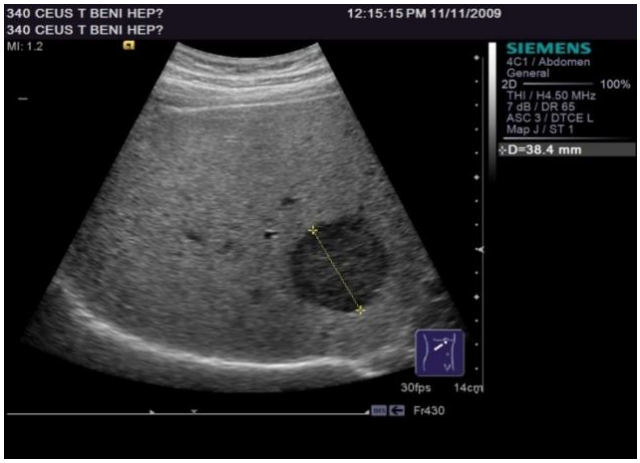


Fig. 11a

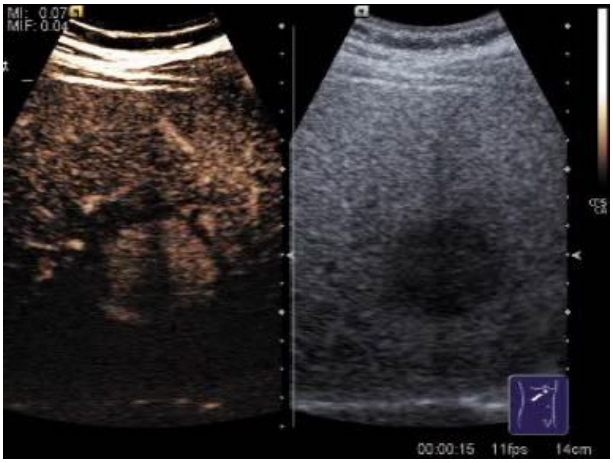


Fig. 11b

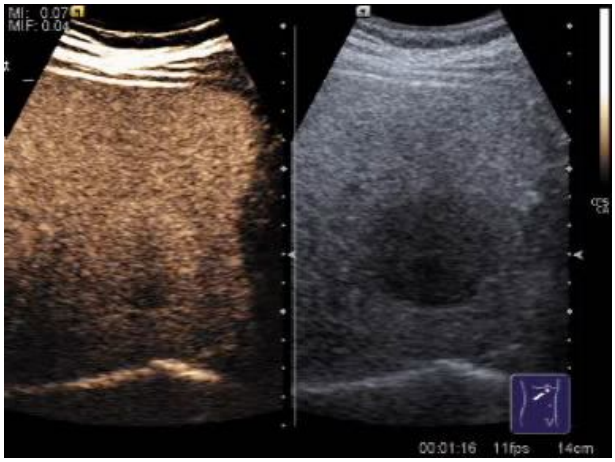


Fig. 11c

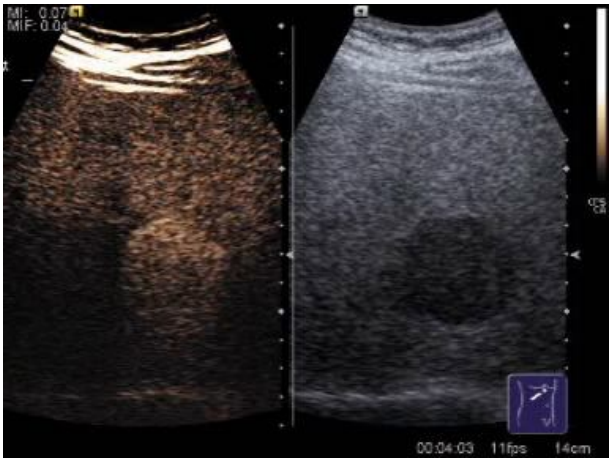


Fig. 11d

Fig. 11: a. Standard US – hypoechoic, homogeneous, well delimited lesion in LLL in a fatty liver. b. CEUS arterial phase – rapid, homogeneous, intense arterial enhancement c. CEUS portal phase – hyperenhancing lesion. d. CEUS late phase - hyperenhancing lesion. Conclusion: Benign liver lesion, with high probability adenoma.

c. Focal nodular hyperplasia (FNH)

Focal nodular hyperplasia is a benign liver tumor, quite frequently encountered in up to 3% of the population, more common in women than in men (ratio 4:1) associated with chronic consume of oral contraceptives. It is actually a hyperplastic hepatocytes regeneration area, secondary to a congenital vascular anomaly. The typical imaging elements of FNH are its arterialization and the central fibrous scar. From the clinical point of view, the FNH is frequently asymptomatic, being an incidental ultrasound finding.

The standard US appearance is not typical, often it is isoechoic (Fig. 12), or it may be discretely hyperechoic (possibly due to mild fatty loading) or discretely hypoechoic (especially in the context of diffuse hepatic steatosis) (Fig.13). Ultrasound demarcation is not always very clear. FNH's typical central scar (Fig. 14) is rarely visible by standard ultrasound. Using Power or color Doppler sometimes may show a typical vascular pattern (multiple, regular, radial vessels with spoke wheel pattern) (Fig. 15).

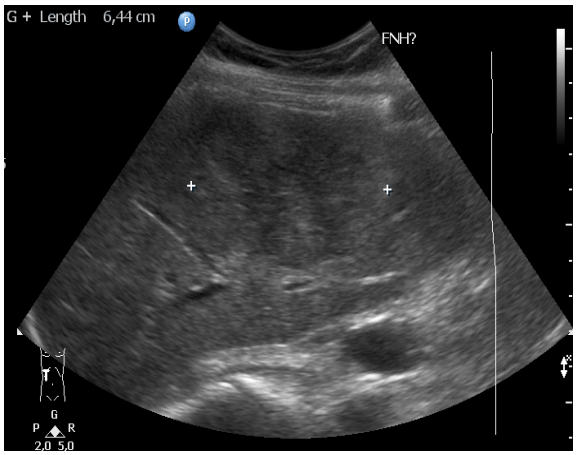


Fig. 12. FNH – isoechoic, inhomogeneous nodule

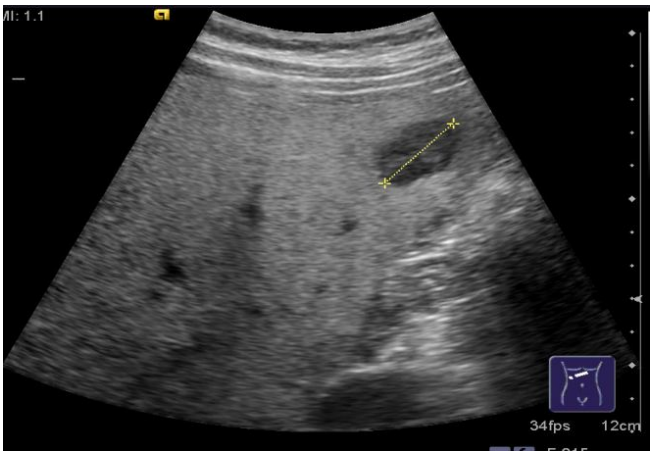


Fig. 13. FNH – hypoechoic nodule on a fatty liver

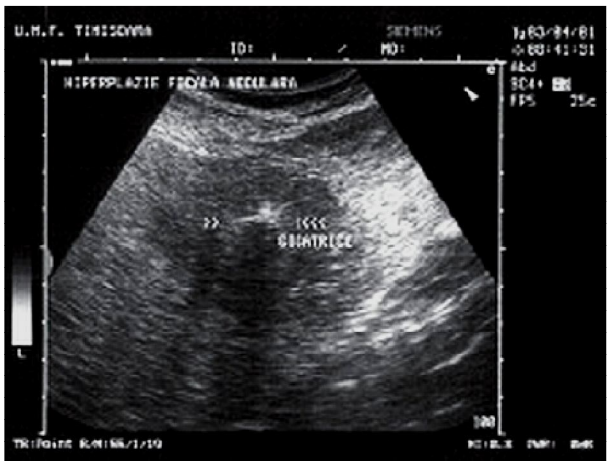


Fig. 14. FNH – central scar

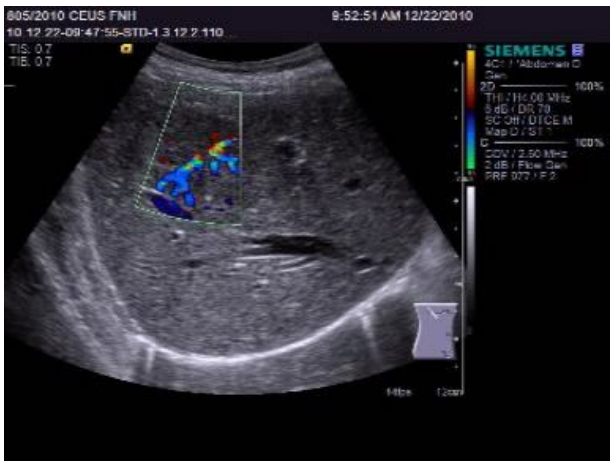


Fig. 15. FNH – Doppler (spoke wheel pattern)

CEUS is a very good method for the diagnosis of FNH, with a specificity surpassing 93%. The typical aspect is lesion with rapid arterial enhancement (10-15 seconds) sometimes with a typical "spoke wheel" pattern. The lesion will remain hyperenhancing in the portal and late phases (Fig. 16 a, b, c, d). In cases with inconclusive CEUS, CT or MRI with contrast will be performed and, maybe, morphological evaluation.

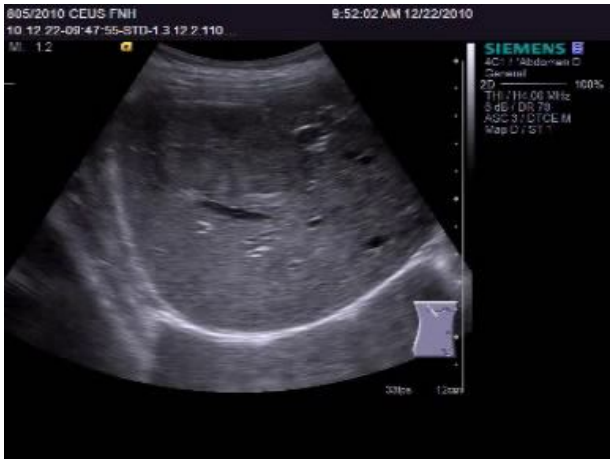


Fig. 16a

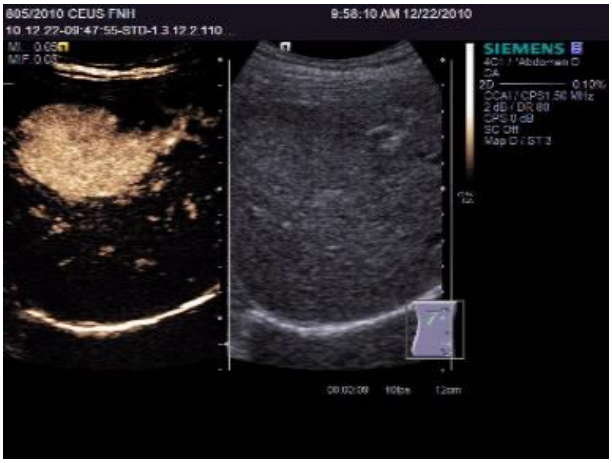


Fig. 16b

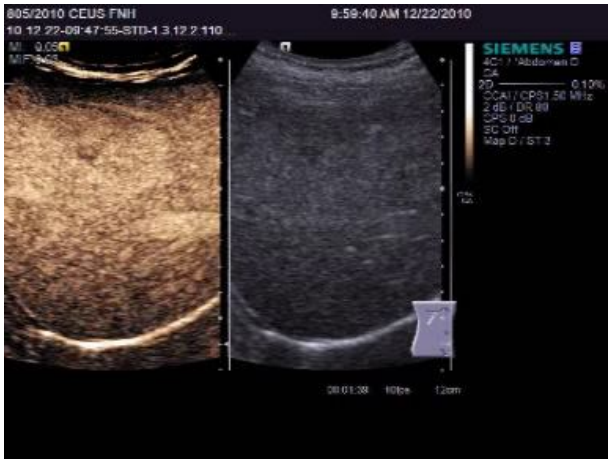


Fig. 16c

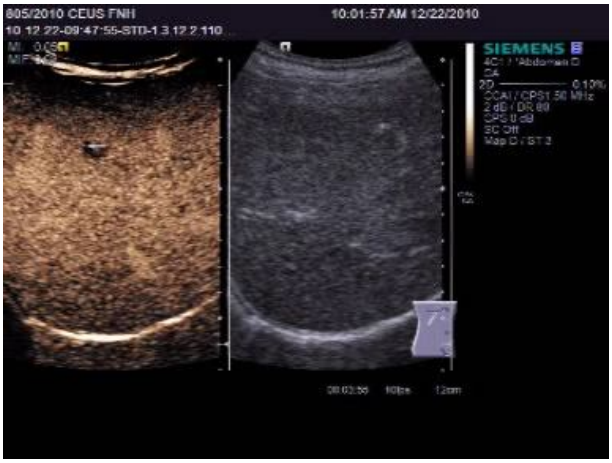


Fig. 16d

Fig. 16: a. Standard US – slightly hypoechoic, inhomogeneous, well delimited lesion in LLL.
b. CEUS arterial phase – rapid, complete, homogeneous arterial enhancement with the visualization of the nutritive artery (right side of the image),
c. CEUS portal phase – slightly hyperenhancing mass with visualization of the central scar (unenhancing central area).
d. CEUS late phase - slight hyperenhancing mass with central scar.
Conclusion: focal nodular hyperplasia.

After a definite diagnosis of FNH is established (most often by CEUS) the patients should interrupt the consumption of oral contraceptives and FNH should be followed-up by ultrasound.

d. Focal fatty liver and fatty-free areas

Focal fatty liver and fatty-free areas are particular situations quite commonly encountered, practically different fat loaded areas in the liver. The standard US appearance is of juxtaposition of areas with different echogenity into the liver: hypoechoic areas in a hyperechoic liver in fatty-free areas (Fig. 16, Fig. 17), or hyperechoic areas in a normoechoic liver in focal fatty lesions (Fig.18). Delimitation of these areas is the often neat, frequently with a geographical contour and variable sizes. It never alters the hepatic surface nor infiltrates or invades the vascular structures.

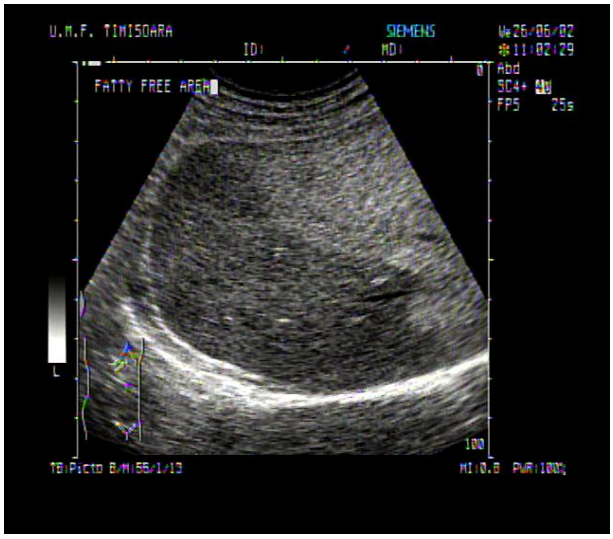


Fig. 16 Fatty-free area in RLL

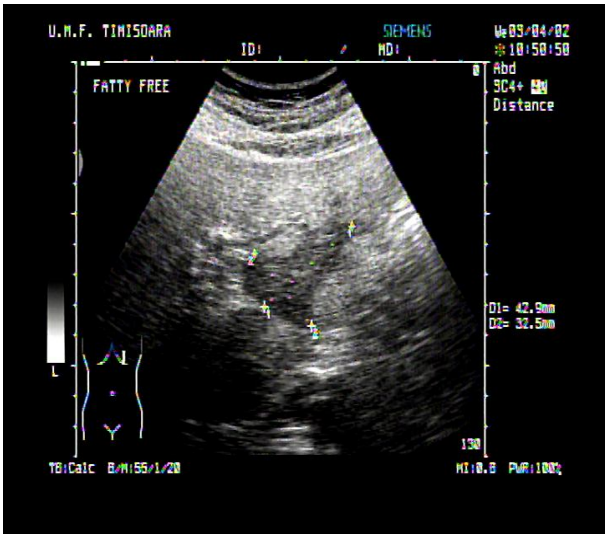


Fig. 17. Fatty-free area in LLL

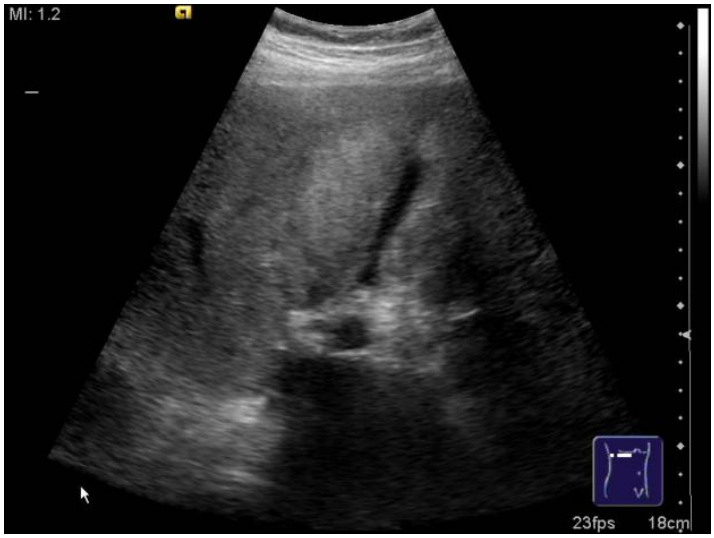


Fig. 18. Focal fatty lesion, surrounding the gallbladder

The differential diagnosis of fatty-free areas, respectively of focal fatty lesions can be difficult, whereas primitive or secondary liver tumors appeared on a fatty liver should be excluded. This cannot be done only by standard ultrasound and contrast imaging methods are needed: CEUS or CT or MRI. On CEUS fatty free areas, respectively focal fatty lesions will enhance similar to the surrounding liver parenchyma in all vascular times (Fig. 19 a, b, c, d; Fig. 20 a, b , c, d).

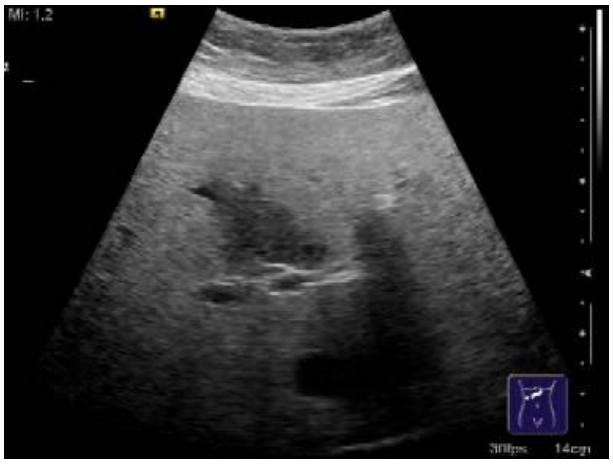


Fig. 19a

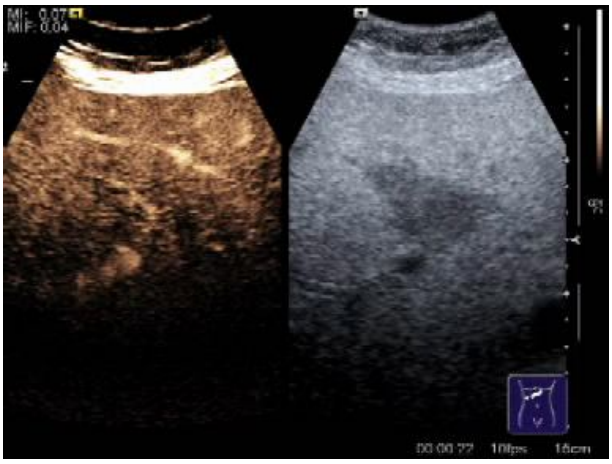


Fig. 19b

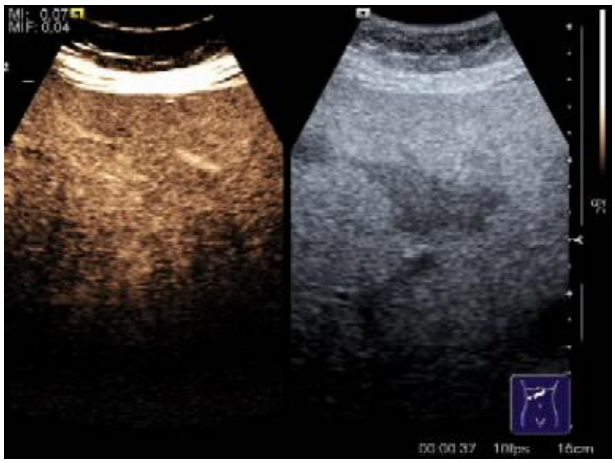


Fig. 19c

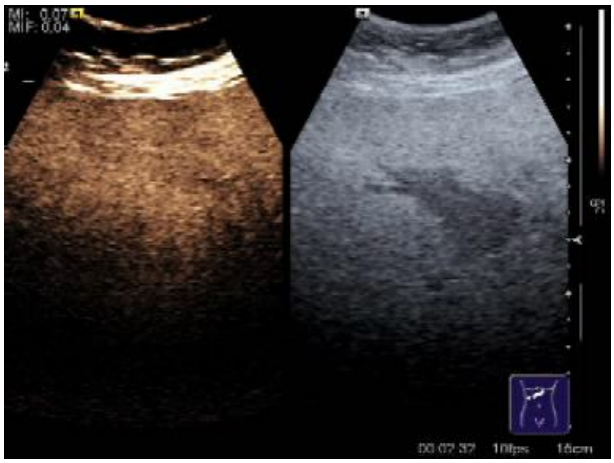


Fig. 19d

Fig. 19: a. Standard ultrasound – hypoechoic, homogeneous, well delimited area with geographical contour in RLL.
b. CEUS arterial phase – the area enhances similarly to the rest of liver parenchyma.
c. CEUS portal phase – the area enhances similarly to the rest of liver parenchyma.
d. CEUS late phase – the area enhances similarly to the rest of liver parenchyma.
Conclusion: Fatty-free area

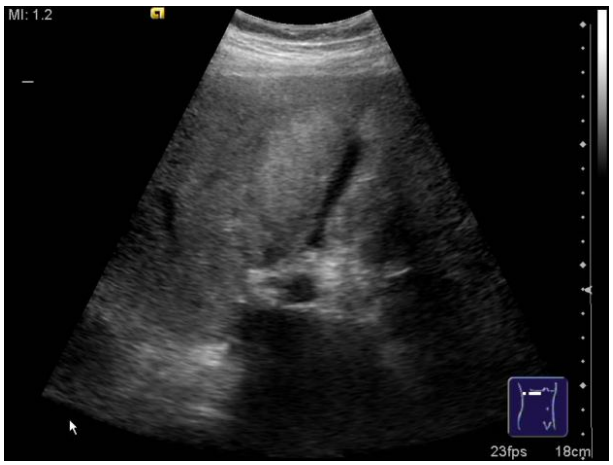


Fig. 20a

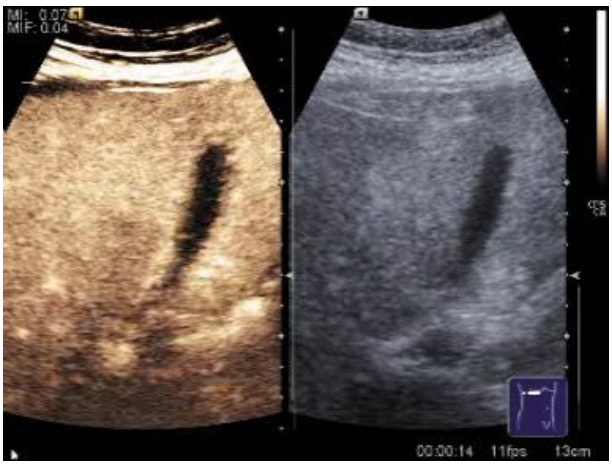


Fig. 20b

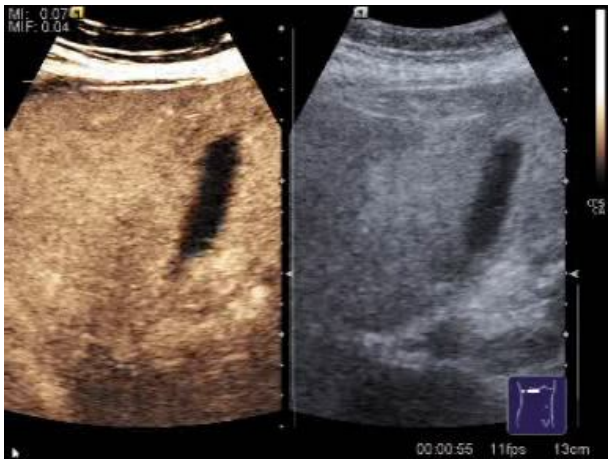


Fig. 20c

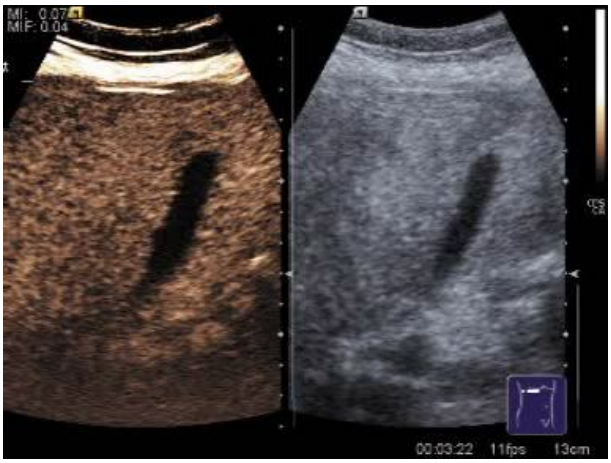


Fig. 20d

Fig. 20: a. Standard ultrasound – hyperechoic, well delimited area, with geographic contour surrounding the gallbladder.
b. CEUS arterial phase – the area enhances similarly to the rest of liver parenchyma.
c. CEUS portal phase – the area enhances similarly to the rest of liver parenchyma.
d. CEUS late phase - the area enhances similarly to the rest of liver parenchyma.
Conclusion: Focal fatty lesion

2. Malignant hepatic tumors

The most frequent malignant hepatic tumors are: hepatocellular carcinoma, cholangiocarcinoma, and liver metastases. It is very difficult to decide based only on standard ultrasound appearance of the tumor if it is benign or malignant. In these cases a contrast imaging method (CEUS, CT or MRI) or biopsy with pathological exam will make the difference. The same difficulty occurs in differentiating a primary from a secondary tumor (metastasis) by ultrasound. A contrast imaging or pathological examination will differentiate them.

a. Hepatocellular carcinoma (HCC)

HCC is the most frequent malignant primitive liver tumor (75-80% of total) with the starting point in the hepatocytes, usually occurring on a background of cirrhosis (94% of cases), more rarely on a background of severe chronic viral hepatitis, of alcoholic hepatitis, of primary biliary cirrhosis, NASH, or hemochromatosis.

Considering all the above, the clinical approach when a hepatic mass that can be a HCC is discovered is different depending on the presence of a concomitant chronic liver disease (particularly liver cirrhosis). In principle, any new nodule detected on a background of cirrhosis should be considered HCC until proven otherwise. When clinical signs of liver cirrhosis are found: "spider naevi" on the chest, abdominal collateral circulation, firm hepatomegaly associated with ultrasonographic signs of cirrhosis (described in detail in the chapter of hepatic diffuse diseases), with elevated liver stiffness assessed by elastography (FibroScan, ARFI) with biological signs of liver injury, with or without esophageal varices at endoscopy, the diagnosis of cirrhosis is relatively simple and therefore the suspicion of HCC is high. If the alpha fetoprotein (AFP) is increased to values above 200-400 ng/ml (diagnostic for HCC) we have an additional argument for the diagnosis of HCC. Unfortunately, only about one third of HCCs have pathognomonic values of AFP, even in large liver tumors.

The standard ultrasound appearance of HCCs can be hypoechoic (Fig.21), hyperechoic (Fig. 22), isoechoic or as a “bull’s eye” appearance (with a peripheral hypoechoic halo) (Fig. 23). No aspect in standard ultrasound is typical. Generally (but not as a rule) small HCCs appear hypoechoic. Large HCCs (more than 5-7 cm) are inhomogeneous due to tumor necrosis that occurs by intratumoral hemorrhage.

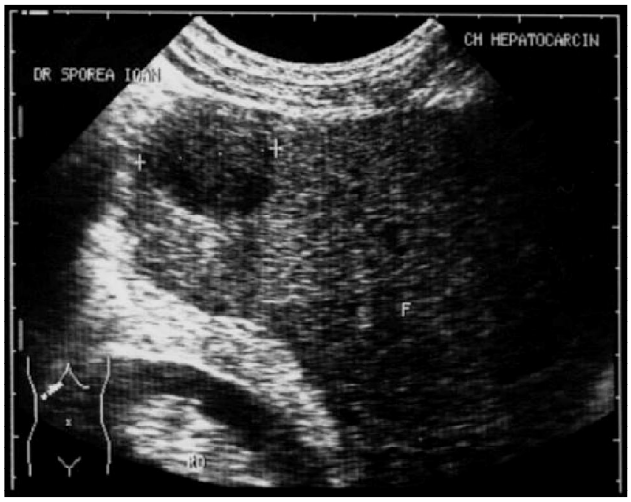


Fig. 21. Hypoechoic HCC

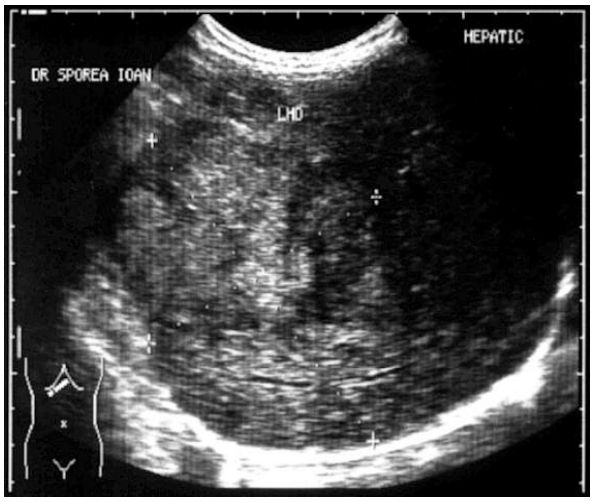


Fig. 22. Isoechoic, inhomogeneous HCC

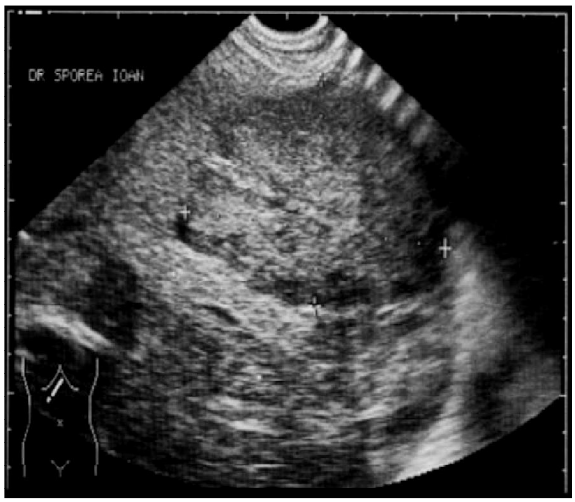


Fig. 23. HCC with "bull's eye" appearance

The differential diagnosis of HCC is based on contrast imaging (CEUS, CT, MRI). Typical for HCC is the arterialization of the nodule, followed by wash-out in portal and/or late phases (Fig.24 a, b, c, d). Wash-out occurs in most cases in late parenchymal phase, but some HCC are isoenhancing in the late phase (about 30% - the well-differentiated ones). CEUS sensitivity for the diagnosis of HCC is 80-85%, and in unclear cases spiral CT or MRI, both mandatory with contrast, should be performed.

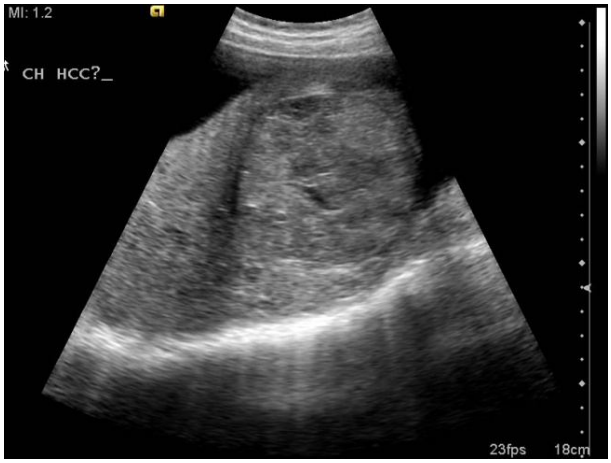


Fig. 24a

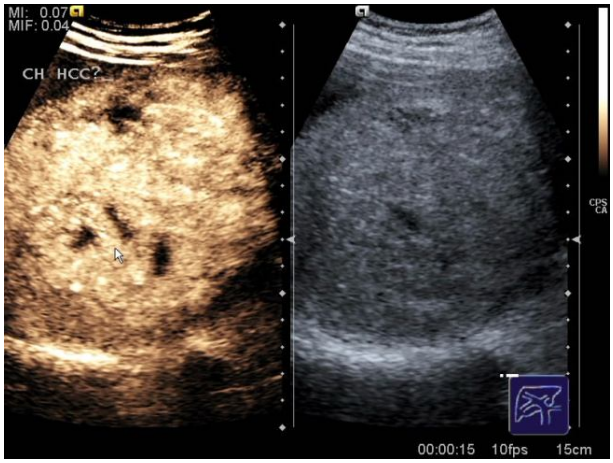


Fig. 24b

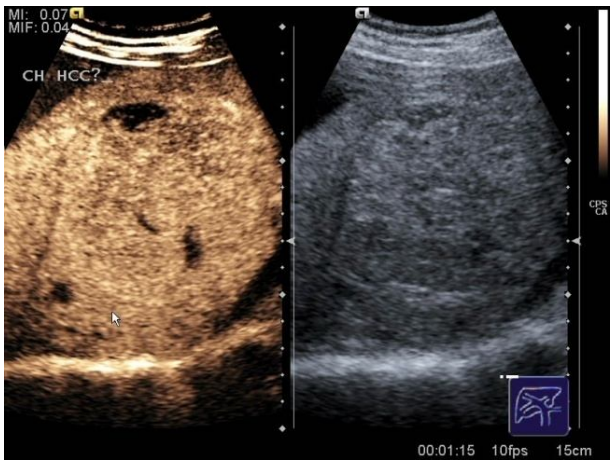


Fig. 24c

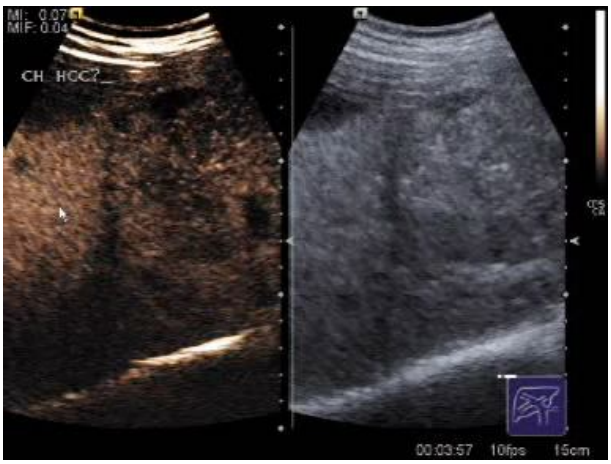


Fig. 24d

Fig. 24: a. Standard US – slightly hyperechoic, inhomogeneous, well delimited mass in the RLL.
b. CEUS arterial phase – rapid arterial inhomogeneous enhancement, with unenhancing ares.
c. CEUS portal phase – isoenhancing mass, with some unenhancing areas.
d. CEUS late phase - the mas is hypoenhancing as compared to the surrounding liver parenchyma – wash-out.
Conclusion: hepatocellular carcinoma.

A relatively frequent diagnostic element found in HCC is portal thrombosis. It appears as a solid like structure in the lumen of the portal vein (Fig. 25). Portal thrombosis can be *global*, affecting the common portal vein, the right and left branches of the portal vein, or it can be *segmental*. The assessment of the portal branches in order to visualize portal thrombosis is essential in any suspicion of HCC, in order to establish the diagnosis as well as the therapeutic strategy (malignant portal thrombosis classically contraindicates surgery).

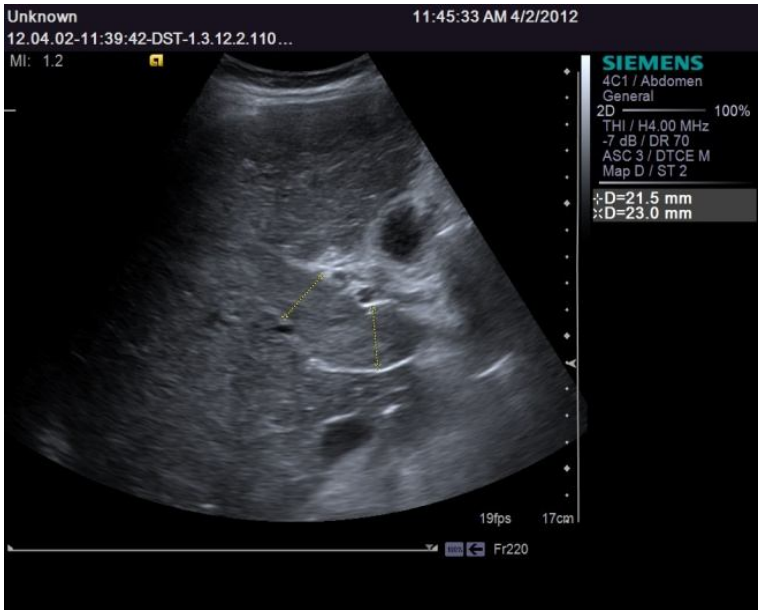


Fig. 25. Portal thrombosis (Common PV) – standard ultrasound

The differentiation between malignant portal thrombosis, in which the thrombus is actually a neoformation vascularized tissue, and benign thrombus, which is actually a blood clot, is done by contrast imaging methods. Since it is avascular, the benign thrombus will not enhance following contrast, while the malignant thrombus, being vascularized, will enhance in the arterial phase and will present wash-out in late phases (Fig. 26 a, b, c; Fig. 27 a, b, c).

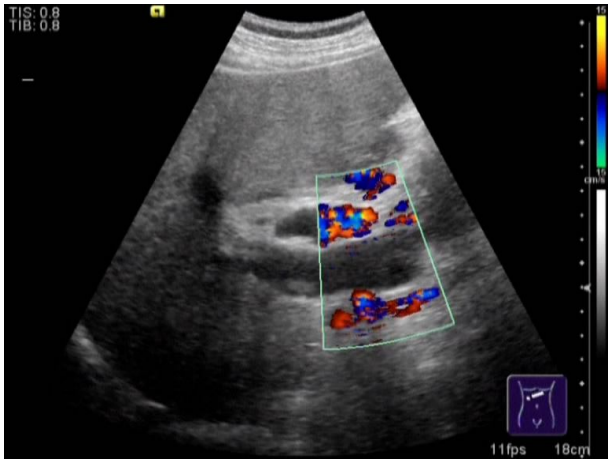


Fig. 26a

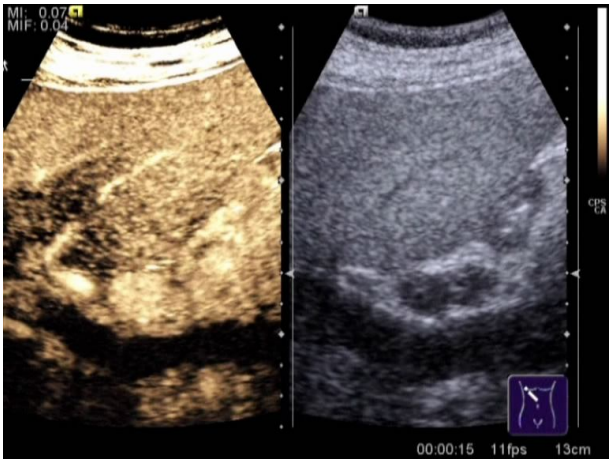


Fig. 26b

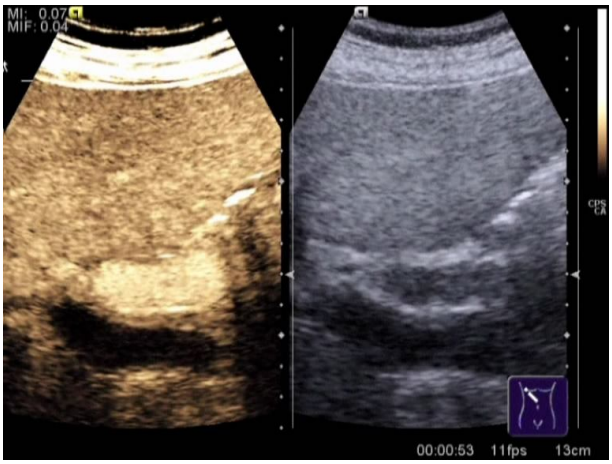


Fig. 26c

Fig. 26: a. Doppler signal absent in the portal vein
b. CEUS arterial phase – absence of contrast in the portal vein
c. CEUS portal phase – absence of contrast in the portal vein.
Conclusion: Benign portal thrombosis.

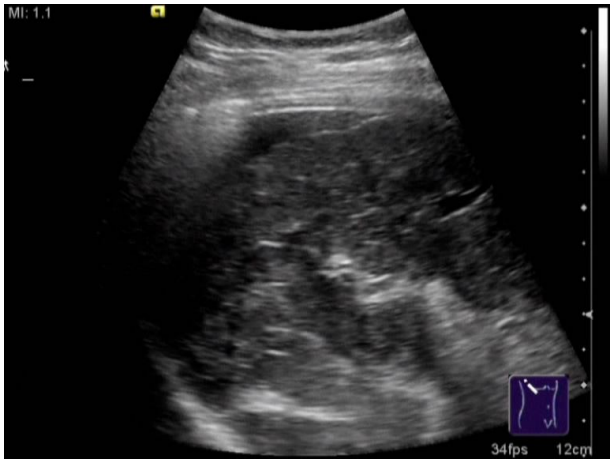


Fig. 27a

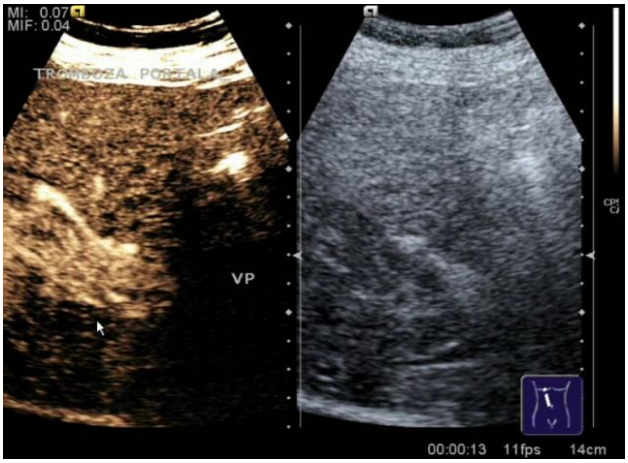


Fig. 27b

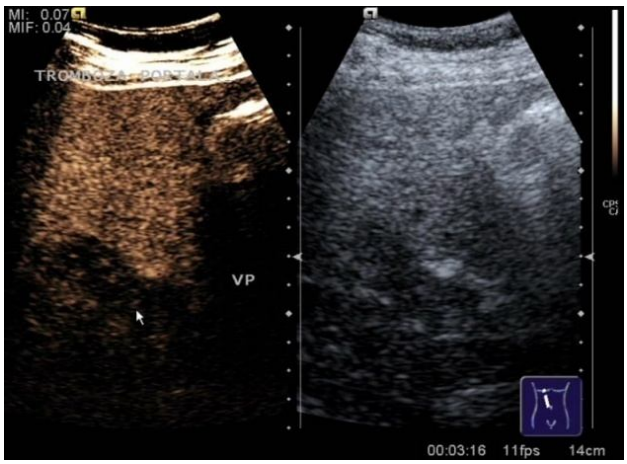


Fig. 27c

Fig. 27: a. Standard US – echodense material in the comon portal vein and in the right portal vein
b. CEUS arterial phase – enhancement in the portal vein
c. CEUS portal phase – wash-out in the portal vein.
Conclusion: Malignant portal thrombosis.

Given the high risk of developing HCC on a cirrhotic liver, screening of these patients for early detection of HCC should be performed. Screening is done primarily by ultrasound and by alpha fetoprotein assessment every 6 months. After the discovery of a HCC in a patient with cirrhosis, it can be treated by various methods, according to the tumor size and to the liver functional reserve. Generally, in tumors less than 5 cm surgical resection or ultrasound-guided techniques (percutaneous ethanol injection therapy - PEIT, or radiofrequency ablation - RFA) are preferred. In tumors larger than 5 cm, the therapy is generally palliative, curative results are very rare. Trans-arterial chemo-embolization (TACE) can be used, or more recently antiangiogenic therapy using Sorafenib (Nexavar).

b. Cholangiocarcinoma

Cholangiocarcinoma is a tumor whose starting point in the biliary epithelium. It is relatively rare; the HCC/cholangiocarcinoma ratio is approximately 15:1.

Depending on location, there are three types of cholangiocarcinoma: peripheral (cholangiolar carcinoma); hilar (Klatskin tumor) and extrahepatic (choledocal tumor).

Hilar cholangiocarcinoma (Klatskin tumor) is difficult to detect by ultrasound because it is a small tumor. In standard ultrasound the intrahepatic bile ducts are dilated in both lobes or only in some segments upstream the lesion (Fig.28), with normal main bile duct and gallbladder. Klatskin tumor is easily visualized by MRCP (magnetic resonance cholangiopancreatography) or ERCP (endoscopic retrograde cholangiopancreatography), a more invasive method but which allows therapy (stent insertion to drain the biliary obstruction).

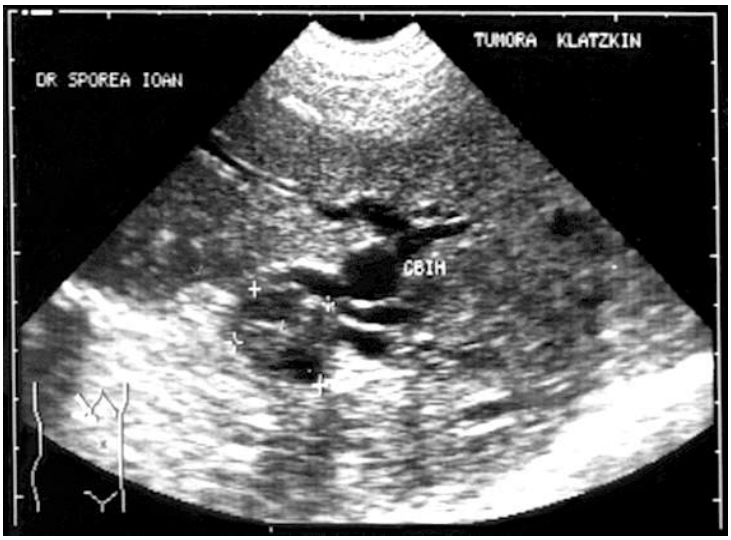


Fig. 28. Klatskin tumor with upstream dilatation of intrahepatic biliary ducts

The ultrasound appearance of cholangiocarcinoma is not typical. It can appear as a peripheral “bulls-eye tumor” or as an inhomogeneous, hypoechoic tumor (Fig. 29). CEUS will visualize a poorly vascularized tumor in the arterial phase, possibly with “rim” enhancement, with wash-out in late phases. Ultrasonography is not the ideal method for the diagnosis of cholangiocarcinoma, but it may suspect the diagnosis which will be confirmed by other techniques (MRCP or ERCP).

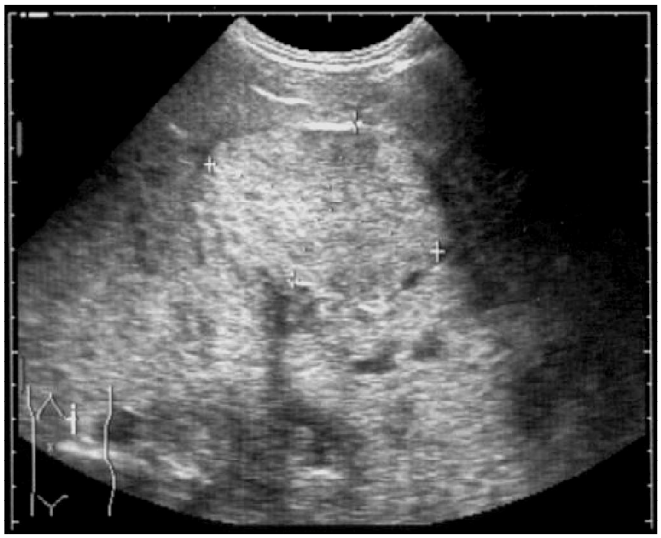


Fig. 29. Peripheral cholangiocarcinoma

3. Liver metastases

Liver metastases are single or multiple hepatic disseminations of a malignant tumor originated in other organs. Liver metastases are most frequently secondary to colorectal cancer, to small cell bronchial carcinoma, to gastric carcinoma, to pancreatic carcinoma, to breast carcinoma, to endocrine tumors of the digestive tract, to malignant melanoma, as well as to renal tumors.

In clinical practice, there are two circumstances in which liver metastases are found: incidental ultrasound detection of liver masses suspected to be metastases, in which case a primary tumor will be searched for; or in a patient with known cancer who is monitored by ultrasound to detect potential secondary hepatic involvement.

The ultrasound appearance of metastases is not typical. Metastases can be hypoechoic, hyperechoic or rosette like (“bull’s eye” appearance). Metastases generated by rapidly growing tumors (pancreatic or pulmonary tumors) (Fig. 30) are most frequently hypoechoic, and so are metastases secondary to breast cancer. Metastases from tumors with a slow evolution (colorectal cancer) are frequently hyperechoic (Fig. 31). The typical image for malignancy in the liver is the "rosette like" or “bull’s eye” appearance (Fig.32), but it cannot differentiate between a primitive and a metastatic tumor.

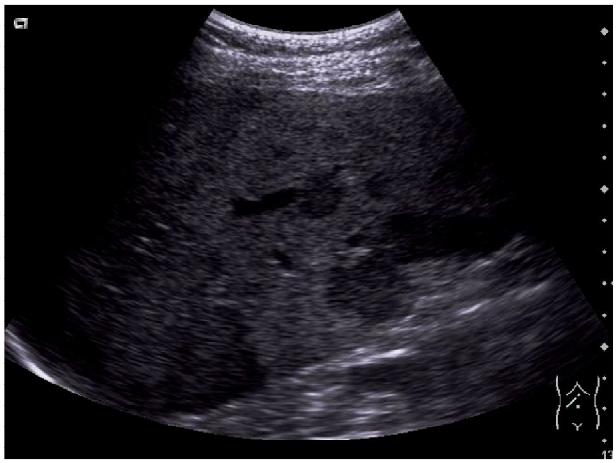


Fig. 30. Hypoechoic metastases

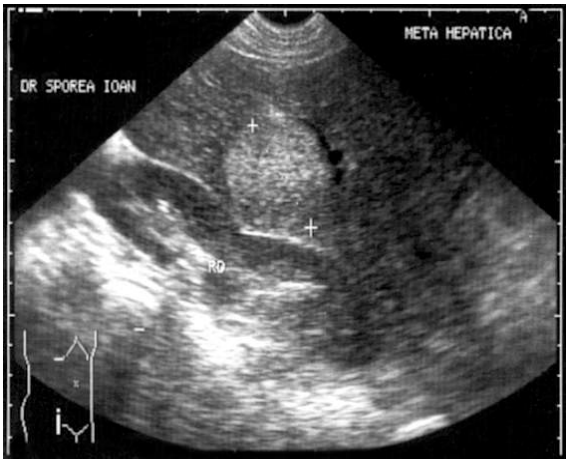


Fig. 31. Hyperechoic metastasis

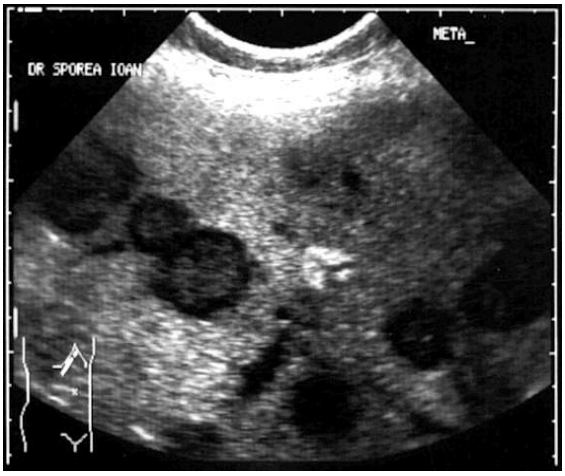


Fig. 32. Rosette or "bull's eye" metastases

Imaging contrast methods (CEUS or CT) can confirm the ultrasound diagnosis of metastasis. At CEUS, metastases can be hypervascular (rapid enhancement following contrast in the arterial phase and rapid wash-out, sometimes at the end of arterial phase) (Fig. 33 a, b, c) or hypovascular (poor enhancement following contrast during the arterial phase, frequently with rim-like aspect, followed by rapid wash-out) (Fig. 34 a, b, c).

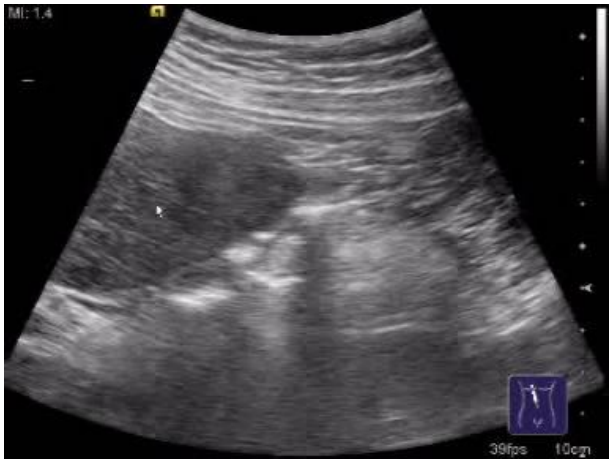


Fig. 33a

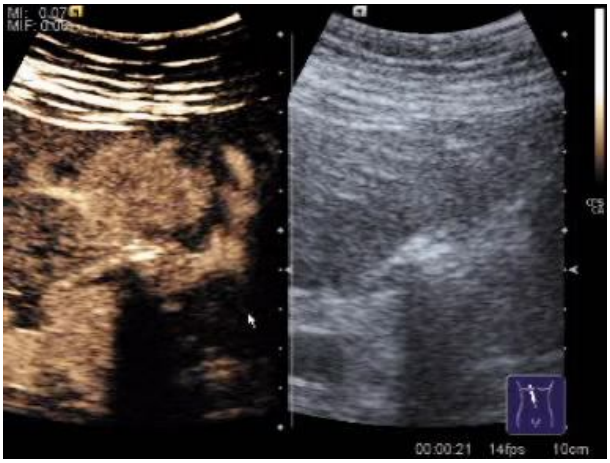


Fig. 33b

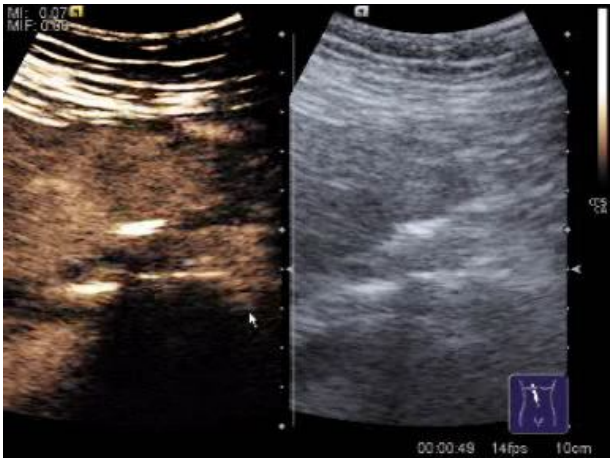


Fig. 33c

Fig. 33: a. Standard ultrasound – slightly hypoechoic mass, difficult to visualize in LLL.
b. CEUS arterial phase – rapid arterial enhancement.
c. CEUS portal phase – the mass becomes hypo-enhancing already at the beginning of the portal phase (second 49): wash-out.
Conclusion: hypervascular metastasis.

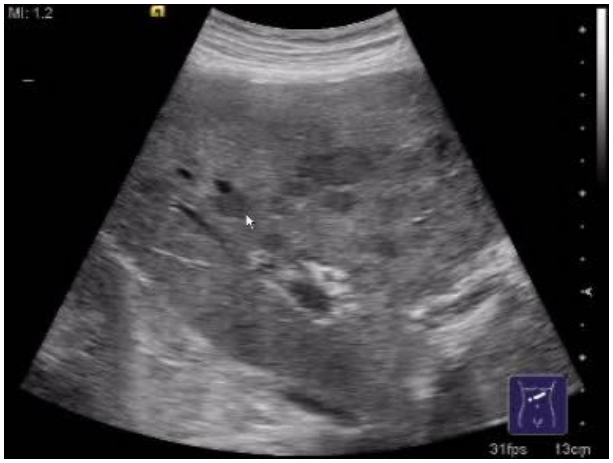


Fig. 34a

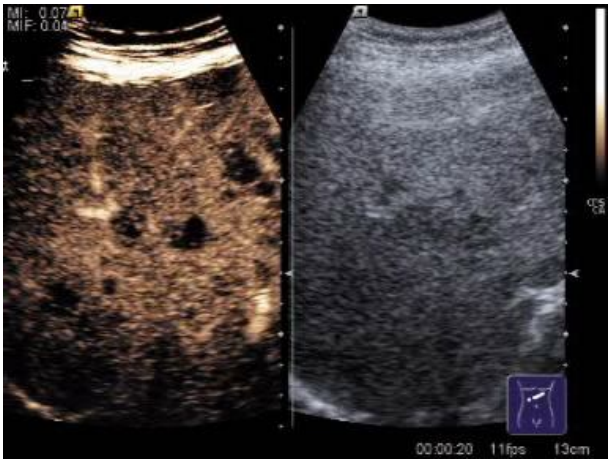


Fig. 34b

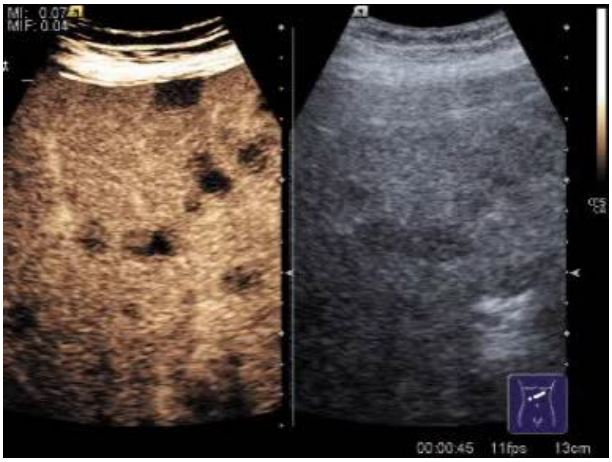


Fig. 34c

Fig. 34: a. Standard ultrasound – multiple hypoechoic and rosette masses in the RLL.
b. CEUS arterial phase – multiple hypo-enhancing masses in RLL.
c. CEUS portal phase – multiple hypo-enhancing masses in RLL.
Conclusion: hypovascular metastases.

Standard ultrasound and CEUS are used to assess metastases under chemotherapy. They may regress in size under chemotherapy or may suffer processes of tumor necrosis (central area becomes hypoechoic or transonic) and calcification.

Chapter 6.

Ultrasound of the gallbladder and biliary tree

1. The gallbladder

The gallbladder is the source of numerous abdominal complaints and is easily examined by ultrasound in most cases. Examination is done through right subcostal recurrent oblique sections, through sagital sections below the right costal grid or through intercostal sections; in dorsal decubitus and, mandatory, in left lateral decubitus position. The examination must be made carefully, with full view of the gallbladder, with special attention on the infundiblar area, where gallstones can be hidden. By turning the patient in left lateral decubitus, the infundiblar area will become more accessible, and possible gallstones can be mobilized, falling by gravity to the bottom of the gallbladder, where they are better visualized.

a) Normal gallbladder

The normal appearance of the gallbladder is of a pear-shaped anechoic structure, with a well-defined, hyperechoic wall (Fig. 1). The normal dimensions are generally below the 8/3 cm, the maximum accepted is 10/4 cm, over this dimension raising the suspicion of hydrops. The normal wall thickness is maximum 4 mm. Postprandial the gallbladder wall appears duplicated, due to the smooth muscle contraction of the gallbladder wall (Fig. 2).

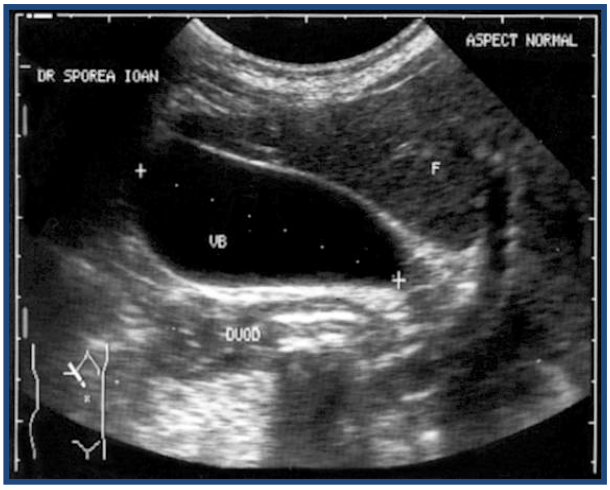


Fig. 1. Normal gallbladder

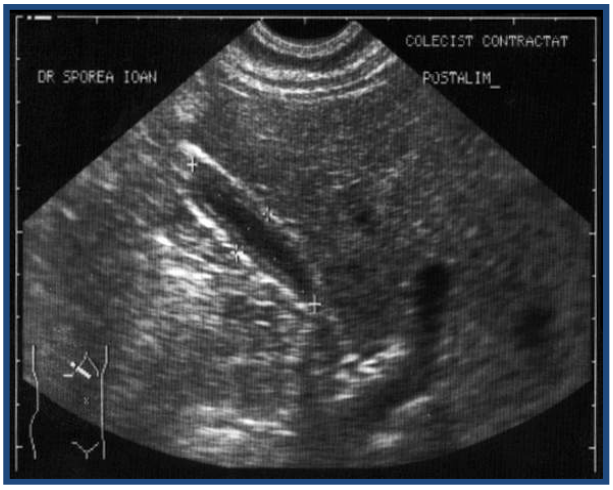


Fig. 2. Postprandial contracted gallbladder

It is important that the ultrasound examination of the gallbradder to be done strictly in fasting conditions (at least 8 hours), avoiding coffee (cholecysto-kinetic effect). Postaprandial the gallbladder contracts and the wall will appear duplicated, raising problems of differential diagnosis with acute cholecystitis, etc. Also, the gallbladder filled with bile provides a good ultrasound view that allows the visualization of gallstones and their mobility.

b) „Malformations" of the gallbladder

The gallbladder "malformations" are particular shapes of the gallbladder that are commonly found, varying from a bisaccular, to a globulous, or to a drop shaped gallbladder. Gallbladder septa can sometimes be present, and should not be confused with normal infundiblar septa (Heister's valves).

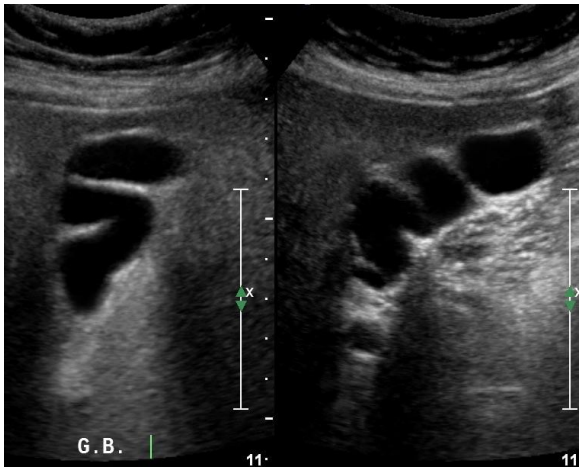


Fig. 3. Gallbladder septa (http://www.ultrasoundpaedia.com/normal-gallbladder/)

The majority of less experienced ultrasonographists pay great attention to the description of these changes in gallbladder shape and to the description of more or less "real" septa, aimed at explaining biliary dyspeptic disorders, terminology that has disappeared from gastroenterology literature for over 20 years. The notion of dysmotility-like functional dyspepsia (nausea, vomiting, bloating) or the notion of irritable bowel (intestinal cramps, bloating, motility disorders) are currently used. Currently, cephalea or headache is no longer considered to be related to gallbladder pathology; vomiting in headache is due to brain edema, not to gallbladder disease. However, uninformed patients, with the support of the medical world, continue to hold the gallbladder responsible for dyspeptic symptoms associated to headache.

c) Gallbladder polyps

Gallbladder polyps are prominences of the gallbladder mucosa; they are mostly cholesterol polyps, but they can also be adenomatous. Gallbladder polyps have a variable frequency in general population, up to 1.5-5% of women and 4 to 6% of men, and they are incidentally found by ultrasound in asymptomatic patients.

Adenomatous polyps have a typical appearance in ultrasound, namely round structures, adherent to the gallbladder wall, which does not change its position when the patient changes position. Their echogenity is similar to the one of the gallbladder wall, and the size ranges between 3 and 10 mm (rarely bigger, when a malignant transformation should be suspected) (Fig. 4 and Fig. 5). Polyps can be single or multiple, and the absence of the posterior shadow and of gravitational fall differentiates them from gallstones. The use of high-frequency transducers (5 or 7.5 MHz) may reveal more diagnostic elements.

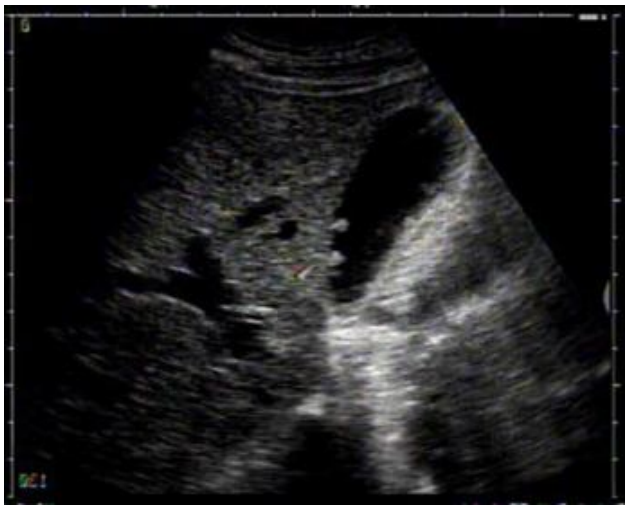


Fig. 4. Two adenomatous gallbladder polyps

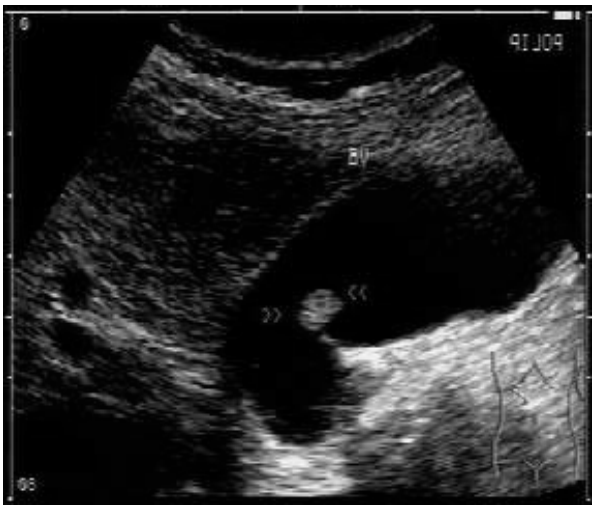


Fig. 5. Single adenomatous gallbladder polyp

Cholesterol polyps appear as hyperechoic protrusions adherent to the gallbladder wall, without gravitational fall, with a "comet tail" appearance, with the transverse diameter generally less than 5 mm. They have no pathological significance (Figure 6 and Figure 7).

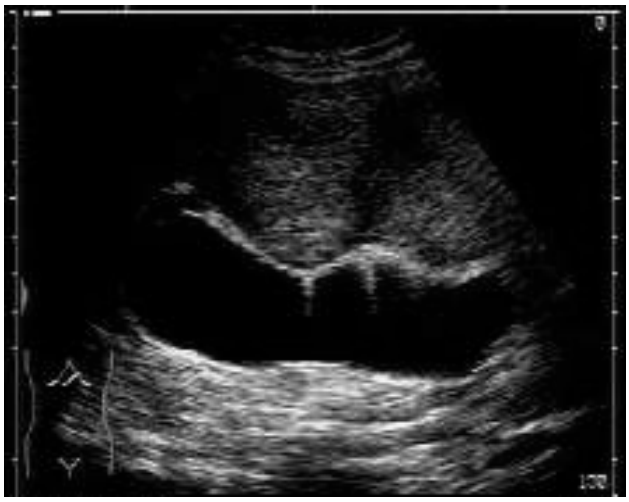


Fig. 6. Cholesterol polyps

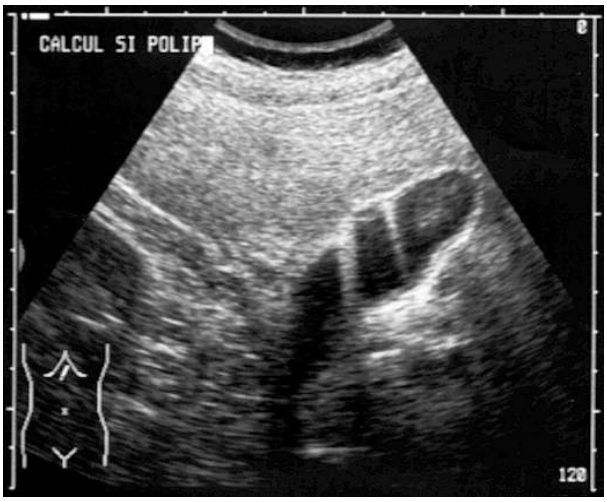


Fig. 7. Cholesterol polyps

Once diagnosed, the gallbladder polyps should only be monitored. Small polyps, up to 10 mm in size, do not pose diagnostic problems, being completely typical. Polyps larger than 10-15 mm should be differentiated from gallbladder carcinoma. However, when the diagnosis is not clear only by imaging methods (CT, MRI), diagnostic cholecistectomy is preferred instead of delaying a diagnosis of malignancy (gallbladder carcinoma has a potential for a very rapid malignant development).

d) Biliary sludge

Biliary sludge is a mixture of mucus, calcium bilirubinate and cholesterol crystals, a consequence of the imbalance of the bile components and of gallbladder evacuation disorders. According to some authors, it is a precursor state for gallstones, while others consider it a reversible condition.

The ultrasound appearance of biliary sludge is typical, in the form of a mobile echoic material in the gallbladder, sometimes with a horizontal level (Fig.8). This echoic material does not display a ”posterior shadow”, and its shape and location in the gallbladder change with the change in the patient’s position. Sometimes, biliary sludge can fill the entire gallbladder (Fig.9), conferring the appearance known as ”hepatization” of the gallbladder (in gallbladder hydrops, during pregnancy, or after prolonged parenteral nutrition). Another particular variant of biliary sludge is the ball-like or pseudotumoral aspect (Fig.10), characterized by a globulous appearance, which can be maintained after gravitational fall, or it can “disintegrate”.

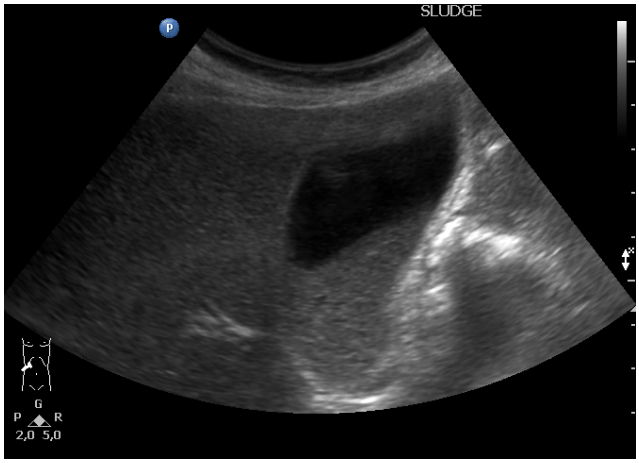


Fig. 8. Biliary sludge with horizontal level

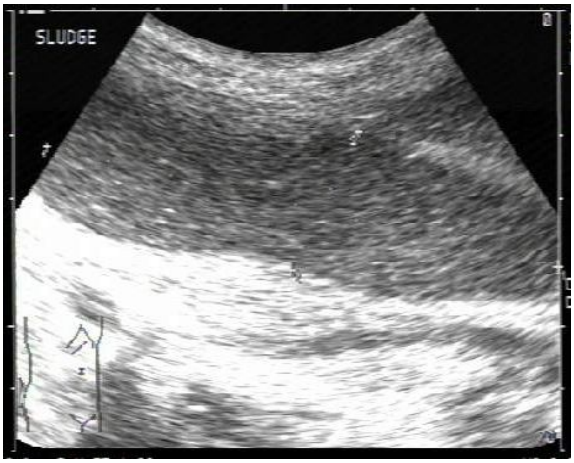


Fig. 9. "Hepatization" of the gallbladder

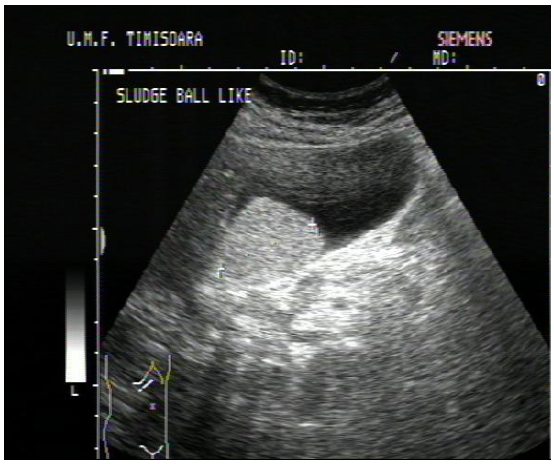


Fig. 10. "Ball-like" biliary sludge

The differential diagnosis of biliary sludge should be made with a gallbladder tumor (easy through contrast ultrasound – CEUS: the sludge will not enhance following contrast, since it is avascular, while the tumor will enhance); with gallbladder polyps (which have no gravitational fall); or with gallbladder stones (they have posterior shadow).

e) Cholelithiasis

Cholelithiasis is defined as the presence of cholesterol or calcium bilirubinate stones in the gallbladder. It is a very common condition, it affects 5 - 20% of the population, and the prevalence is influenced by genetic factors, the presence of obesity, dyslipidemia and diabetes. Not all patients with gallstones should undergo cholecistectomy, only those who have *symptomatic cholelithiasis*, namely those who had a biliary colic. Biliary colic is an intense pain located in the epigastrium and/or right hypochondrium, which lasts for more than 30 minutes. Nausea, vomiting, bloating and headache in the context of a patient with gallstones are not part of the diagnosis of symptomatic cholelithiasis, so they are not an indication for surgery.

Asymptomatic cholelithiasis should only be followed-up. Large studies that followed patients with cholelithiasis for 20 years have shown that only about 20% of the cases of asymptomatic cholelithiasis became symptomatic and only 10% had complications. In case of biliary colic, we reevaluate the diagnostic and recommend cholecistectomy.

The ultrasound appearance of cholelithiasis is generally typical, transabdominal ultrasound is very efficient for its diagnostic, with a sensitivity of 95-96%. The typical aspect is of a hyperechoic image, with posterior shadow, with "gravitational fall" when changing the patient's position (Fig.11). It should be added that the echodense image must be inside the gallbladder (thus, it will be differentiated from digestive air, situated outside the gallbladder – Fig. 12). The only situation when the gallstone does not have gravitational fall is when it is blocked in the infundible, in gallbladder hydrops (Fig.13). Regarding the posterior shadow, it can be weak or almost absent for small stones (2-3 mm), due to their size (Fig. 14).

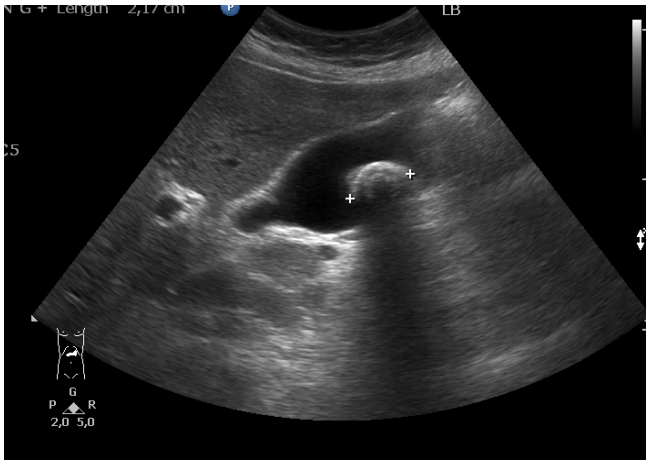


Fig. 11. Single gallstone

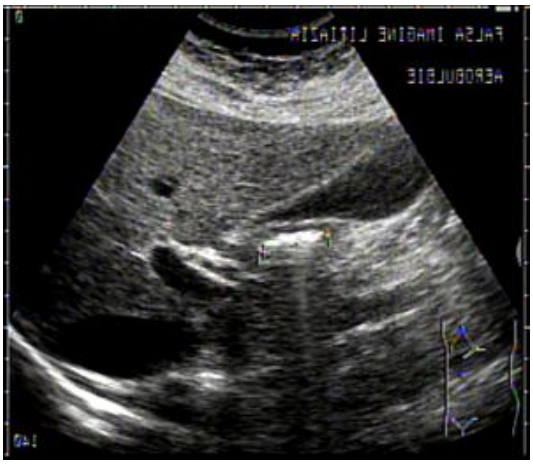


Fig. 12. Air in the duodenal bulb

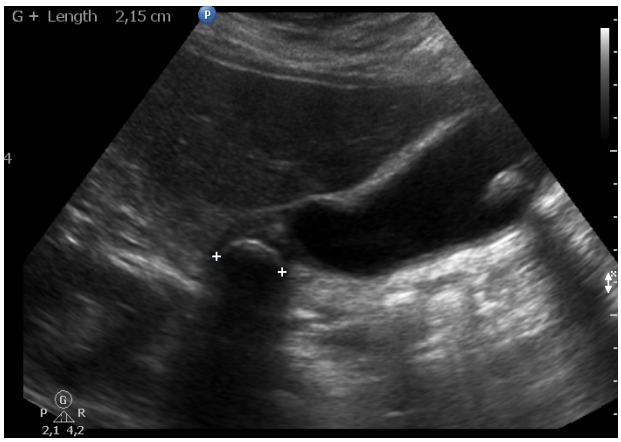


Fig. 13. Gallbladder hydrops, gallstone stuck in the infundibulum, ant 2 others on the bottom of the gallbladder

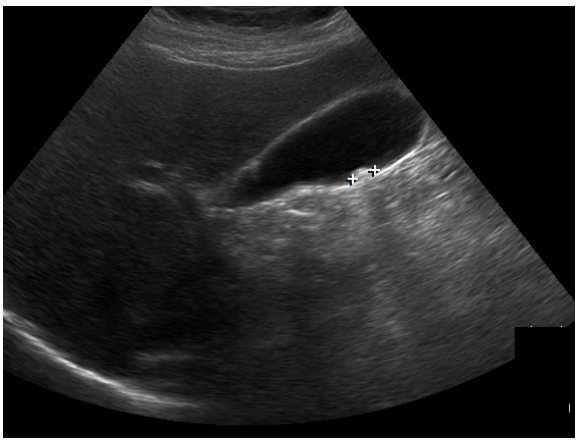


Fig. 14. Small gallstone, with weak posterior shadow

The ultrasound diagnosis of cholelithiasis is relatively easy and it can establish whether it is single or multiple lithiasis (Fig.16), their approximate size (without necessarily counting or measuring them exactly). It is important to demonstrate the mobility of a calculus and to do so, the patient's position should be changed, from dorsal decubitus to left lateral decubitus. Thus the infundiblar area will be better visualized and the gallstones hidden in this area will be seen when they mobilize to the bottom of the gallbladder.

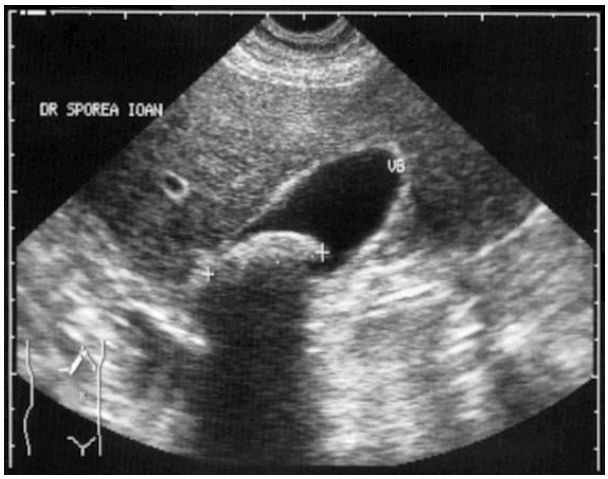


Fig. 15. Single gallstone

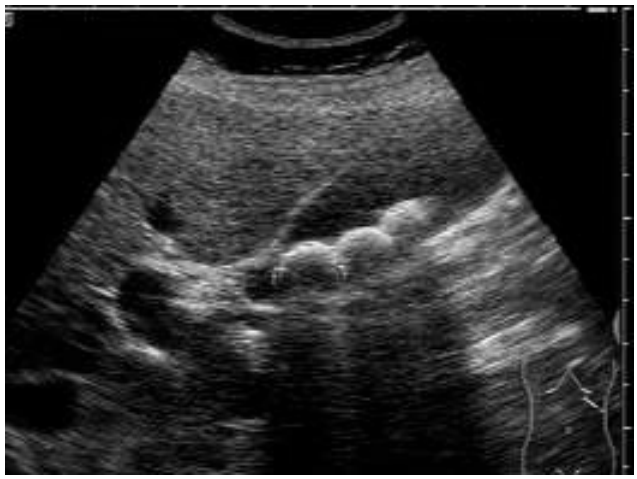


Fig. 16. Three gallstones

Although the ultrasonic image of a gallbladder calculus is typical, *ultrasound differential diagnostic* problems can sometimes occur, namely: in case of a gallbladder full of calculi, when the absence of the bile makes the vesicular bed difficult to visualize, but in this case the intense posterior shadow must draw our attention (Fig .17); a large vesicular calculus, occupying all the gallbladder, where again, the bile is absent, and it will generate the "shell sign" (reflective crescent with large posterior shadow) (Fig. 18); small gallstones (1 - 2 mm), which may or may not generate posterior shadow, difficult to distinguish from biliary sludge - to which there is an etiopathogenic similitude (Figure 19); gallstones that do not generate posterior shadow (generally those of bilirubinate) that may be mistaken with a gallbladder polyp, but the latter will not fall with gravity.

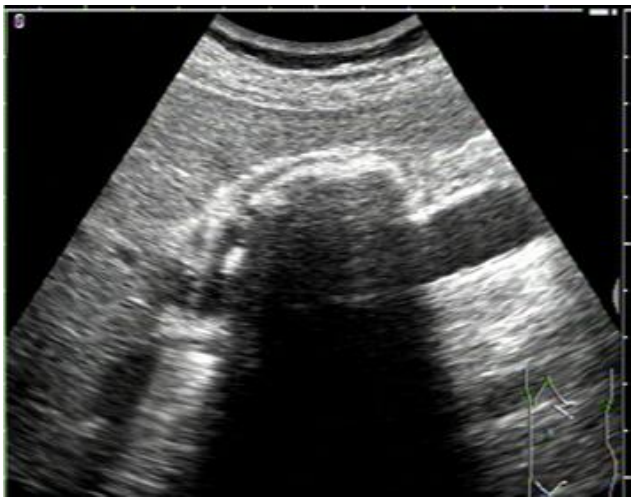


Fig. 17. Gallbladder molded on gallstones

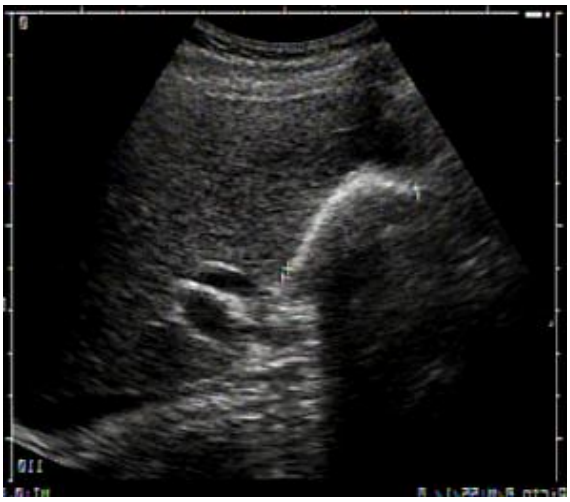


Fig. 18. Single large calculus - the shell sign

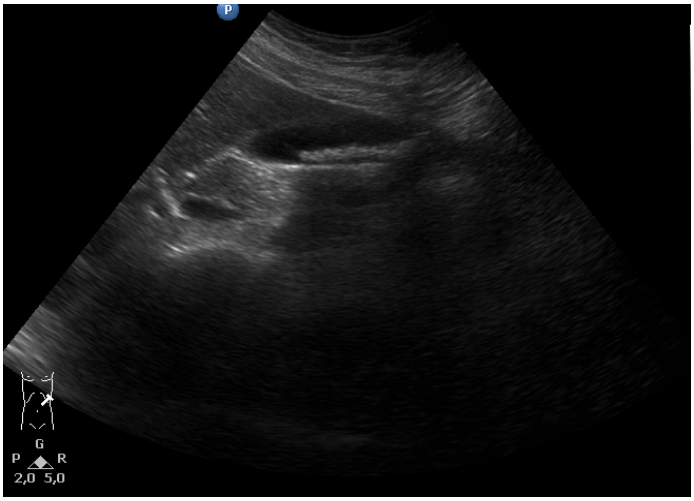


Fig. 19. Small gallstones, with weak posterior shadow – differential diagnostic with biliary sludge

Ultrasound is a sensitive and specific diagnostic method in cholelithiasis (sensitivity of up to 96%), however, the diagnosis can be missed, especially by beginners. One cause is a poor ultrasound window, but its quality can be improved by examining through the intercostal spaces, by mobilizing the patient, by inviting the patient to make a prolonged inspiration. Patient mobilization is required and it will facilitate mobilization of a potential gallstone initially missed, which will become visible. The infundiblar blocked gallstone is also difficult to assess (Fig. 20), in which case what draws our attention is just a large gallbladder with the "feeling" that is under tension (globular).

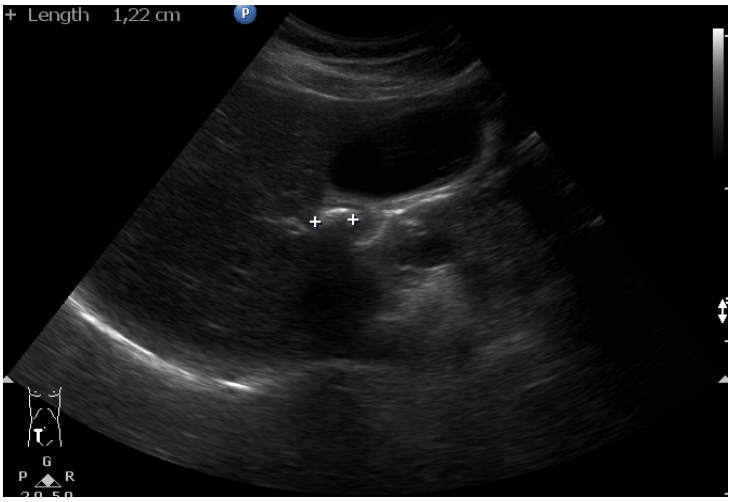


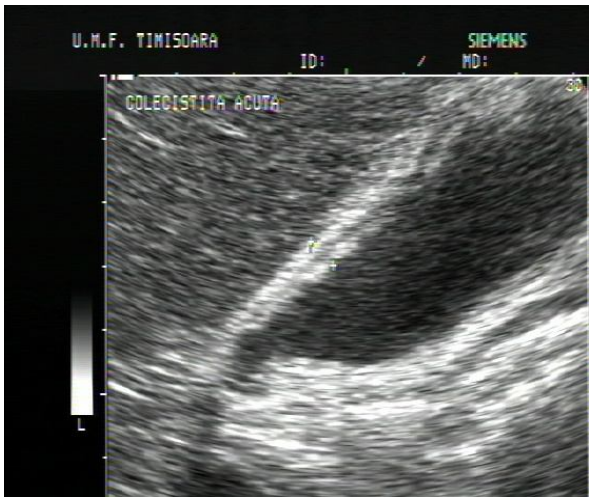
Fig. 20. Blocked infundiblar gallstone

f) Acute cholecystitis

Acute cholecystitis is an acute inflammation of the gallbladder wall. The most frequent cause is biliary lithiasis. Rarely acute cholecystitis is non-lithiasic, generated by germs such as Salmonella, Escherichia coli, Streptococcus fecalis, etc). Another rare cause of cholecystitis is ischemia that may occur in shock, after surgery, after chemoembolization or after local ultrasound guided therapy of liver tumors (PEIT, PAAI or RFA).

To support the diagnostic of acute cholecystitis clinical elements are required: intense pain in the right hypochondrium and/or the epigastrium, frequently with right subscapular radiation, with or without fever and chills. Objective examination will evidence pain on palpation in the right hypochondrium (Murphy's sign), which can lead to muscular defense.

The ultrasound appearance in acute cholecystitis is of thickened and usually, duplicated gallbladder wall (Fig. 21, Fig. 22). From the normal size of 4 mm, in acute cholecystitis due to edema the gallbladder wall may reach 6-8 mm (even 10 mm). The duplication of the gallbladder wall with a "sandwich" appearance is quite common.



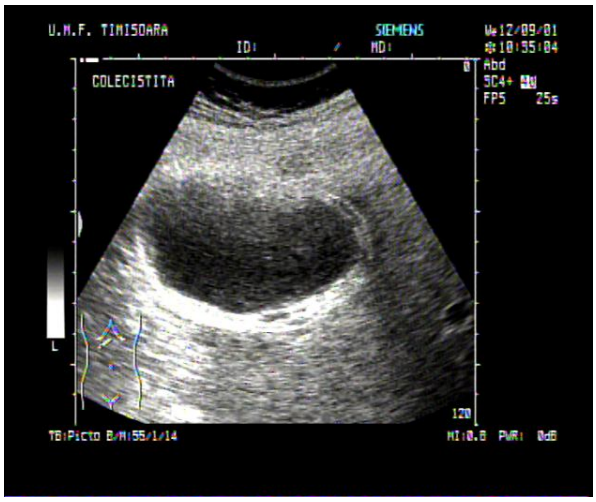


Fig. 21, 22. Acute cholecystitis – thickened and duplicated gallbladder wall

In addition to these parietal changes, frequently inflammatory pericholecystic exudate can be found, which appears as an anechoic or hypoechoic band. The integrity of the gallbladder wall can be investigated, parietal discontinuities suggesting gallbladder perforation. Another suggestive sign of gallbladder perforation into an aerated digestive organ is the presence of air in the gallbladder (a hyperechoic image in the upper portion of the organ, mobile with the patient's movements). In most cases acute lithiasic cholecystitis is involved, the presence of biliary lithiasis or of a calculus impacted in the infundible will be revealed (Fig. 23). Frequently, ultrasound will visualize along with gallstones the presence of biliary sludge, difficult to distinguish from gallbladder empyema (Fig. 24). In ultrasound, pain at "palpation" with the transducer is the ultrasound Murphy's sign. Murphy's sing associated with biliary lithiasis has a 92% positive predictive value for the diagnosis of acute cholecystitis.

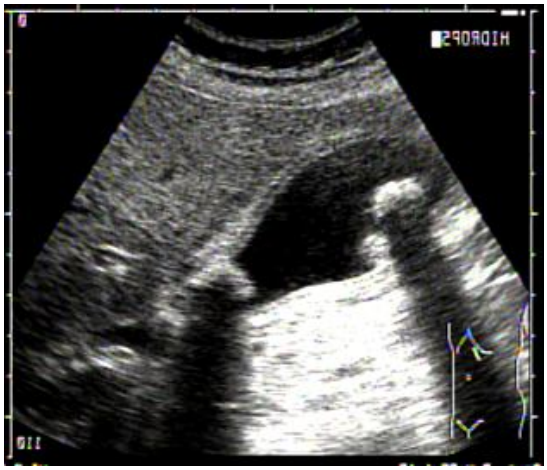


Fig. 23. Acute cholecystitis, gallbladder hydrops, Calculus blocked in the infundibulum, and 2 others on the bootom of the gallbladder, doubled gallbladder wall

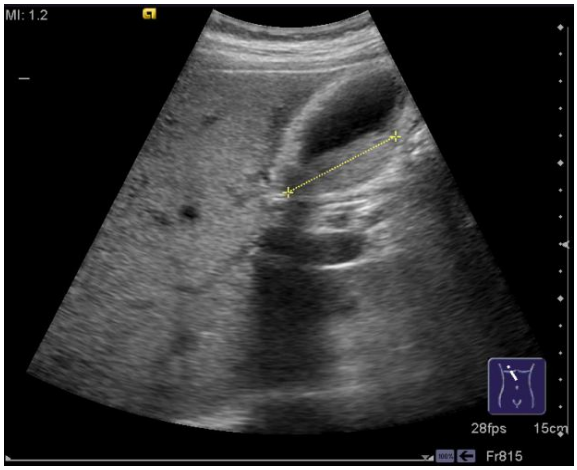


Fig. 24. Acute cholecystitis, thickened and doubled wall, echoic material–biliary sludge?

The ultrasound differential diagnosis of acute cholecystitis should be made with chronic cholecystitis (in which the gallbladder wall is thicker, hyperechoic, but not duplicated). Another difficult differential diagnosis involves a thickened and duplicated gallbladder wall in cirrhosis, acute viral hepatitis, nephrotic syndrome, chronic renal failure (gallbladder duplication is due to hypoalbuminemia). Attention should be paid to the postprandial duplicated gallbladder wall (due to the thickening of the muscular layer by contraction), but the anamnesis does not reveal pain in this case, instead food intake is related.

The most difficult ultrasound differential diagnosis is between acute lithiasic cholecystitis and a thickened and duplicated gallbladder wall in a patient with biliary lithiasis and liver cirrhosis. The defining elements are the clinical symptoms and the presence or absence of ultrasound Murphy's sign.

g) ***Chronic cholecystitis***

Chronic cholecystitis is a chronic inflammatory process of the gallbladder wall that occurs in the presence of cholelithiasis. The diagnosis of chronic cholecystitis can be suspected by ultrasound and is confirmed by pathological examination after cholecistectomy.

The ultrasound appearance is of thickening of the gallbladder wall over 4 mm, most commonly with a hyperechoic appearance (Fig. 25), generally without duplicated aspect, and with negative ultrasound Murphy's sign.

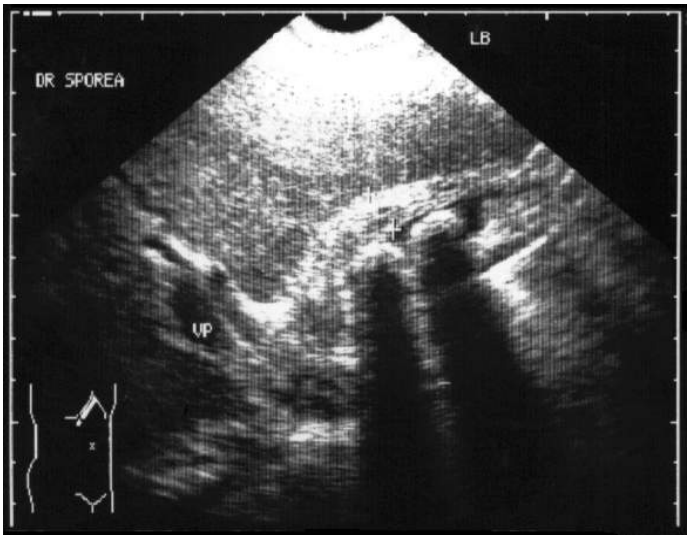


Fig. 25. Chronic lithiasic cholecystitis: thick, hyperechoic gallbladder wall

The ultrasound differential diagnosis of chronic cholecystitis will be made with: acute cholecystitis, gallbladder cholesterolosis, gallbladder adenomyomatosis, early gallbladder carcinoma, porcelain gallbladder. Porcelain gallbladder is a particular situation characterized by partial or complete calcification and thickening of the gallbladder wall (Fig. 26, Fig. 27). It is considered as a precancerous state, which is why it is an indication for cholecistectomy, even if asymptomatic. The ultrasound aspect is of a hyperechoic crescent, with intense posterior shadow in the projection area of the gallbladder. It can be a difficult differential diagnosis with a gallbladder filled with stones or with a large stone that completely fills the gallbladder.

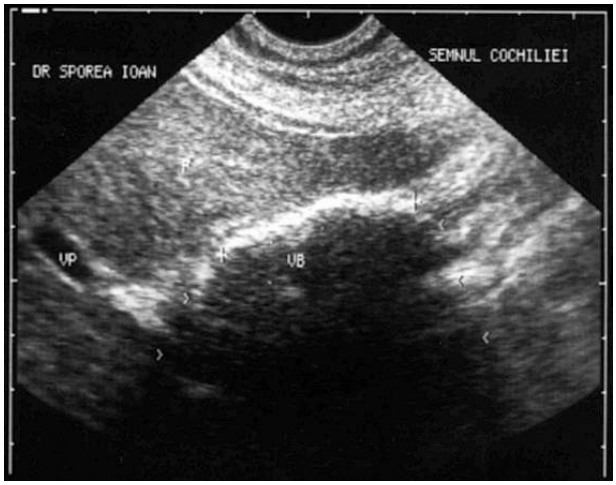


Fig. 26. Porcelain gallbladder

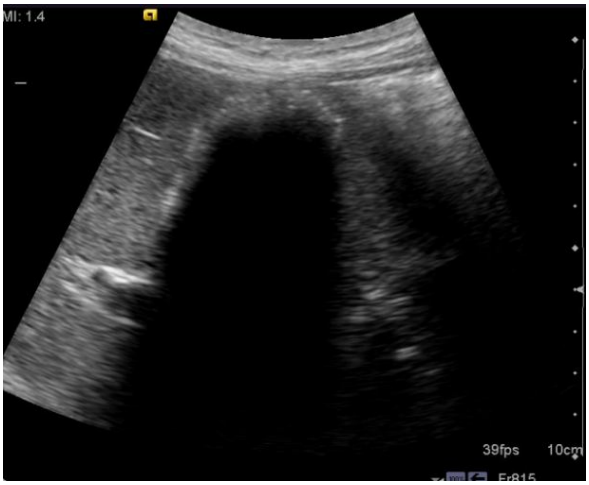


Fig. 27. Porcelain gallbladder

h) Gallbladder carcinoma

Gallbladder cancer is a relatively rare entity, considering the high number of cholelithiasis cases in the general population and the fact that it almost always occur on a background of biliary lithiasis. It is a disease of the elderly, occurring usually after 70 years. Unfortunately it is a cancer that is diagnosed too late, given the fact that it's clinical signs are frequently absent or non-characteristic (colicky pain in the right hypochondrium or persistent painful discomfort). Sometimes it is accidentally detected by ultrasound.

The ultrasound appearance of gallbladder carcinoma is not typical. In the early phases, the polypoid gallbladder cancer appears as an endoluminal excrescence, similar to a gallbladder polyp. The echogenity of the mass is similar to parenchyma with an irregular outline, not unlike a large gallbladder polyp (Fig. 28) (always suspect malignancy when the polyp size is larger than 15-20 mm). With high frequency transducers we can detect disruption in the continuity of the gallbladder wall layers and invasion in the adjacent liver parenchyma.

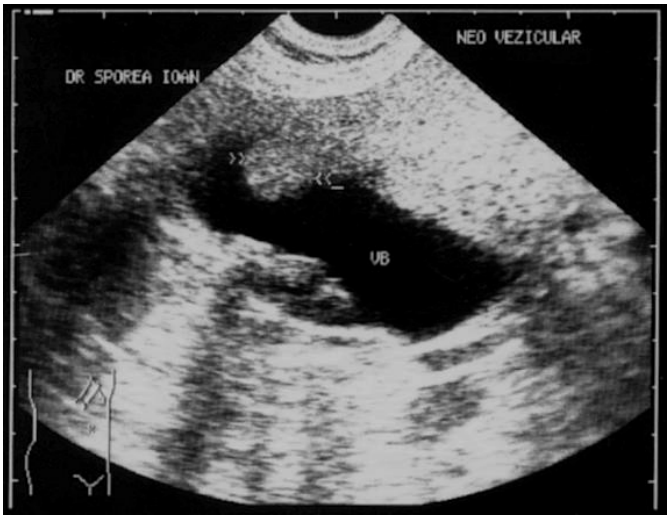


Fig. 28. Polypoid gallbladder cancer (>>). Invasion in the adjacent liver parenchyma (hypoechoic area) and biliary stones

In scirrhous carcinoma forms that infiltrate the gallbladder, the imaging diagnosis is particularly difficult, given the frequent association with cholelithiasis. The ultrasound differential diagnosis with chronic or acute cholecystitis may raise serious problems. In case of gallbladder carcinoma the gallbladder wall is thick, anfractuous, hypoechoic, thickening is usually much more obvious and irregular as compared to cholecystitis.

In advanced forms, with invasion in the adjacent liver parenchyma, a hypoechoic mass will be seen by ultrasound in the projection area of the gallbladder, centered by a calculus image (hyperechoic image with posterior shadow) (Fig. 29). Frequently in this situation liver metastases and/or carcinomatous ascites (Fig. 30) are found.

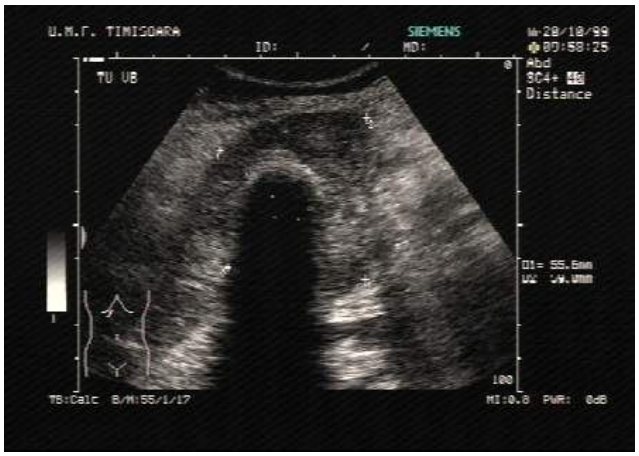


Fig. 29. Advances gallbladder carcinoma, with invasion of the adjacent liver. Ultrasound aspect of a hypoechoic area centered by a calculus.

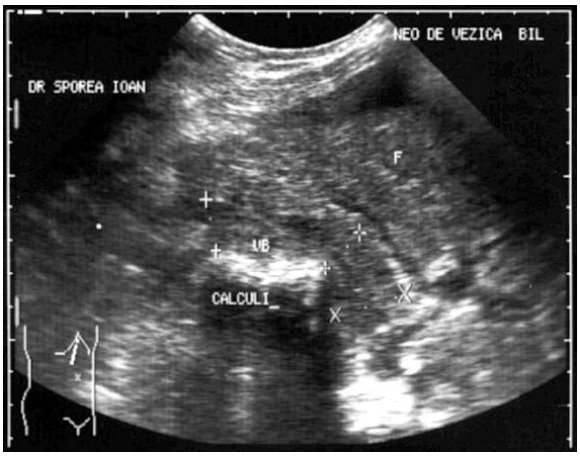


Fig. 30. Advanced gallbladder carcinoma. Hypoechoic mass centered by a calculus. Carcinomatous ascites (top right)

2. Ultrasound of the biliary tree

a) Normal aspects

The choledochus or the main biliary duct (MBD) is examined through a perpendicular section on the costal grid and is located anteriorly from the portal vein (PV) (Fig. 30). The maximum normal diameter is 5-6 mm, a normal value of 7-8 mm being accepted in patients with cholecistectomy. MBD, PV and the hepatic artery (HA) are constituent elements of the hepatic hilum. The HA intersects at a point the MBD and PV, passing between them. MBD and PV have the same direction, thus making the appearance of a "double-barreled shotgun," but the barrels are uneven, the thinner barrel, located anteriorly, is the MBD. When the ratio between the diameter of the MBD and the PV reverses, it is a diagnostic sign for obstructive jaundice.

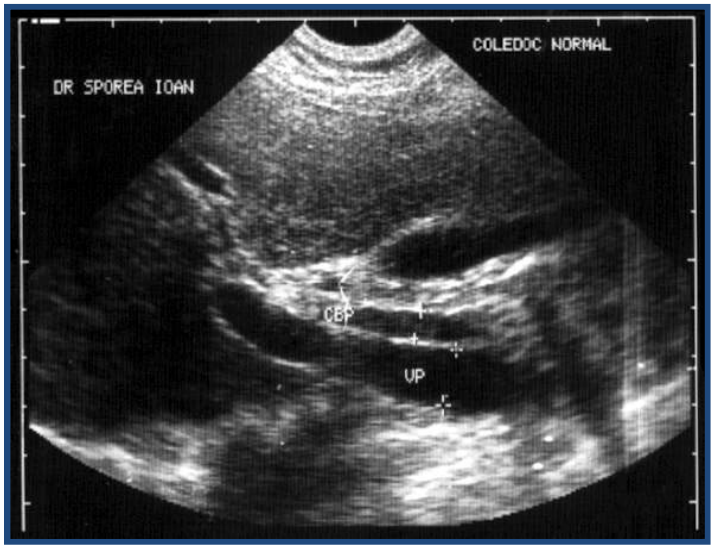


Fig. 30. The choledochus (MBD) and the common portal vein (PV) in the hepatic hilum

The intrahepatic bile ducts are not normally seen in standard ultrasound, since they have a very fine caliber. They will become visible when there is a downstream obstacle, appearing as anechoic structures parallel with the branches of the portal vein, thus making the appearance of a "double duct". If dilatations are important, a "spider-like" appearance will be seen in the incidence that visualizes the portal bifurcation (Fig.31).

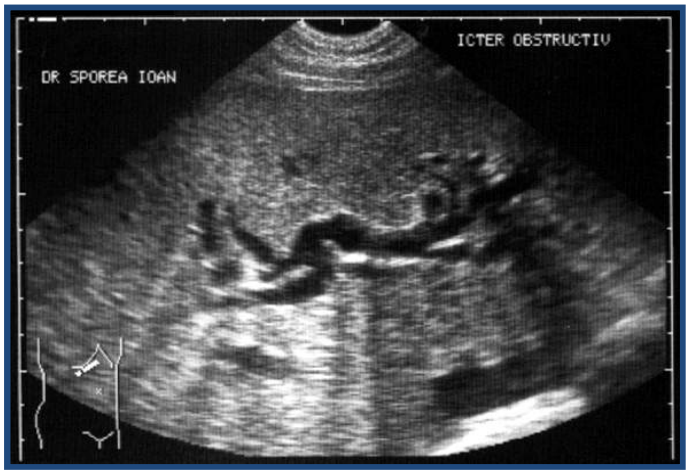


Fig. 31. Dilated intrahepatic bile ducts – "spider-like" appearance

After surgery with a biliodigestive anastomosis or after endoscopic sphincterotomy, gas will permeate from the digestive tract in the MBD and sometimes in the intrahepatic bile ducts, making it difficult to assess their size. The air in the biliary ducts appears as a hyperechoic eyebrow with a “dirty” posterior shadow, situated in the highest area (Fig. 32, Fig. 33).

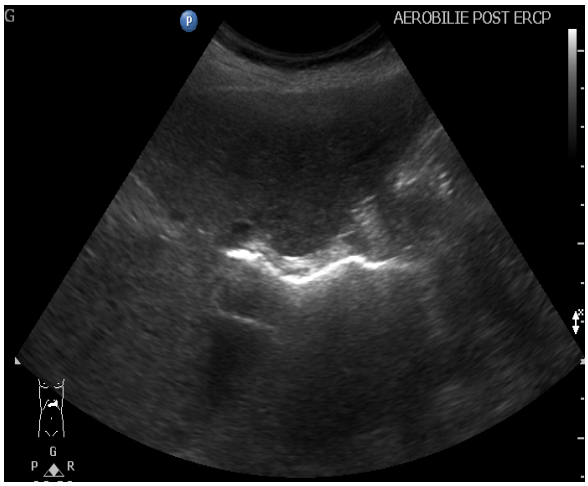


Fig.32. Air in the choledochus

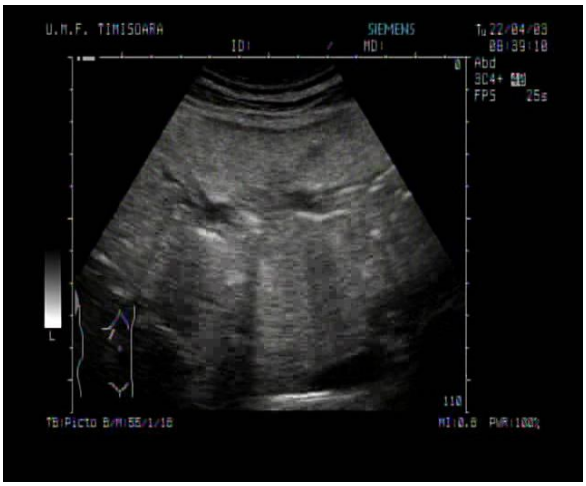


Fig.33. Air in the intrahepatic bile ducts post ERCP

Abdominal ultrasound is one of the key elements in the differential diagnosis of jaundice syndrome. The first question which we have to answer is if the jaundice is parenchymatous or obstructive. The answer is simple. If in ultrasound, dilations of the MBD and/or of the intrahepatic bile ducts are not seen, it is a parenchymatous jaundice. In the same time we can assess etiologic elements of a parenchymatous jaundice such as: irregular liver surface, heterogeneous liver structure, caudate lobe hypertrophy, splenomegaly, ascites – suggestive for liver cirrhosis; multiple hepatic masses – suggestive for hepatic metastasis, in this case the jaundice is secondary to displacement of normal hepatic parenchyma.

Abdominal ultrasound is a sensitive diagnostic method in obstructive jaundice, with 90-95% sensitivity, through highlighting dilation of intrahepatic bile ducts and/or of the choledochus. The next step is establishing the level of obstruction, in which abdominal ultrasound has an accuracy of about 90%, and establishing the cause of obstructive jaundice, in which the accuracy of ultrasound is only approximately 70%.

a) Obstructive jaundice with high level obstruction

In the high level obstruction only dilations of the intrahepatic biliary ducts will be visible, segmental or in the whole liver, depending on the etiology; a choledochus with normal size and a small gallbladder. The most common cause of obstructive jaundice with high level obstacle is a tumor, either by central cholangiocarcinoma (Klatzkin tumor) (Fig.34), either by other primary liver tumors (hepatocellular carcinoma, peripheral cholangiocarcinoma) or by liver metastasis which invades the hilum or the biliary tree branches, causing their upstream dilation (Fig.35), or through compression by near adenopathy on the hilar MBD.

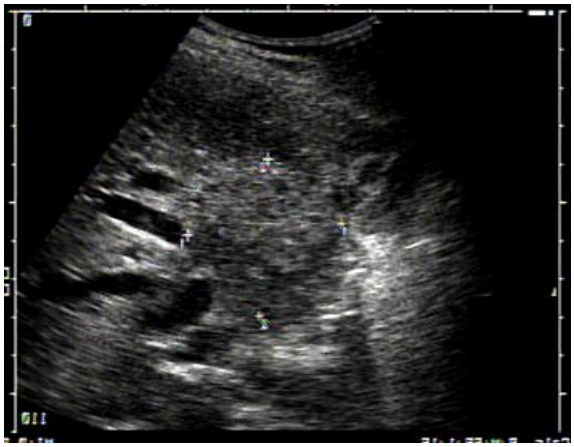


Fig. 34. Obstructive jaundice – Klatzkin tumor

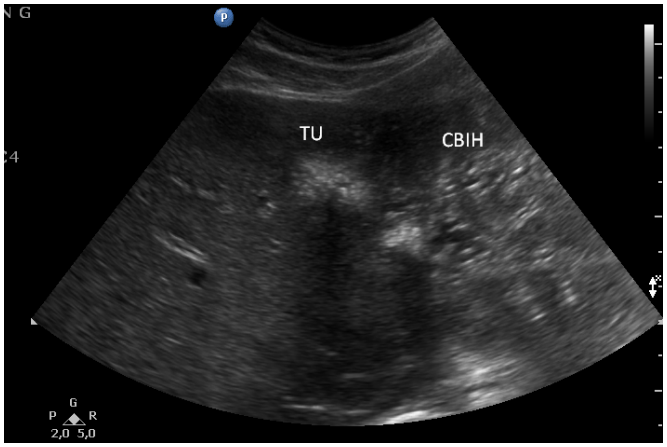


Fig.35. Segmental dilations of intrahepatic bile ducts in the left liver lobe through tumoral invasion

In Europe, a rare cause of obstructive jaundice with high level obstacle is intrahepatic lithiasis. In ultrasound dilation of intrahepatic bile ducts will be seen upstream of linear hyperechoic images, with intense posterior shadow (Fig. 36).

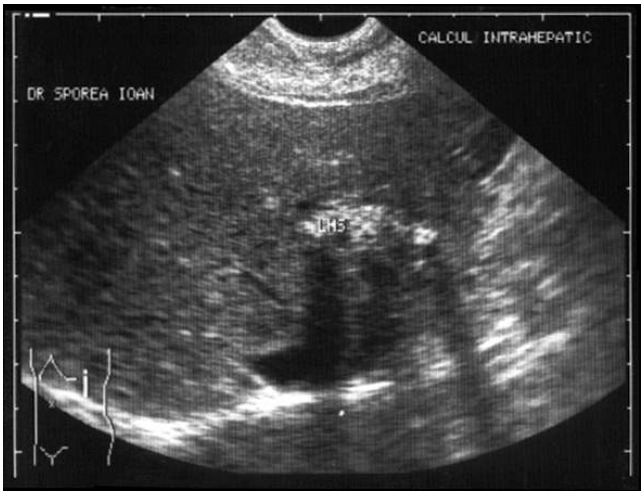


Fig. 36. Intrahepatic lithiasis

b) Obstructive jaundice with low level obstruction

In obstructive jaundice with low obstacle, besides dilation of the intrahepatic bile ducts we will notice also the dilation of the choledochus (Fig. 37) and gallbladder (patients without cholecistectomy) – the Courvoisier-Terrier ultrasound sign (Fig. 38)

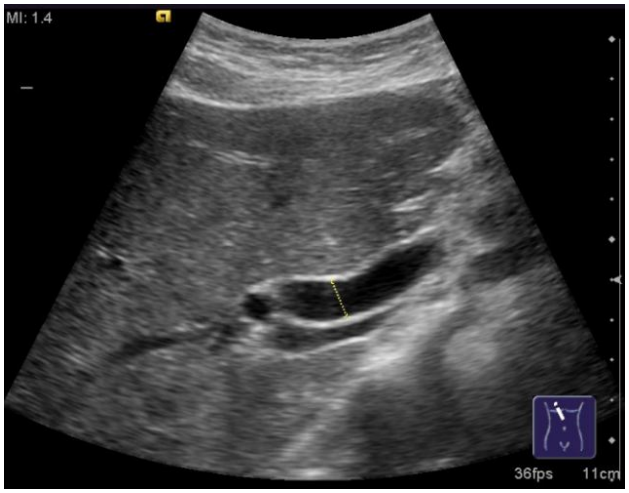


Fig. 37. Obstructive jaundice – 10 mm MBD

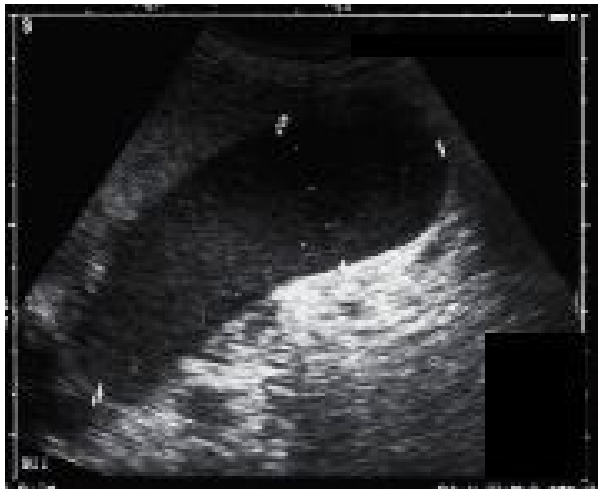


Fig. 38. Courvoisier-Terrier ultrasound sign

The most frequent cause of obstructive jaundice with low level obstruction are MBD stones, in which the ultrasound appearance is of a dilated MBD with a hyperechoic image with posterior shadow in the lumen (Fig. 39, Fig. 40). It is a difficult diagnosis, since the distal end of the MBD is deeply located. We should always suspect common bile stones in a patient with sudden obstructive jaundice installed after colic, especially if he or she was known with gallstones. The examination should be done with patience and perseverance to confirm or rule out this diagnostic.

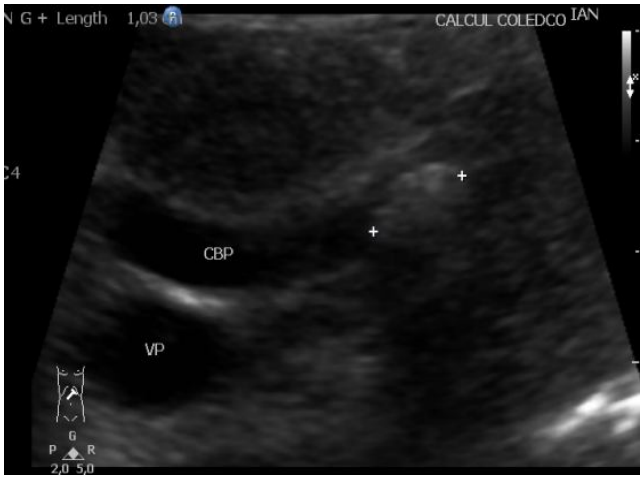


Fig. 39. Obstructive jaundice – MBD stone

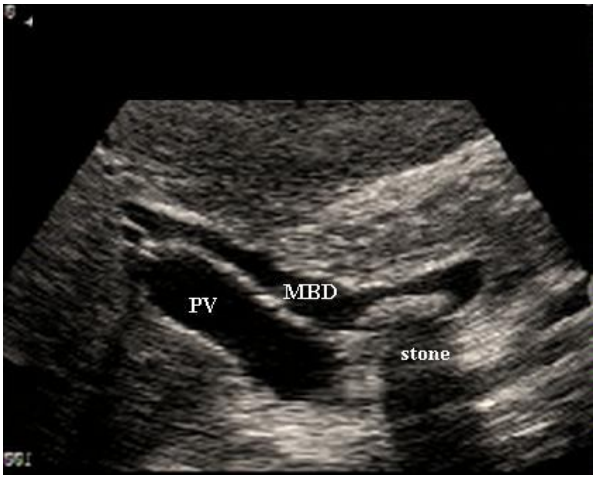


Fig. 40. Obstructive jaundice – MBD stone

Sometimes multiple stones can be seen in the MBD lumen, in some cases even a „gritted MBD" – multiple stones in the MBD, symptomatic or not. In this case the lumen of the choledochus will be occupied by hyperechoic images with posterior shadow (Fig. 41).

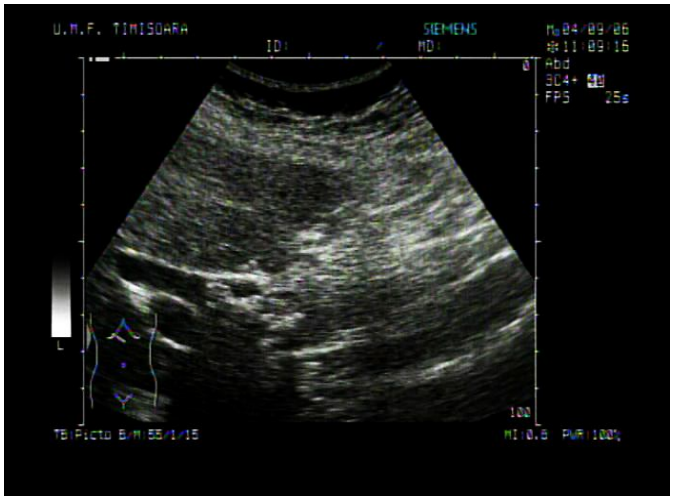


Fig. 41. "Gritted MBD" (three stones in the distal MBD)

Pancreatic head carcinoma is another common cause of obstructive jaundice. This time it is a progressively installed jaundice, with no colic, or accompanied by progressive mild pain and weight loss. In addition to ultrasound signs of obstructive jaundice with low level obstacle, a hypoechoic structure can be seen in the pancreatic head (Fig. 42, Fig. 43).

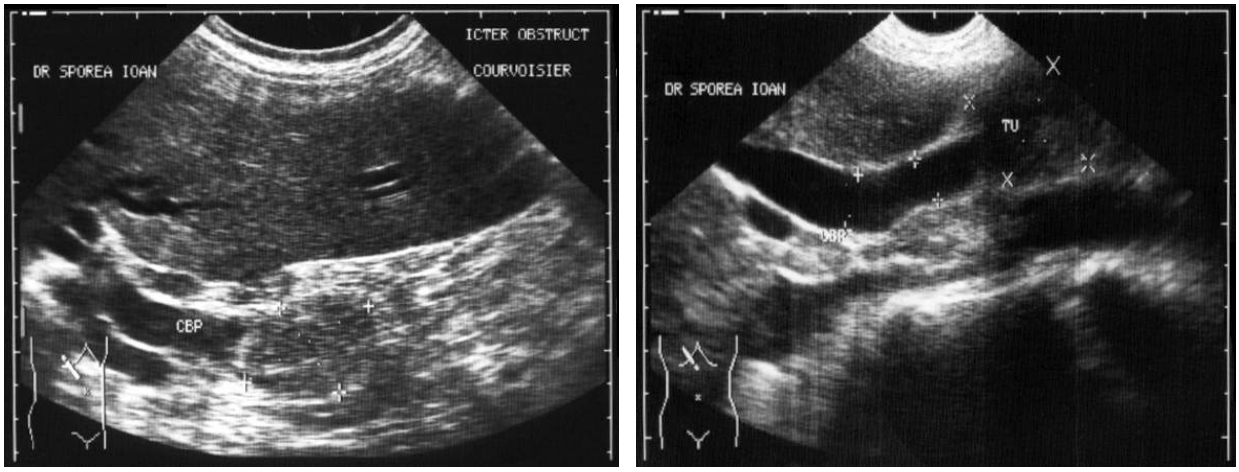


Fig. 42 and Fig. 43: Dilated MBD ending in a hypoechoic mass in the pancreatic head

Chronic pancreatitis can generate obstructive jaundice due to pancreatic head hypertrophy or to pancreatic head pseudocyst. Ultrasound will show signs of chronic pancreatitis (pancreatic calcifications, dilation of the Wirsung duct, Wirsung stones) and an enlarged pancreatic head (Fig. 44). The MBD is compressed by a large pancreatic head, frequently inhomogeneous, with calcifications, or by a pancreatic head pseudocyst (an anechoic image with echoic walls).

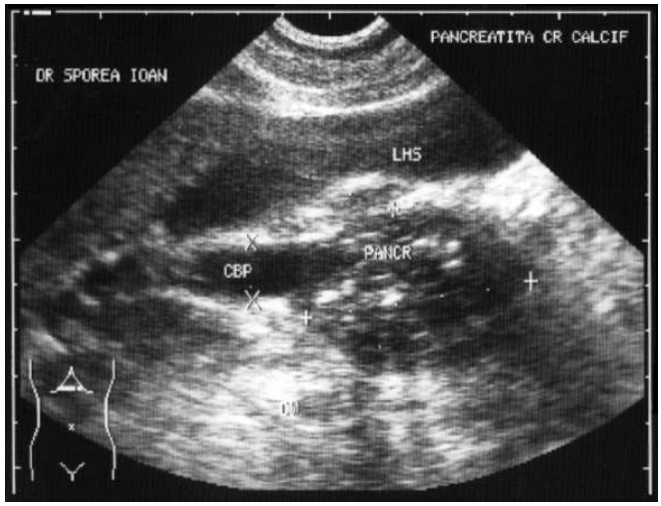


Fig. 44. Dilated MBD ending in a hypertrophic pancreatic head with calcifications

Vater's ampulloma is a rare cause of obstructive jaundice with progressive evolution, without pain, sometimes accompanied by moderate anemia. Since it is a small tumor that grows toward the duodenum, it is not usually visible by ultrasound, only signs of obstructive jaundice with low level obstacle are visualized.

Malignant MBD tumor (cholangiocarcinoma) or benign MBD tumor (papilloma) can generate the ultrasound aspect of obstructive jaundice, the tumor will appear as an echoic mass in the lumen of the MBD, without a posterior shadow (Fig. 45), which enhances following contrast bolus in CEUS, a differential diagnosis element from lithiasis and microlithiasis of the choledochus, which do not enhance following contrast.

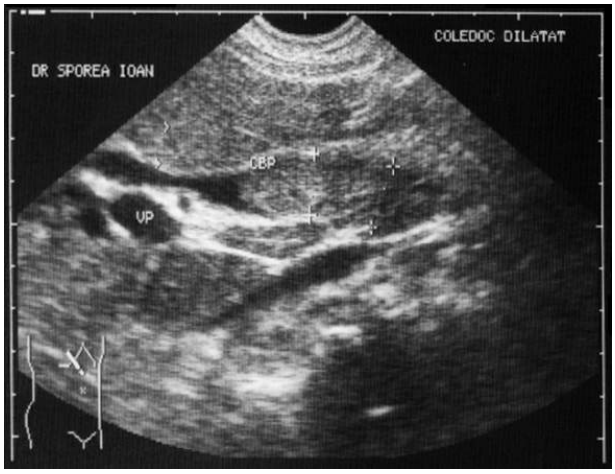


Fig. 45. Echoic material without posterior shadow in the lumen of a dilated MBD

Rare cases of obstructive jaundice are due to extrinsic compressions of the MBD by hilar adenopathies or by migrating parasites (ascarids).

Chapter 7.

Pancreatic Ultrasound

1. Normal pancreas

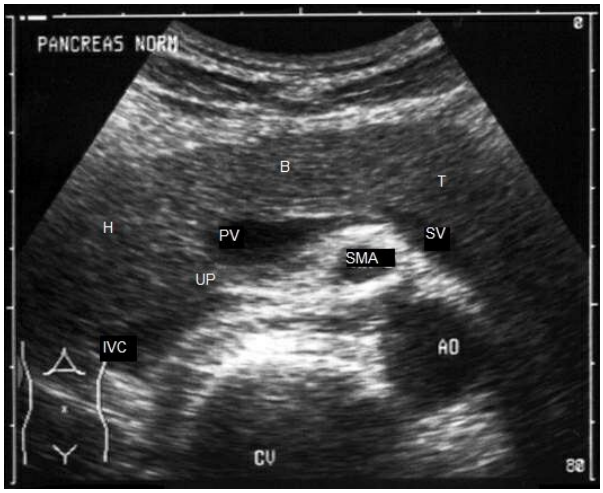
Pancreatic ultrasound is the touchstone of ultrasound examination. This is why the examination of this organ is a permanent stress for the beginner in ultrasound. Hundreds or thousands of explorations are still required before the examination of the normal or pathological pancreas is no longer a difficult task. Examination difficulties derive from the deep anatomical location of this retroperitoneal organ, which can be masked by the intestines, the intestinal gas acting as a screen that prevents the penetration of ultrasound waves.

Is very important to know the anatomical landmarks that delimit the pancreas: posteriorly the spleno-portal axis and anteriorly the gastric antrum and the left liver lobe.

Examination starts from high in the epigastrium, mild progressive compression ease the examination by mobilizing the bowel content. We use transverse epigastric sections in particular, a 3.5 MHz (or multi-frequency) convex transducer is preferred; in thin (or cachectic) persons a 5 MHz linear transducer is needed. Ideal to examine the patients in fasting conditions (7-8 hours), to avoid interposition of food and gas from the stomach between the pancreas and the transducer.

The pancreas will be examined over the gastric antrum (if the transducer is placed high in the epigastrium), trans-gastric, or more rarely, below the antrum (the position of the transducer is midway between the xiphoid appendix and the umbilicus). The best ultrasound window for pancreatic examination will be obtained by high sections (avoiding the colon), through the left hepatic lobe (which behaves like an ultrasound window for the pancreas), or trans-gastric. For best trans-gastric pancreatic examination, the stomach (antrum) should contain no air (aerogastria) or liquid should be present in the stomach. The presence of liquid in the stomach plays the role of an ultrasound window for the examination of the pancreas. Hence the practical situation used in case of difficult visualization of the pancreas, when the patient will be administered 500-700 ml plain water that will form an ultrasound window in the stomach. After ingestion, 10-15 minutes are required for the debubbling of the ingested liquid.

The examination of the pancreas through a transverse epigastric section will firstly detect the spleno-portal axis (the portal vein and the splenic vein), which delimits the pancreas posteriorly and which appears as a transonic (anechoic) image situated anteriorly of the spine, aorta and inferior vena cava (Fig 1.).The pancreas is delimited anteriorly by the gastric antrum or the left hepatic lobe (depending on the level at which the transverse section is performed) (Fig. 2).



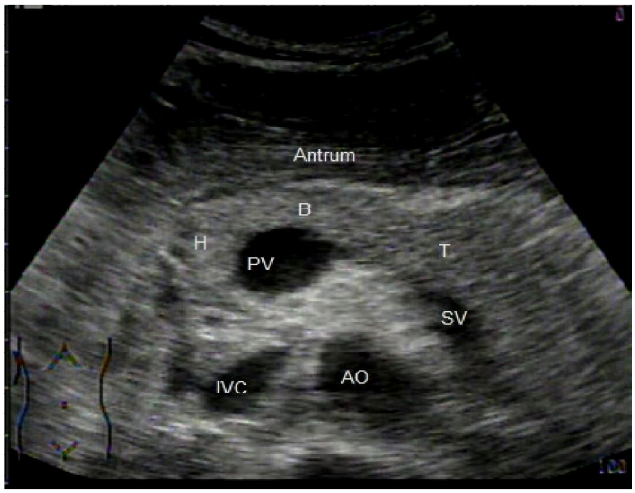


Fig. 1, Fig. 2: Normal pancreas- anatomical landmarks: PV –portal vein; SV –splenic vein; SMA –superior mesenteric artery; AO-aorta; IVC –inferior vena cava; CV –spine; H –head of pancreas; B –body of pancreas; T –pancreatic tail

Another important vascular reference is the celiac trunk, specifically the pancreatic artery and the hepatic artery: at their emergence from the celiac trunk these "lie" on the top of the pancreas. For this reason, when you see the emergence of the celiac trunk from the aorta (the appearance of "flying albatross") (Fig. 3), the transducer must be angled slightly down and the pancreas will appear in the examination plan.

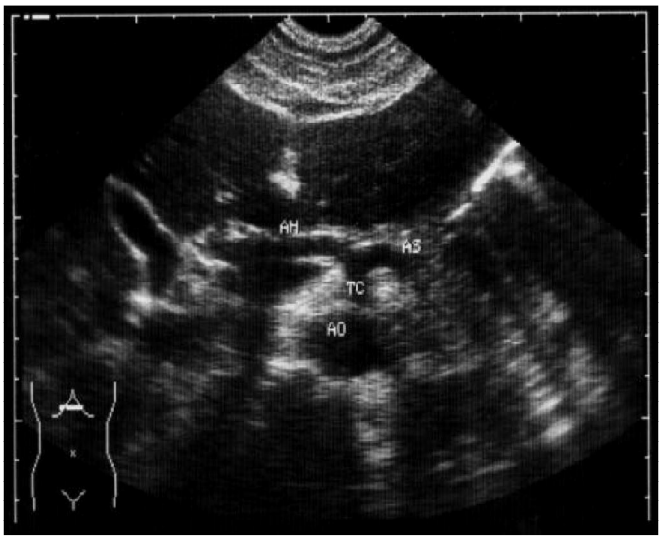


Fig. 3. Emergence of celiac trunk (TC) from aorta (AO), with hepatic artery (AH) and splenic artery (AS)

Between the posterior landmark (the spleno-portal axis) and the anterior landmark (the gastric antrum and the left hepatic lobe) the parenchymal structure of the pancreas is found.

The normal pancreatic parenchyma has an echogenity close to that of the liver (sometimes slightly hypoechoic). In obese patients (fatty loading) or elderly patients (fibrosis), the pancreas will have an increased echogenity. All these appearances are normal, provided that the structure of the pancreatic parenchyma is homogeneous (Fig 4). The Wirsung duct can be visualized particularly in young persons, with a normal diameter of up to 2 mm. Usually only a fragment of the Wirsung duct is seen, rarely its entire length (Fig. 5).

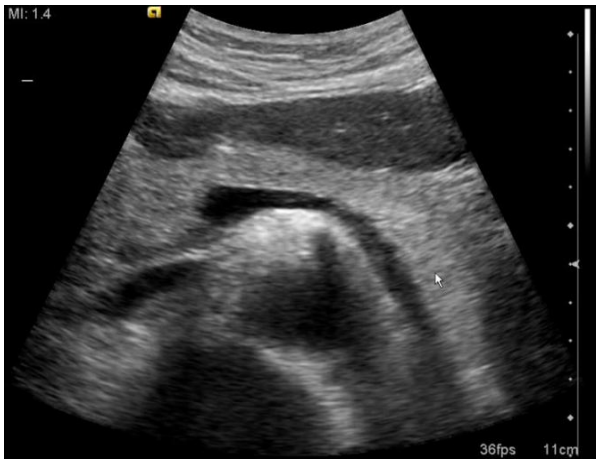


Fig. 4. Normal pancreas– hyperechoic

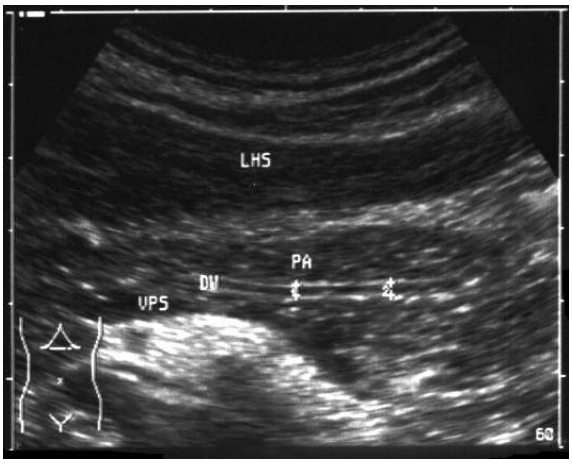


Fig. 5. Normal pancreas with normal Wirsung Duct

Pancreatic examination in transverse section will visualize an important part of the pancreas, but the entire pancreas is almost never seen in one section. This is due to the slightly ascending trajectory of the pancreas tail which is harder to examine because of the interposition of the gastric body. It is sometimes more easily visualized by left subcostal oblique section (Fig. 6). To examine the pancreatic head a sagital section is preferred. Together with the pancreatic head the uncinate processus must be identified, which appears as a parenchymal extension surrounding the portal vein (Fig. 7).

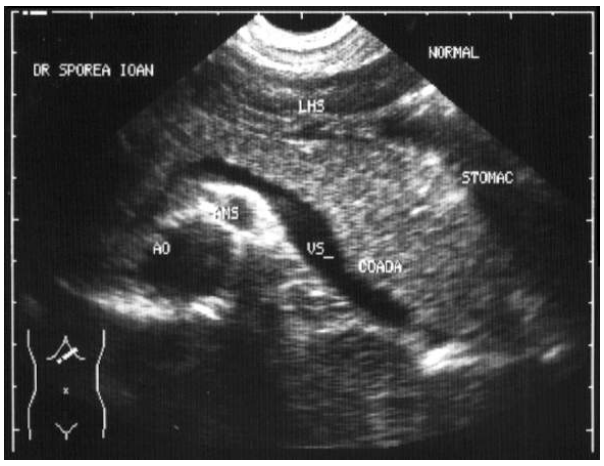


Fig. 6. Pancreatic tail

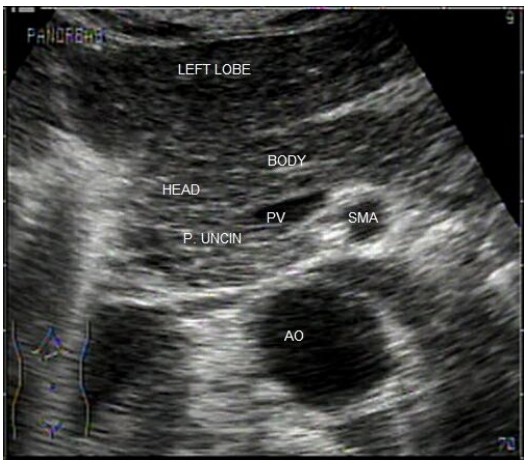


Fig. 7. Uncinate processus (P UNCIN)

Regarding the normal size of the pancreas opinions are divided. We do not consider pancreatic size as very important because of its wide individual variability. The easiest to measure is the body of the pancreas, by antero-posterior measurement in transverse epigastric section the normal diameter being 10-20 mm. The head of the pancreas is considered normal up to 30 mm and the tail of the pancreas up to 20-25 mm.

The ultrasound examination of the pancreas can be performed as a routine procedure, during a routine abdominal ultrasound, or as a targeted procedure, in cases with painful epigastric symptoms. The main pancreatic diseases that will be described in this chapter are: acute pancreatitis, chronic pancreatitis, pancreatic cysts and tumors.

2. Acute pancreatitis

It is an acute inflammation of the pancreas, most frequently generated by alcohol abuse and/or cholelithiasis. It is a potentially severe disease (possible mortality in cases of acute necrotic-hemorrhagic pancreatitis). However, the great majority of acute pancreatitis cases are mild, edematous forms.

The main *causes of acute pancreatitis (AP)* are *acute alcohol consumption* (acute alcoholic pancreatitis) and *cholelithiasis* (acute biliary pancreatitis). Rare causes are: drugs, mumps, severe hypertriglyceridemia, pancreatic trauma, ERCP (endoscopic retrograde cholangio-pancreatography), pancreatic anatomic anomaly (pancreas divisum), familial pancreatitis, etc. The clinical picture of acute pancreatitis is typically characterized by "band like pain" or epigastric pain, often with posterior irradiation, with or without vomiting, the alteration of the general state that can go up to shock. A suggestive prognostic factor for severe evolution is an increased value of C-reactive protein (CRP) greater than 150 mg%.

Useful tests in AP are: abdominal ultrasound (possibly contrast enhanced ultrasound) computed tomography (CT); possibly ERCP (for sphincterotomy, in cases of acute biliary pancreatitis).

The ultrasound appearance of AP is not always very suggestive. In mild edematous forms of AP, ultrasound may not provide diagnostic data. The most typical element of AP is the pancreatic edema, which causes an enlargement of the pancreas (large hypoechoic pancreas) (Fig. 8). In severe cases of AP an enlargement and hyperechoic aspect of the bursa omentalis can be observed (bursa omentalis is a virtual cavity, delimited anteriorly by the stomach and posteriorly by the anterior margin of the pancreas) (Fig. 9). In severe AP left pleural effusion as well as peritoneal effusion in various locations can be seen, also paretic intestinal loops filled with transonic fluid visible in peripancreatic areas, or collections in bursa omentalis (Fig. 10, Fig. 11).

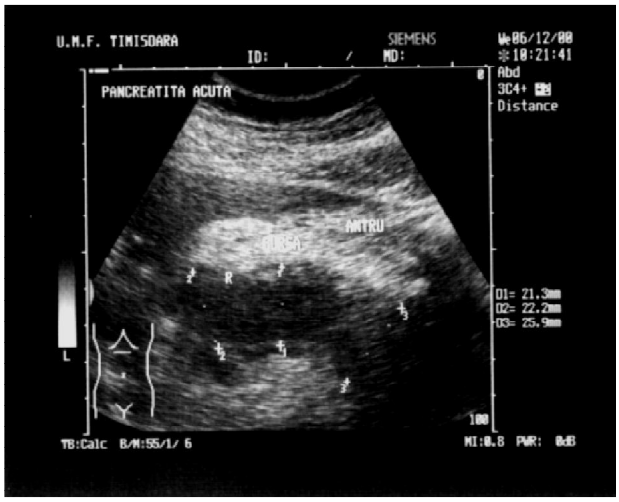


Fig. 8. AP: Hypoechoic pancreas, imprecisely defined

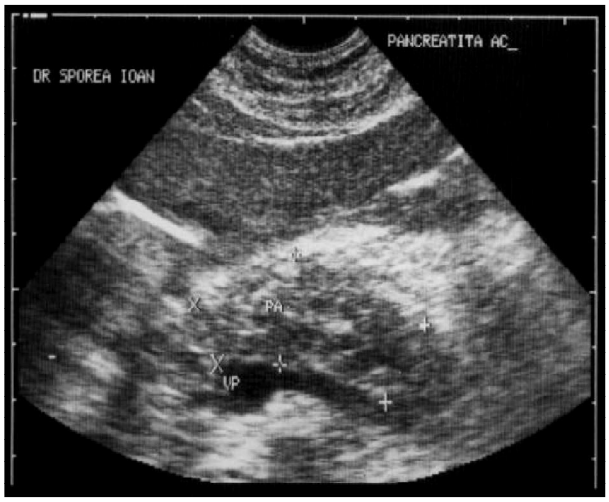


Fig . 9. AP: Hypoechoic pancreas, bursa omentalis

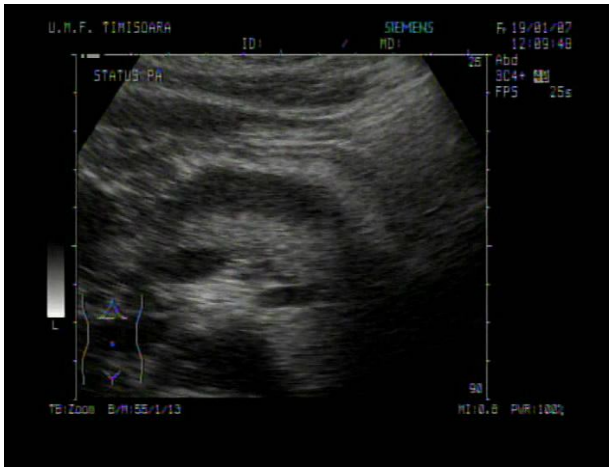


Fig. 10. AP – collection in the bursa omentalis

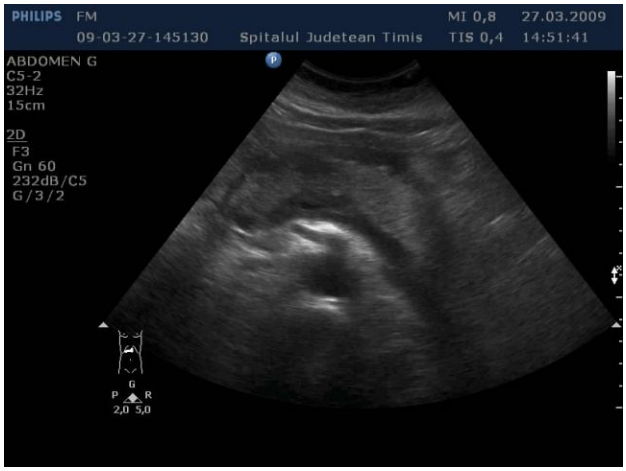
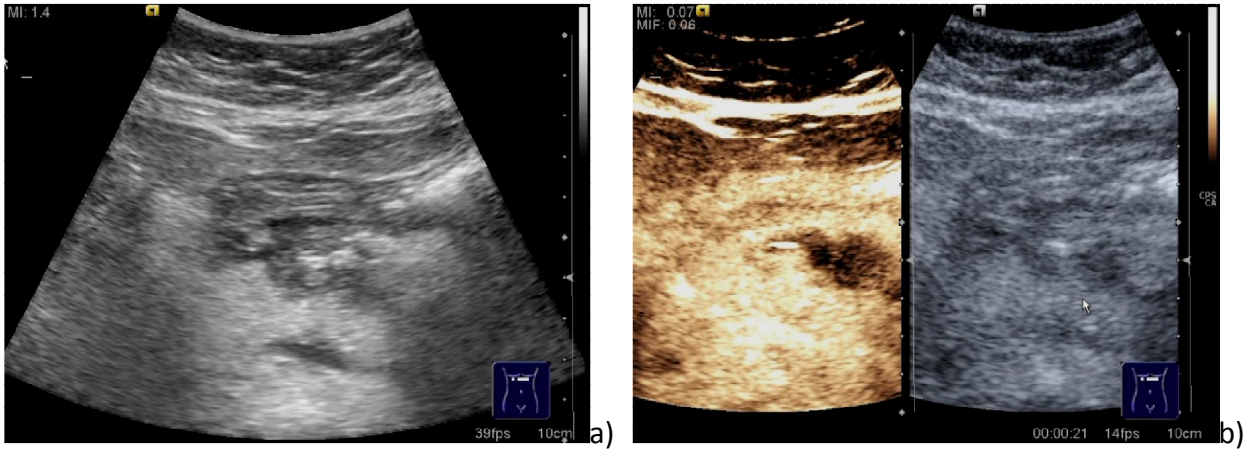


Fig.11. AP – Peripancreatic collection

An important issue is the case of patients with AP in whom the pancreas cannot be adequately assessed by ultrasound (high amount of air in the colon, paretic loops, extreme obesity, intense pain in the superior abdomen on pressure with the transducer, etc.). In these cases, CT is preferred, which will accurately assess the lesions.

US assessment of the gallbladder is useful for defining AP etiology. Mainly, potential gallbladder stones can be seen, which can involve small calculi (micro-cholelthiasis). The evaluation of the main bile duct (MBD) is sometimes difficult, but a potential dilation is not difficult to visualize. The terminal part of the MBD is best seen by endoscopic ultrasound.

Contrast enhanced ultrasound (CEUS) can be performed usually on the 4th day after pain onset and is useful for the evaluation of pancreatic necrosis, in cases with severe acute pancreatitis, but only if the patient has a proper sonographic window. After the injection of SonoVue, the pancreas enhances with the contrast agent in seconds, with the exception of necrotic areas (allowing their assessment) (Fig. 12. a, b).



a) b)

Fig. 12. a) AP standard US – in the pancreatic body, inhomogeneous area; b) AP – CEUS arterial phase, the inhomogeneous area in standard US does not enhance following contrast injection – necrotic area

The contribution of **computer tomography** to staging AP is unquestionable, so that it is recommended in all severe AP cases in which CEUS could not be performed or was irrelevant.

If the echogenity of the necrotic areas is not completely anechoic and a pancreatic abscess is suspected (a hypoechoic/anechoic area in the pancreas in a patient with fever and leucocytosis (Fig 14), ultrasound guided fine needle aspiration biopsy can be performed. Fluid culture or slide examination will be conducted, which will allow to evaluate fluid infection. Also, the collection can be drained by ultrasound guided placement of a drain tube.

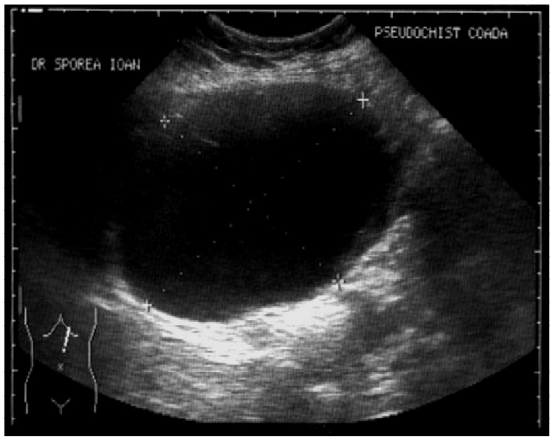


Fig. 13. AP – anechoic area in the tail of the pancreas-pseudocyst?

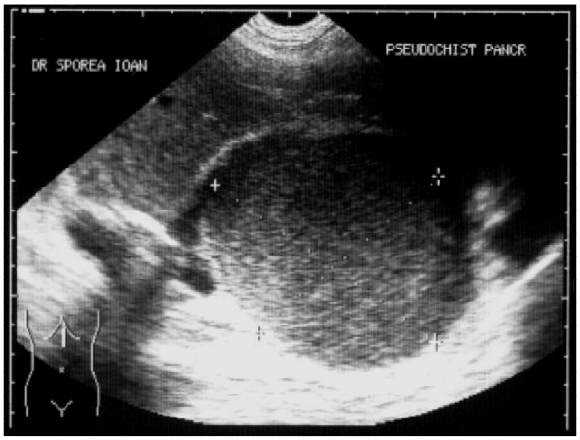


Fig. 14. AP-hypoechoic area in the body of the pancreas - abscess?

3. Chronic pancreatitis

Is a chronic inflammatory pancreatic process that evolves towards the progressive destruction of the organ, accompanied by parenchymal calcifications, along with the dilation of the Wirsung duct due to the presence of calculi. The *etiology* of chronic pancreatitis (CP) involves as the main factor chronic alcohol consumption in pathological doses (higher than 60-70 grams pure alcohol/day in men and 30-40 grams/day in women). Other etiological factors are much less common: hyperparathyroidism, chronic familial pancreatitis, etc.

Abdominal ultrasound is a useful diagnostic method in CP. It may establish the diagnosis of CP in a patient that is frequently asymptomatic or paucisymptomatic (incidental detection), or may be part of the evaluation of a patient with abdominal symptoms, where it will clarify the cause of the disease.

Ultrasound changes in CP are related to the parenchymal structure changes and duct changes. In chronic pancreatitis, the parenchymal structure will be heterogeneous, inhomogeneous (due to fibrotic areas) (Fig. 15). Pancreatic calcifications may occur. These calcifications are usually small, difficult to evidence by ultrasound, but sometimes they are large, generating a posterior shadow (Fig. 16). The pancreatic outline can be irregular. The size of the pancreas can be slightly enlarged or on the contrary, it can be smaller, in atrophic CP.

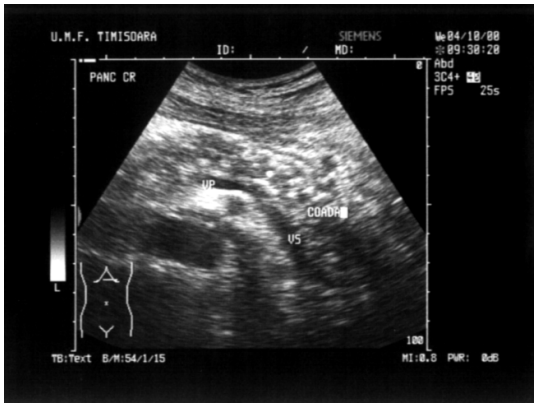


Fig. 15. CP – Heterogeneous pancreas

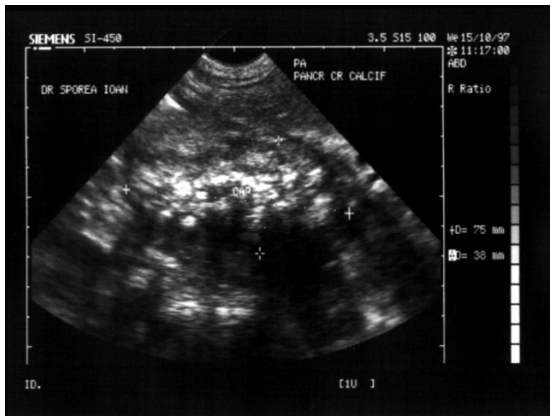


Fig. 16. CP – Pancreas with calcifications

Changes of the Wirsung duct (WD) are key elements for PC. WD appears to be "too well seen", in typical cases with a diameter larger than 2 mm (Fig 17). In severe forms, the WD is 7-9 mm in size (Fig.18), and may almost completely replace the body of the pancreas. In many cases, the WD is irregular, with enlargements and strictures and sometimes stones of variable sizes, up to 10 mm, that generate a marked posterior shadow (Fig. 19, Fig. 20) can be visualized.

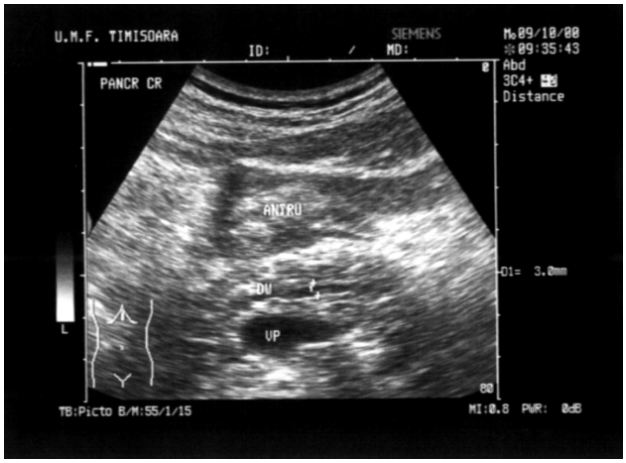


Fig. 17. CP – Large WD – 3 mm

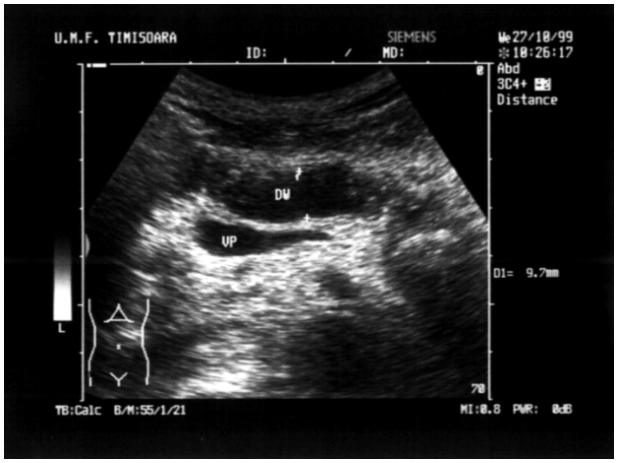


Fig. 18. CP – Large WD – 9 mm

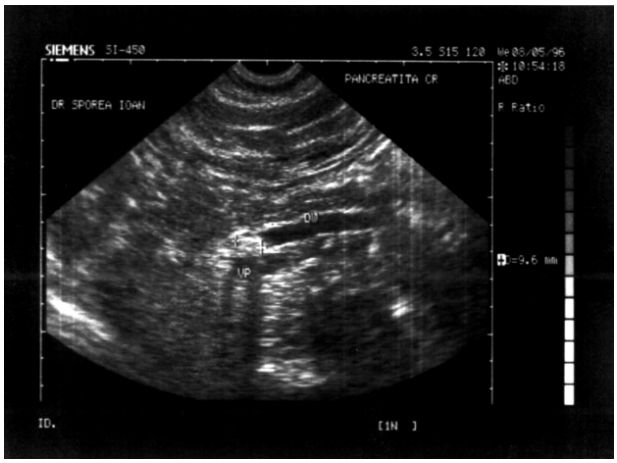


Fig. 19. CP: stone in WD

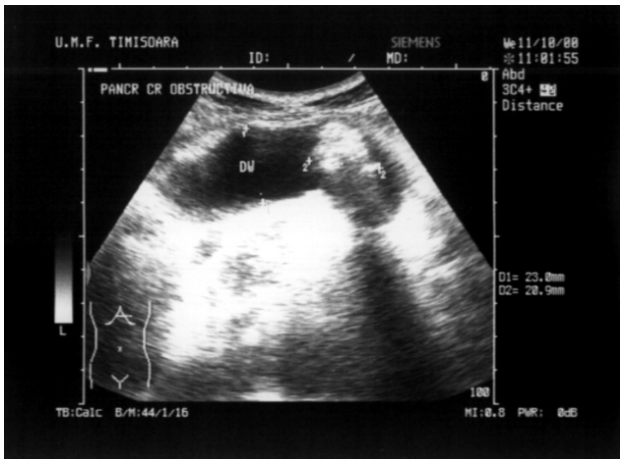


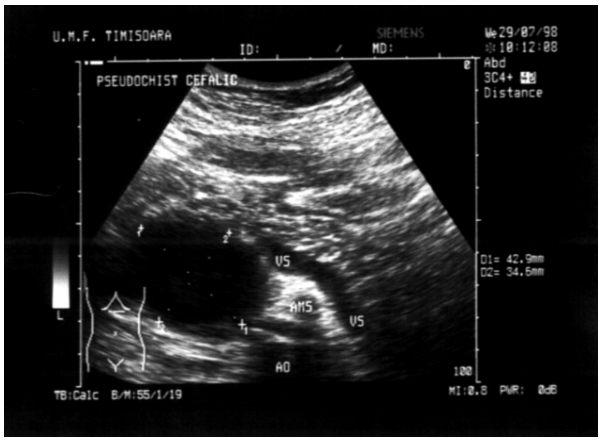
Fig. 20. CP: Large WD with 2 cm stone

It is possible to mistake the spleno-portal axis or the splenic artery with a dilated Wirsung duct. In order to clarify this situation, the spleno-portal axis must be detected, possibly by following the portal vein up to the hilum and the splenic artery from its emergence from the celiac trunk and visualize them by using color Doppler or power Doppler (Doppler absent in WD).

In cases of hypertrophic CP it is relatively difficult to differentiate chronic cephalic pancreatitis from a carcinoma of the pancreatic head. The latter usually appears as a hypoechoic mass, with hypoenhancing at CEUS. CA 19-9 is elevated in pancreatic cancer and is useful for the differential diagnosis.

In all cases of CP, the aspect of the main bile duct must also be evaluated because of its potential compression by a hypertrophic PC and also intrahepatic biliary ducts must be evaluated (whether they are or not dilated).

The presence of pancreatic pseudocysts is relatively common in CP. They appear as anechoic masses, with a definite wall, with different locations and sizes. (Fig. 21, Fig. 22).



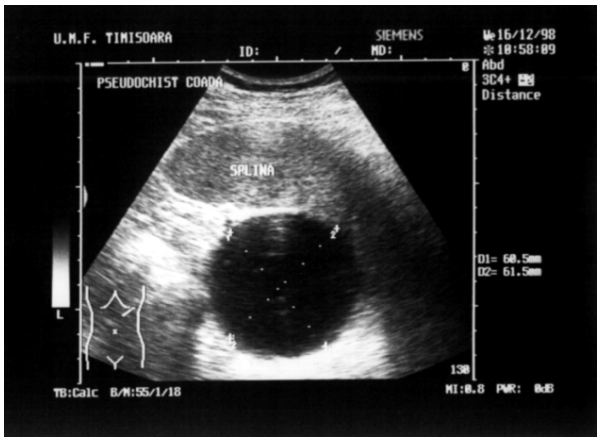


Fig. 21. Pancreatic head pseudocyst **Fig. 22. Pancreatic tail pseudocyst**

Cysts once diagnosed can be measured and followed-up (growth or resorbtion). It is also possible to perform ultrasound-guided puncture, to differentiate them from cystadenocarcinomas, or for therapeutic reasons. The pseudocysts of the body of the pancreas are easy to diagnose, even by a beginner in ultrasound, but those of the head and tail of the pancreas may raise ultrasound diagnostic problems. For the head of the pancreas, sections perpendicular on the right costal margin are useful, while the evaluation of the pancreatic tail in procubitus, with the visualization of the caudal pseudocyst through the ultrasound window of the left kidney, may be helpful.

The ultrasound differential diagnosis of CP is made with acute pancreatitis (where the pancreas is enlarged and hypoechoic); with Vater's ampulloma (in which the dilation of the WD is usually accompanied by the concomitant dilation of the MBD); with retroperitoneal tumors situated in the upper abdomen; or with pancreatic tumors, intraductal papillary mucinous tumors (IPMT) of the pancreas that should not be overlooked.

Differential diagnosis with pancreatic tumors is difficult. There may be problems regarding the differentiation between hypertrophic chronic cephalic pancreatitis and pancreatic head tumor (the latter is most frequently hypoechoic); CEUS, endoscopic ultrasound (EUS) or EUS elastography are used for differentiation. Also, it is difficult to differentiate a pancreatic pseudocyst with septa from a pancreatic cystadenoma or cystadenocarcinoma or from a mucinous pancreatic tumor.

Computed tomography is the most efficient method for the evaluation of the pancreas in chronic pancreatitis, which can evaluate the presence of calcifications, of hypodense areas suspected of malignancy and of pseudocysts.

EUS is also a sensitive method of evaluation in CP, showing fine details as local changes in the pancreatic structure or discrete dilations of the WD. Also ultrasound guided biopsy will be possible in any lesion suspected of malignancy.

4. Pancreatic tumors

Pancreatic tumors include all tumors that start in pancreatic tissue. They can be benign or malignant. Among the latter the most common is pancreatic carcinoma, but there are also neuro-endocrine pancreatic tumors, cystic neoplasms and finally ampulary tumor (ampuloma).

a) Pancreatic carcinoma is more frequent in men than in women and it usually develops in patients older than 60 years. The ultrasound appearance is of a mass most frequently hypoechoic, with variable sizes (1-5 cm) (Fig. 23, Fig. 24, Fig. 25) generally poorly delimited (Fig. 23, Fig 25), sometimes inhomogeneous (large tumors in particular), and frequently invading adjacent vessels (Fig. 26). This invasion can be evidenced using power Doppler or contrast enhanced ultrasound, and is useful for preoperative evaluation.

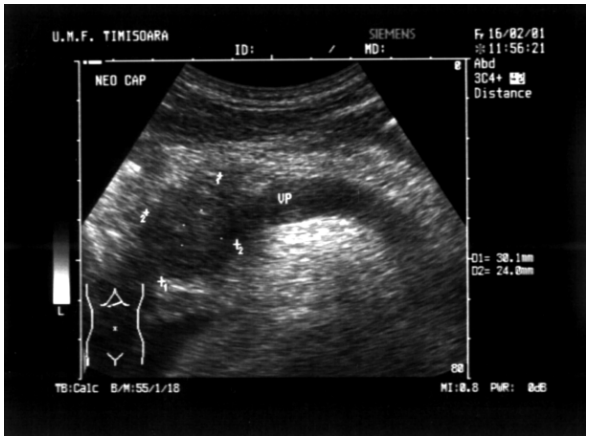


Fig. 23. Pancreatic head tumor

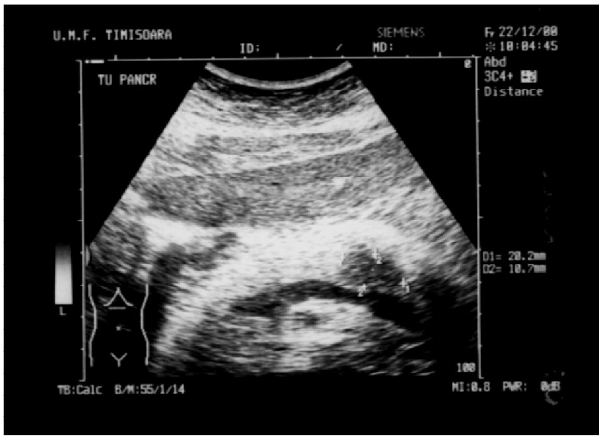


Fig. 24. Small tumor of pancreatic body

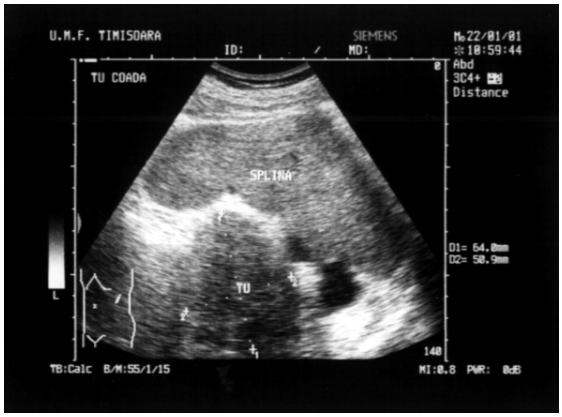


Fig. 25. Pancreatic tail tumor

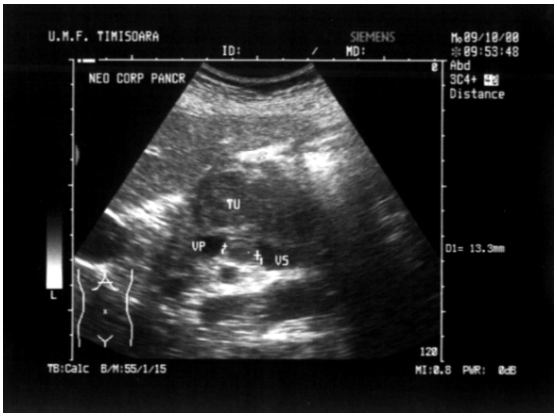


Fig. 26. Pancreatic body tumor– invasion of spleno-portal axis

Frequently we may be confronted with an elderly patient with a painless jaundice syndrome. Ultrasound will relatively easy establish the diagnosis of obstructive jaundice, but evidencing the cause of the obstruction by ultrasound is more difficult, even for an experienced examiner (hypoechoic pancreatic head mass that obstructs the MBD) (Fig. 27).

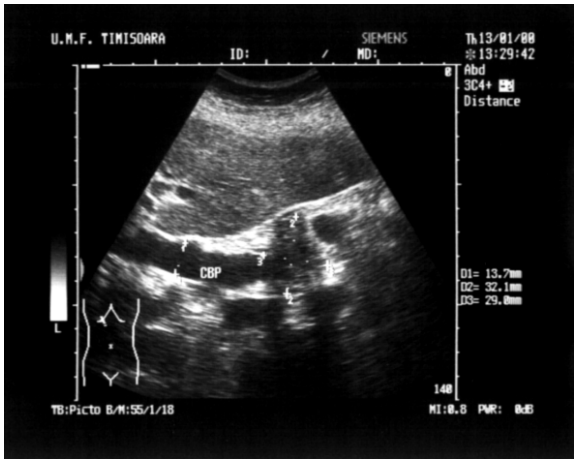


Fig. 27. MBD that ends in a pancreatic head tumor

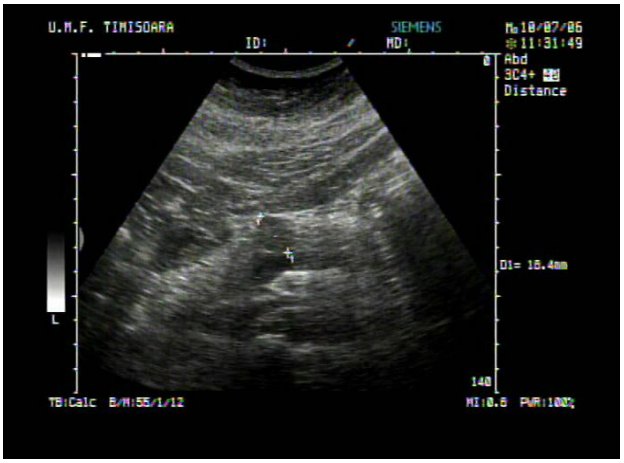


Fig. 28. Small neuro-endocrine body tumor

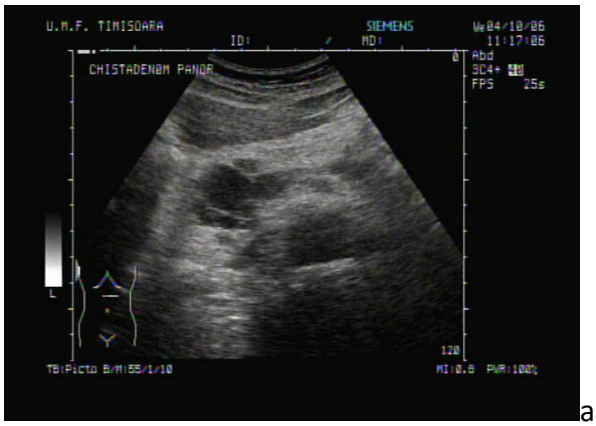
b) Endocrine pancreatic tumors are relatively rare. The main endocrine pancreatic tumors are: gastrinoma, insulinoma, glucagonoma, somatostatinoma, VIPoma. One of their characteristics is the high hepatic metastasis rate, even when they are small, liver metastasis are often firstly diagnosed.

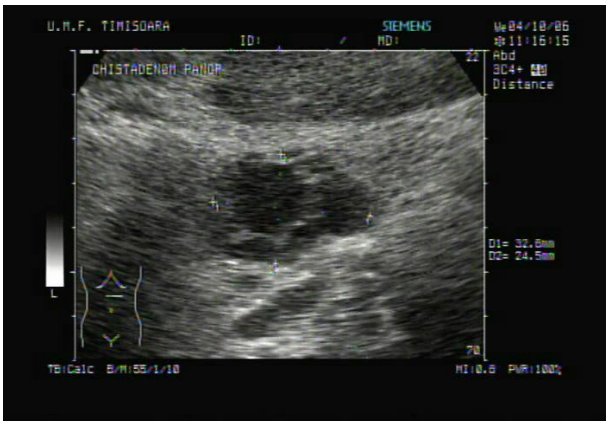
The **ultrasound appearance** of endocrine tumors is that of pancreatic masses, generally well delimited, of small size (5-20 mm), hyper- or hypoechoic (Fig 28). An endocrine tumor is diagnosed by ultrasound quite rarely; it is most frequently evidenced by CT and particularly, by EUS, the method of choice which allows ultrasound guided biopsy that will certify diagnosis. The ultrasound or EUS appearance does not allow the differentiation of a pancreatic carcinoma from an endocrine tumor. Only biopsy (most frequently EUS guided) can make this differentiation. CEUS reveals a hypervascular tumor, unlike adenocarcinoma, which is usually hypovascular.

c) Cystic pancreatic tumors are relatively rare. An anechoic pancreatic image detected by ultrasound is in most cases a pancreatic pseudocyst. If there is no history of acute pancreatitis or imaging signs of chronic pancreatitis, the problem of a cystic pancreatic tumor must be raised.

Cystic pancreatic tumors can be of two types: microcystic adenoma and mucinous cystadenoma.

Microcystic adenoma is a benign tumor formed by multiple small cysts, less than 2 cm in size, and is most frequently found in the head of the pancreas, usually an accidental discovery. The ultrasound aspect is of conglomerate multiple small anechoic images (Fig 29), usually so small that they cannot be individualized, thus generating an inhomogeneous, heterogeneous aspect.



a)


b)

Fig. 29. a) Cephalic pancreatic microcystic adenoma (transonic conglomerated images). b) the same image with higher magnification.

Mucinous cystadenoma or pancreatic cystadenoma (which can sometimes be malignant: cystadenocarcinoma) appears in ultrasound as a anechoic image, usually larger than 2 cm in diameter, uni or multilocular, generally located in the tail of the pancreas. The presence of a hypoechoic rather than anechoic appearance or of inner excrescences suggests the diagnosis of carcinoma (Fig.30, Fig. 31). Ultrasound guided percutaneous or EUS guided biopsy from the cyst is useful; it will evidence a free-running mucinous fluid that will certify the diagnosis. Unlike post PA pseudocysts where the aspirated fluid is not mucinous, mucinous cystadenomas have surgical indication due to the high risk of malignancy. The diagnosis of cystadenocarcinoma is certified by dosing the carcinoembryonic antigen (CEA) in the aspirated fluid.

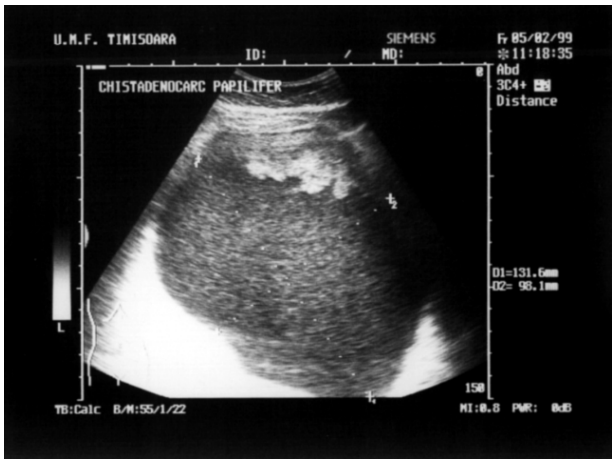


Fig. 30. Pancreatic tail cystadenoma.

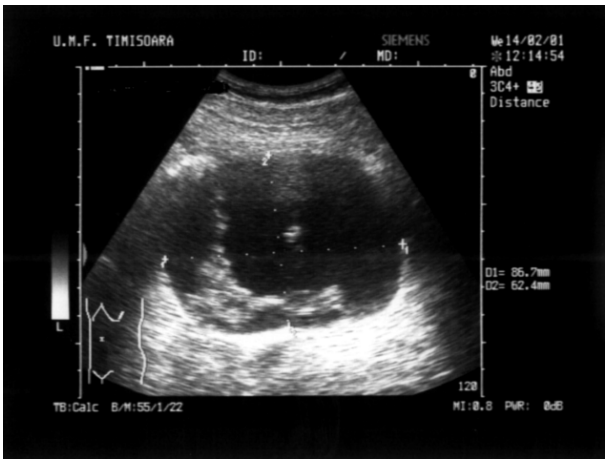


Fig. 31. Pancreatic tail cystadenoma

Other imaging techniques that help diagnosis in cystic pancreatic tumors are CT, MRI, ERCP and particularly, EUS. The last will easily visualize microcysts in microcystic adenoma or will evidence excrescences inside the cyst in pancreatic cystadenocarcinoma.

Chapter 8.

Ultrasound of the spleen

1. Normal spleen

The spleen, the largest lymphatic organ of the body, located in the splenic space, appears in ultrasound as a parenchymal structure with echogenity close to that of the liver. The best ultrasound evaluation is through left intercostal sections, if possible in deep blocked inspiration, so that the spleen (located under the diaphragm, entirely into the thorax) is accessible to the examination. Frequently the spleen has the shape of a crescent or of a coffee bean, with the maximum longitudinal diameter of 12 cm. Normal shapes are also globular, triangular shape or the shape of a comma. The spleen has a convex face, in contact with the diaphragm and a concave face, median oriented that includes the splenic hilum. The splenic upper pole is related to the diaphragm and the lower pole is in contact with the left colon.

Accessory spleens may exist: round or oval structures with similar echogenity to that of the spleen, located near the hilum or to one of the spleen poles. Usually there is a unique accessory spleen, rarely multiple, with diameter approximately 10-25 mm. An accessory spleen is completely asymptomatic, is often an accidental discovery and occurs in 5-15% of the population. The ultrasound differential diagnosis of accessory spleen should be made with enlarged lymph nodes in hematological diseases, with pancreatic tail tumors (hypoechoic), or possibly with aneurismal thrombosis of the splenic vein. The use of CEUS may help differential diagnosis, because following contrast bolus, the accessory spleen enhances similarly to the adjacent spleen.

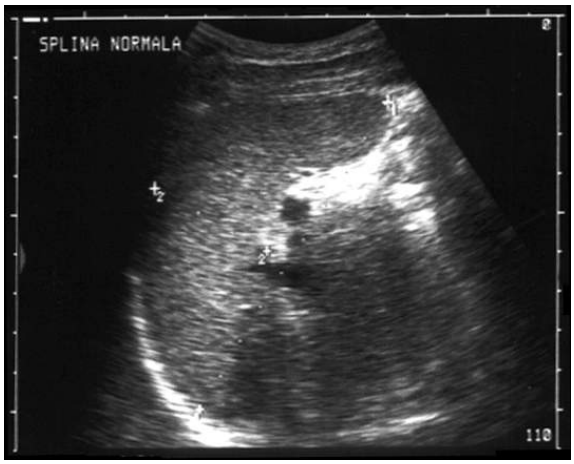


Fig. 1. Normal spleen

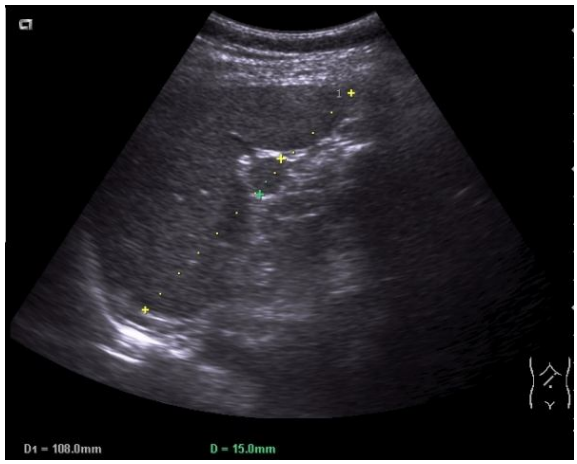


Fig. 2. Normal spleen with an accessory spleen in the hilum

The examination of the spleen will be conducted so as to include both splenic poles in the ultrasound plane, allowing the accurate measurement of both longitudinal and transverse diameters. The spleen echogenity is usually similar to that of the liver, or slightly more hypoechoic, but homogenous. The spleen may be heterogeneous in hematological diseases (lymphoma), in portal hypertension, in granulomatous diseases, amyloidosis, etc (Fig. 3).

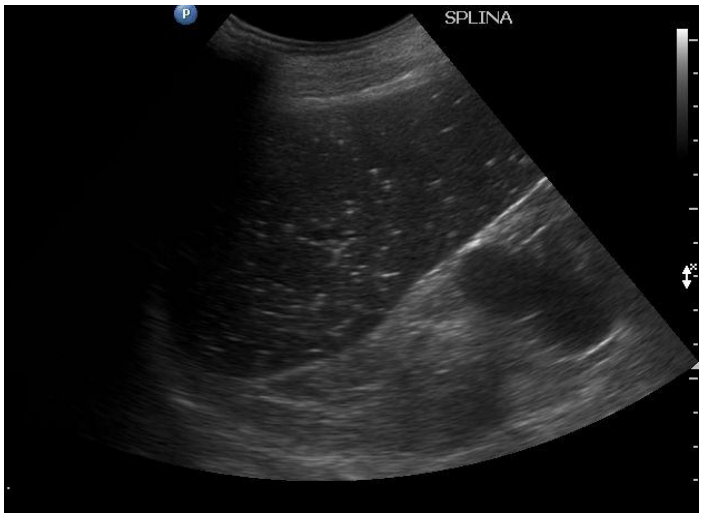


Fig. 3. Splenomegaly with calcifications

From a clinical point of view, the ultrasound evaluation of the spleen is important in the context of hematological, infectious and liver diseases, after abdominal trauma or surgery, in cases of fever with unknown cause.

2. Splenomegaly

Splenomegaly is defined as an enlargement of the spleen exceeding 12 cm along its long axis. Some authors consider a normal spleen size up to 11 cm, others, up to 13 or even 14 cm, but the majority of the ultrasonographists consider 12 cm as being the upper limit of normality. Of course there are variations depending on height and gender. A 12 cm spleen does not have the same significance in a 195 cm tall man weighing 110 kg and in a 155 cm tall woman weighing 50 kg.

In current clinical practice, splenomegaly is caused mainly by ***hepatic*** and ***hematologic*** disorders. Splenomegaly in infectious or parasitic diseases is exceptional in Romania. *The clinical signs* are most frequently absent. In some cases, splenomegaly is discovered incidentally. At other times, there are signs of an underlying hepatic disease (jaundice of the skin and sclera, ascites, collateral abdominal circulation, gingival bleeding or epistaxis), or of a hematologic disease (anemia, asthenia, fever, peripheral enlarged lymph nodes). Important enlargements of the spleen may cause painful discomfort or a sensation of weight in the left hypochondrium.

The ultrasound appearance is of an increase in the volume of the organ. There may be mild splenomegaly (up to 13-14 cm), moderate splenomegaly (15-16 cm), and important splenomegaly (over these values). (Fig. 4, Fig. 5). Regarding the alteration of the spleen echogenity, the hepatic or hematologic etiology cannot be established based on echogenity changes alone.

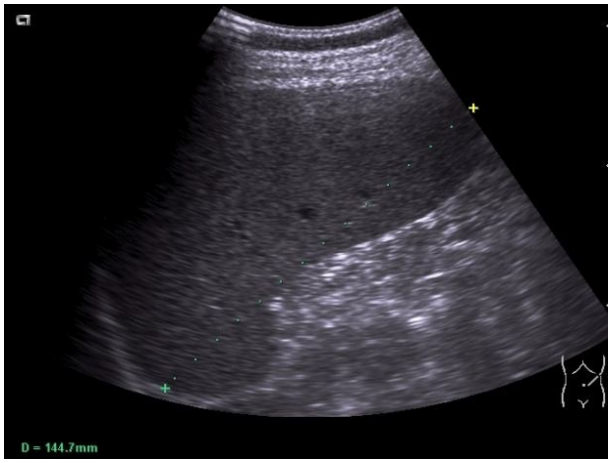


Fig. 4. Moderate splenomegaly – 144 mm

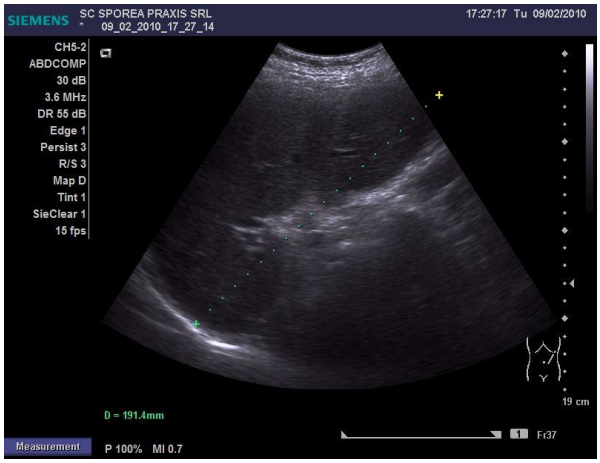


Fig. 5. Important splenomegaly - 191 mm

Ultrasound will evaluate the possible signs of portal hypertension (e.g. splenic varices) (Fig. 6) or liver cirrhosis (hepatic heterogeneity – Fig.7, nodule development on the liver surface, doubled gallbladder wall, ascites – Fig. 8), in this case it is clear that splenomegaly is due to chronic hepatic disease. Abdominal lymphadenopathies (celiac and aorto-caval enlarged lymph nodes) with dimensions of 2-5 cm (Fig. 9) and a rapid growth in size of the spleen are suggestive of a hematological malignancy.

The power Doppler ultrasound examination of the spleno-portal axis is useful for visualizing a possible thrombosis with secondary splenomegaly.

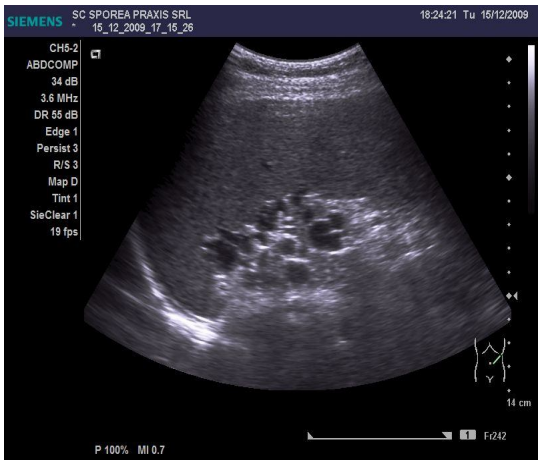


Fig. 6. Splenic varices

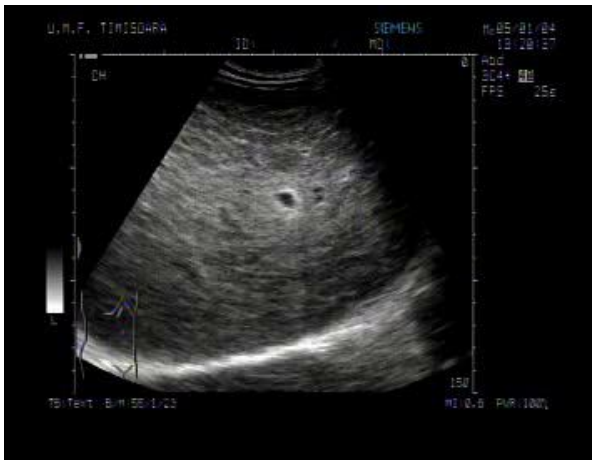


Fig. 7. Hepatic heterogeneity – liver cirrhosis

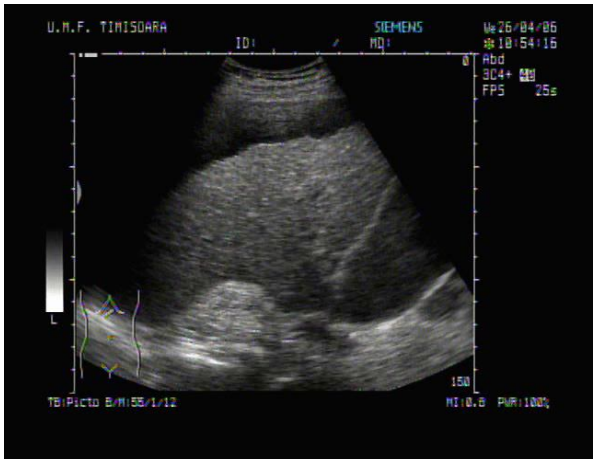


Fig. 8. Irregular surface of the liver, ascites – liver cirrhosis

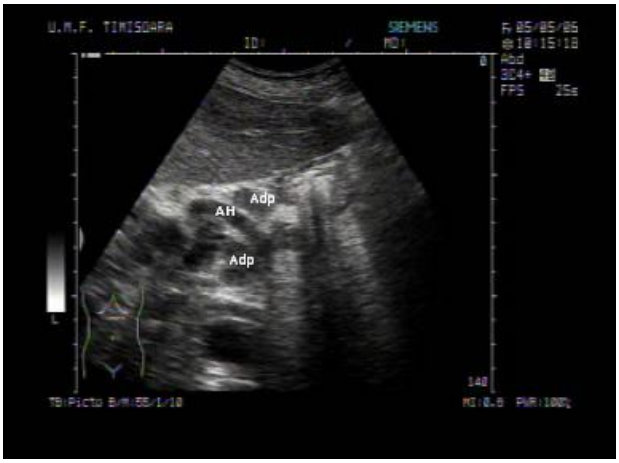


Fig. 9. Epigastric lymphadenopathies – lymphoma

3. Trauma of the spleen

Over the past period, as a result of the increase in the number of road traffic accidents, the number of splenic traumas has also increased. Thus, spleen ruptures or intrasplenic or subcapsular hematomas have become more frequent. The ultrasound examination of a patient with spleen trauma is difficult (patient in a severe or critical state, who is sometimes polytraumatized and cannot cooperate in deep inspiration or stopping of breathing). At the same time, the potential severe evolution of an undiagnosed splenic lesion (splenic hematoma with rupture in two phases) may endanger the patient's life. For this reason, if there is no certainty that the spleen was completely and correctly visualized by ultrasound, an abdominal CT with contrast will always be recommended. However, if the ultrasound window is good and the ultrasonographist is experienced, CEUS can be done, thus avoiding patient irradiation. CEUS will properly identify the area of hematoma (no enhancement following contrast), and will subsequently allow monitoring if surgery is not performed.

The ultrasound examination of a patient injured as a result of road traffic trauma, falls or blows, starts by looking for fluid in the peritoneal cavity. Fluid may be detected in the Douglas space, perisplenic, or in the Morrison space (hepatorenal recess), with a hypoechoic rather than anechoic appearance. In case of uncertainty, ultrasound guided exploratory paracentesis can be performed for evidencing blood.

The evaluation of the splenic space may reveal a completely normal spleen or pathological changes. It is important to visualize each region of the spleen and to verify the integrity of the splenic capsule. Omitting the scanning of either pole may lead to missing a pathological area. The diaphragmatic face of the spleen (the convex face) should be attentively

scanned, since it can sometimes be masked by the air from the base of the left lung. Alternating intercostal spaces, examining both in supine and in right lateral decubitus can minimize this risk.

Spleen rupture involves, in addition to hemoperitoneum, the appearance of a discontinuity in the splenic capsule, with the presence of a poorly circumscribed hypoechoic peripslenic hematoma. Subcapsular hematoma has variable sizes and appears as a hypoechoic crescent surrounding the spleen. Intrasplenic hematoma appears as a poorly circumscribed hypoechoic area, situated inside the organ. There is a risk of a two phase rupture of the subcapsular hematoma, with severe secondary hemorrhage.

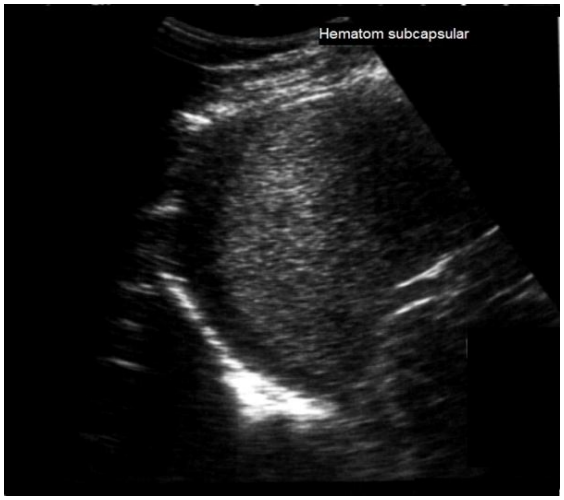


Fig. 10. Subcapsular hematoma

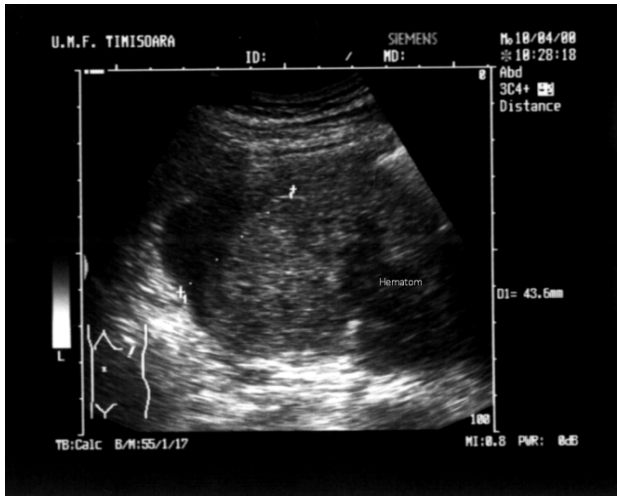


Fig. 11. Intrasplenic and perisplenic hematoma

4. Splenic infarction

Splenic infarction occurs more frequently in patients with important splenomegaly, due to the interruption of blood flow to a certain part of the spleen. Splenic infarction is clinically translated by sudden pain in the left hypochondrium, more or less intense. In ultrasound examination, splenic infarction appears as an iso or hypoechoic triangular area, with the tip to the splenic hilum and the base to the splenic capsule (Fig. 12). In CEUS, splenic infarction will appear as an unenhancing area as compared to the surrounding tissue (in all vascular phases) (Fig.13) with poorly delimited or clear edges. In the arterial phase, the amputated artery may be observed at the tip of the infarction zone, and also a rim enhancement of the infarction area may be seen. In atypical, round or oval infarctions, the diagnosis is suggested by the lack of contrast intake. A massive infarction comprising almost entirely the spleen will appear as fully unenhancing as compared with the adjacent kidney.

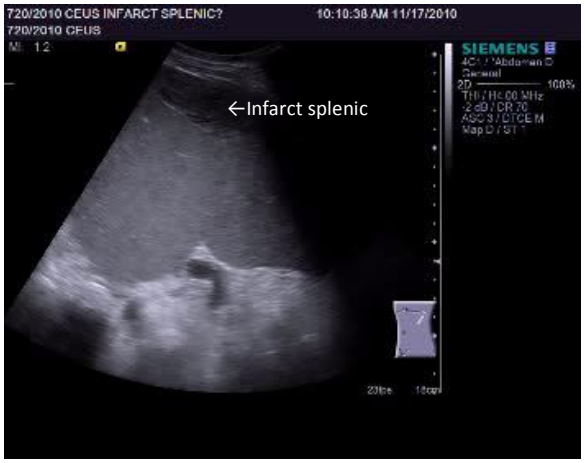


Fig. 12. Splenic infarction – standard US

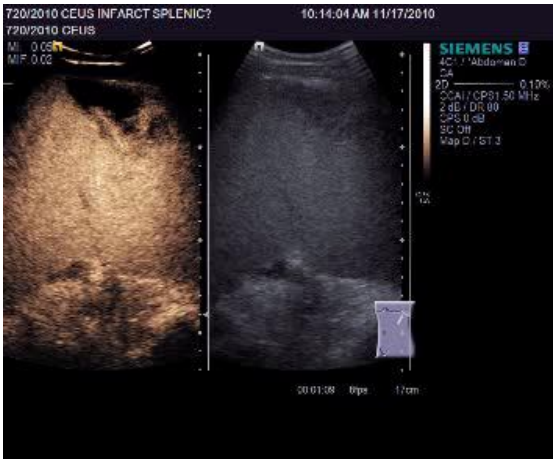


Fig.13. Splenic infarction – CEUS (unenhancing)

5. Abscess of the spleen

A splenic abscess is defined as a purulent collection in the splenic space. It can be secondary to surgery, to splenic trauma (infected hematoma), or it may develop during the evolution of bacterial endocarditis. The clinical picture is frequently septic, with fever, chills, altered general state.

The ultrasound appearance is that of a poorly delimited hypoechoic mass (Fig.14). Splenic abscesses can also be inhomogeneous or, more rarely, hyperechoic. The ultrasound differential diagnosis is made with splenic tumors, splenic cysts, splenic infarction or a splenic hematoma. Clinical information is important as the septic state is highly suggestive for the diagnostic. CEUS and CT with contrast, possibly diagnostic fine needle aspiration are extremely useful for the final diagnosis.

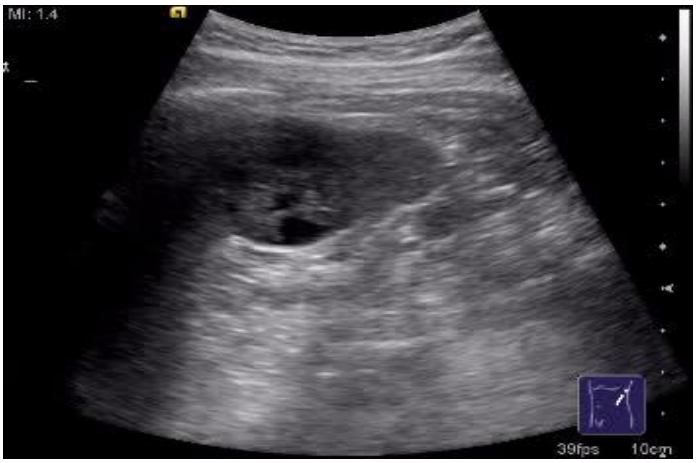


Fig. 14. Splenic abscess (slightly hypoechoic area, including anechoic areas in the spleen concavity)

6. Splenic cysts

Splenic cysts are not very frequent. In ultrasound they appear as anechoic areas in the spleen. They can be hydatid cysts (rare, as a result of Echinococcus granulosus infection in the spleen) or non-parasitic cysts.

Splenic hydatid cysts have a thick wall, and daughter vesicles (thick inner septa) can be frequently seen – similar appearance with hydatid cyst in the liver. The serological test for echinococcosis is usually positive.

Non-parasitic cysts have a fine wall and anechoic content (Fig. 15), rarely with fine inner septa (Fig. 16) - similar appearance with hepatic biliary cysts. Posterior enhancement behind the cysts is present. They are not symptomatic and can rarely become complicated by intracystic hemorrhage.

Both hydatid cysts and non-parasitic cysts are non-enhancing in contrast imaging (CEUS and CT).

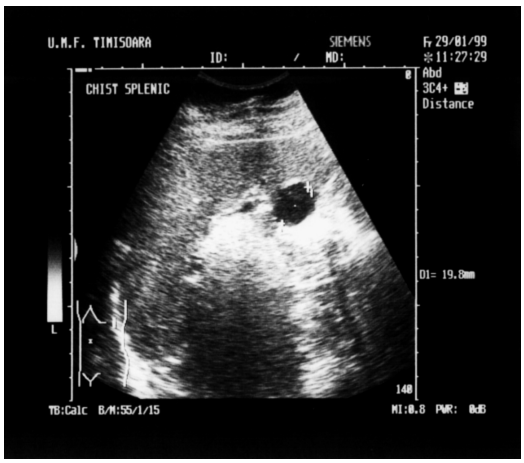


Fig. 15. Small splenic cyst – upper pole

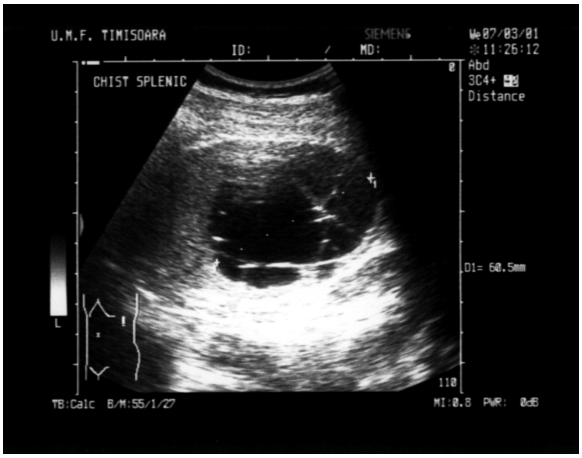


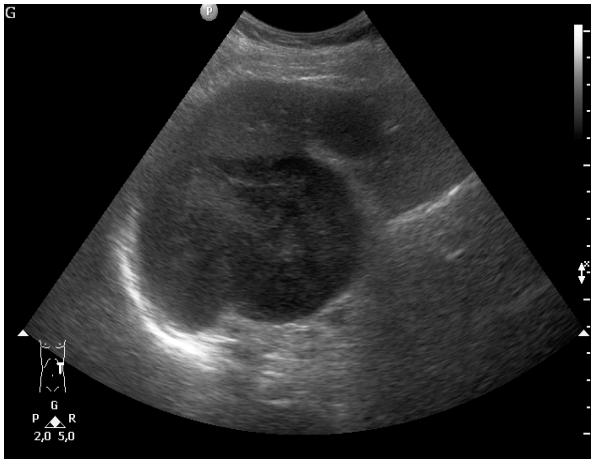
Fig. 16. Splenic cyst with fine inner septa

7. Tumors of the spleen

In clinical practice, splenic tumors are relatively rare. Frequently they are malignant tumors, particularly lymphomas and sarcomas. Among benign tumors, hemangiomas are the most common.

The ultrasound appearance of splenic lymphomas is generally hypoechoic, frequently inhomogeneous and poorly circumscribed. It can be the first discovery of the disease or can be detected in a patient with known Hodgkin or non-Hodgkin lymphoma. The detection of splenic lesions will be followed by search for potential enlarged abdominal lymph nodes.

Other malignant splenic tumors are sarcomas (Fig. 17) and metastases (Fig. 18).



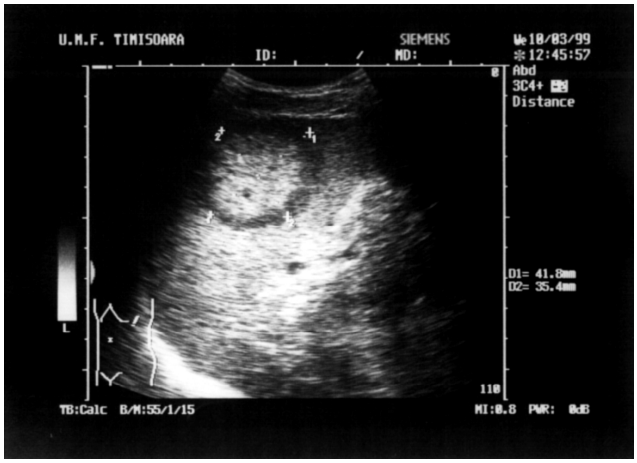


Fig. 17. Spleen sarcoma

Fig. 18. Splenic metastasis

Splenic hemangiomas have a hyperechoic appearance, are well defined, with similar appearance as hepatic hemangiomas (Fig. 19).

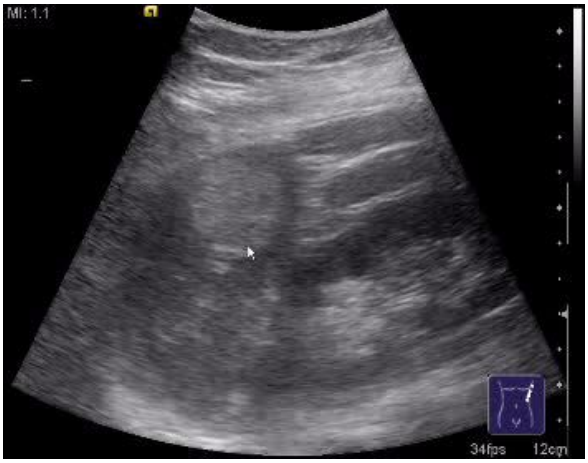


Fig. 19. Splenic hemangioma

The ultrasound differential diagnosis should be made with splenic abscess, hematoma and infarction.

In clinical practice, the detection of a splenic mass raises serious differential diagnosis problems. This is frequently a malignant lesion, and the other diagnostic methods (CT, MRI) provide no additional information. If imaging fails to clarify a splenic mass, ultrasound guided biopsy from the mass may be an option, hemorrhagic accidents after fine needle splenic biopsy being relatively rare (approximately 1%).

Chapter 9.

Ultrasound of the kidneys

1. Normal kidneys

Ultrasound is currently the most frequently used imaging method used for the investigation of the kidney. Intravenous urography is mainly used for investigating the functional aspect of the kidney, while CT and MRI are for the differential diagnosis of tumor masses.

The kidneys are retroperitoneal organs, with sizes of 10-12cm/5-6 cm/3 cm. Starting from the average normal size of these organs, smaller or larger kidneys can be suggestive for a diatincive kind of renal pathology. Renal ultrasound examination is performed with standard 3.5 MHz, preferably convex transducers. The renal ultrasound approach can be through the loins (with the patient in ventral decubitus), by lateral approach (right lateral decubitus for the examination of the left kidney, while scanning is performed through the left lateral abdominal region, and viceversa for the right kidney), or through sagittal sections in a patient in dorsal decubitus. Generaly, the right kidney is easier to visualize in lateral sections or with the patient in dorsal decubitus, using the liver as an acoustic window. For the left kidney, examination is easier in lateral or dorsal sections. Additional intercostal sections are often used for renal ultrasound examination.

The normal ultrasound anatomy of the kidney includes the evaluation of the pyelum and of the parenchyma (cortex). The normal pyelum is hyperechoic and the cortex is hypoechoic (Fig.1). The ultrasound differentiation between the cortex and the medulla is possible only in children and thin persons. In current ultrasound practice, this distinction is not possible, so only the pyelum and the parenchyma will be described when assessing the kidney.

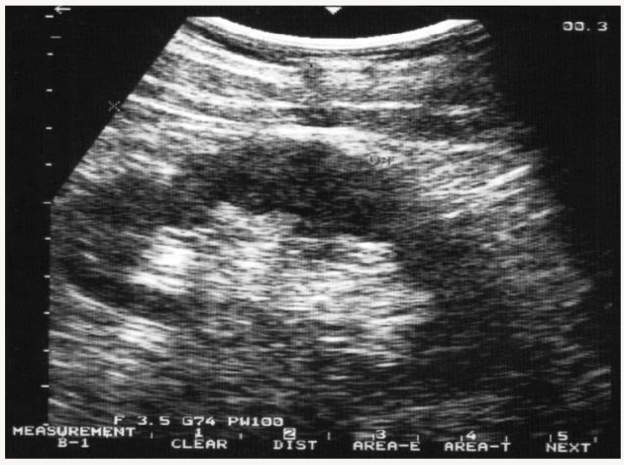


Fig. 1. Normal aspect of the kidney

The measurement of the renal size is useful in some renal disorders. The size of the kidneys decreases with age (renal senescence) or in chronic renal failure; the kidneys can be enlarged in acute renal failure, in some diseases such as amyloidosis or diabetes mellitus, etc.

Renal **ultrasound examination** will have to answer the following questions:

– are the kidneys present bilaterally (single congenital kidney)?

– do the kidneys have a normal size, shape and location?

– are there any changes in renal echogenicity (like in CRF)?

– are there kidney stones, and if so, are these obstructive (hydronephrosis)?

– are there tumor or cyst formations in the kidney? In case of a tumor, locoregional invasion will be established, and for cysts, it will be determined whether they are isolated or part of a polycystic disorder: renal or hepato-renal or hepato-renal-pancreatic.

When discussing renal ultrasound examination, some normal entities that can pose difficult differential diagnosis problems should be described:

a) Renal fetal lobulation may persist in the adult and will generate a bosseled renal outline on ultrasound (Fig.2), and it should be differentiated from a tumor or cyst is raised.

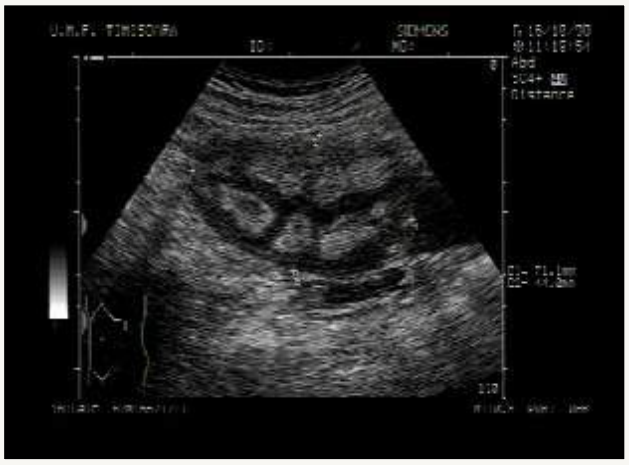


Fig. 2. Renal fetal lobulation in an adult

b) A renal anomaly termed **medio-renal bulge** or ”dromedary hump” is frequently found in ultrasound practice. This is an excrescence in the mid-kidney that will have the same echogenicity as the renal parenchyma and cannot be delimited from the latter (Fig.3). Differentiation will be made with a renal tumor (which generally has a different echogenicity from that of the kidney and can be delimited). In these cases, contrast enhanced ultrasound is extremely useful, because renal anomalies will behave like the renal parenchyma, while a renal tumor will have a completely different CEUS behavior (hyper pr hypoenhancement following contrast).

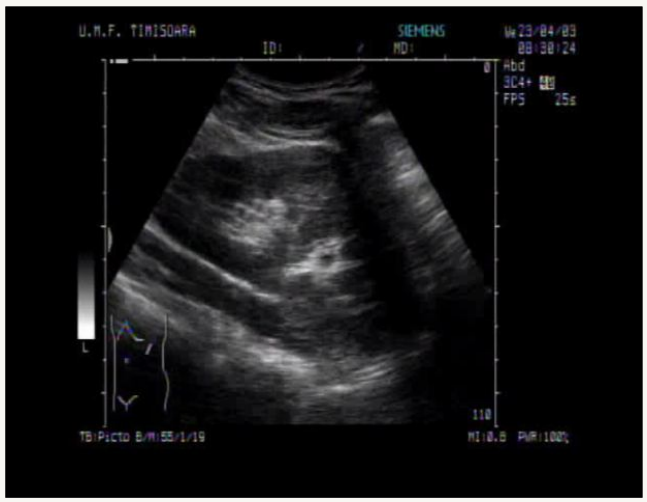


Fig. 3. Medio-renal bulge

c) Bertin's column hypertrophy may generate a renal mass effect. It is a hypoechoic mass that protrudes from the cortex towards the pyelum ((Fig. 4. Fig. 5)). It has an identical echogenicity to that of the cortex, it continues the renal cortex, is well delimited towards the pyelum and is less than 3 cm in size. Ultrasound differentiation is made with a renal tumor, which is poorly circumscribed and has a different echogenicity from that of the cortex. In these cases, contrast enhanced ultrasound is extremely useful, because Bertin's columns will behave like the renal parenchyma, while a renal tumor will have a completely different CEUS behavior (hyper pr hypoenhancement following contrast).

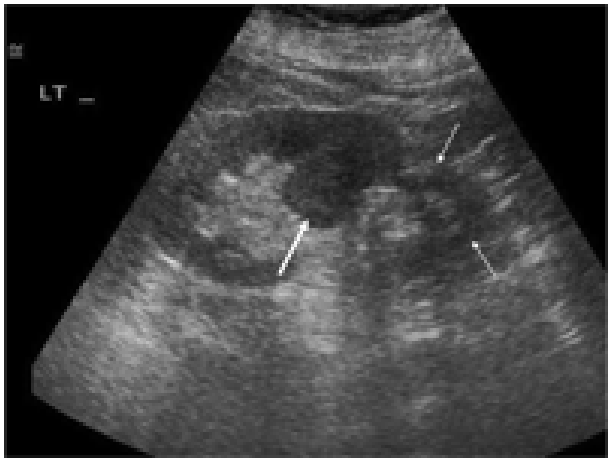


Fig. 4. Bertin column (large arrow) and chronic pyelonephritis changes (small arrows)

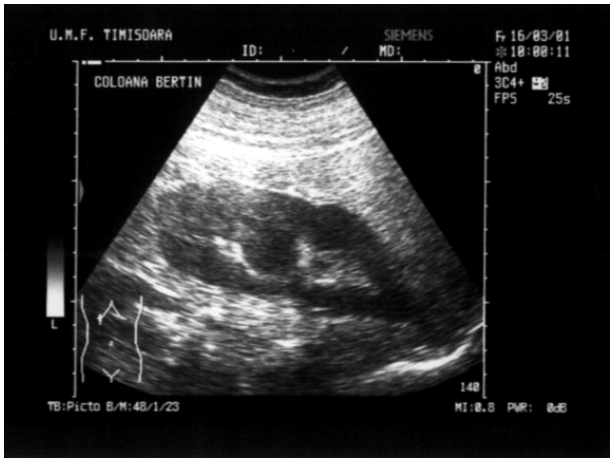


Fig. 5. Bertin column

2. Kidney malformations

a) Congenital single kidney is a rare entity. Diagnosis is made based on lack of visualiasation of one of the kidneys during ultrasound examination. Usually the single kidney is larger (over 12 cm long axis), but morphologically normal. Confirming the absence of a kidney will be done by intravenous urography and / or abdominal CT, since a small congenital kidney or a pyelonephritic kidney may not be revealed by ultrasound, since they have similar echogenicity to the surrounding tissues. The presence of even a small amount of secretion will identify a small kidney during urography. Also CT urography may reveal possible ectopic kidneys, whose existence may be suspected by the visualization during standard ultrasound of a kidney-like structure in another place than the renal lodge, most commonly the pelvis.

b) Unilateral small kidney may be congenital or secondary. Ultrasound may appreciate exactly the dimensions of the kidneys; a difference greater than 2 cm between the two kidneys is considered pathological. Secondary small kidney may be the consequence of a unilateral chronic pyelonephritis, a renal artery stenosis, etc. Inequal kidneys may be the cause of secondary hypertension, hence the need for careful measurement of renal long axis to reveal any inequalities.

c) Pielo-caliceal duplication is a frequent situation, ultrasound diagnosis is made based on the presence of two completely separate central echoic areas in the kidney, which must be confirmed by examination in several sections. Duplicate ureters cannot be assessed by ultrasound. The exam of choice for the diagnosis of pielo-caliceal duplication +/- ureteral duplication is urography.

d) Horseshoe kidney is a rare situation in which ultrasound can suggest the diagnosis by viewing of an altered renal axis, by lack of delimitation of the lower of upper renal pole and especially by viewing the isthmus connecting the two kidneys (Fig. 6). Experience of the ultrasnographist followed in unclear cases by urography and CT helps the diagnosis.

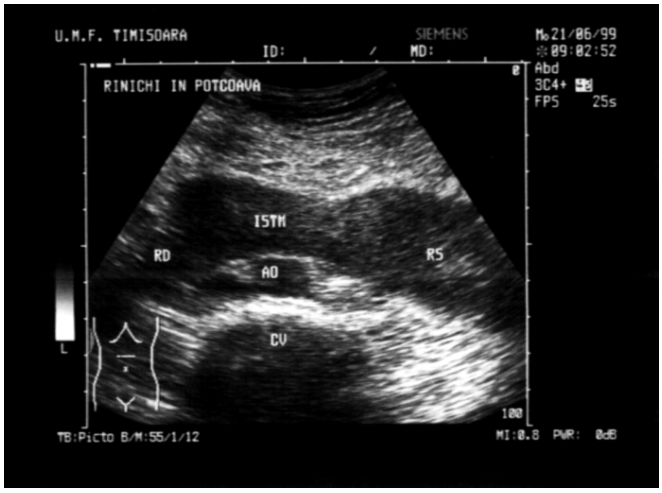


Fig. 6. Horseshoe kidney – the isthmus connecting the two kidneys

3. Renal cysts

a) Simple renal cysts are entities with an unknown etiology and a relatively high frequency that increases with age. It occurs as a serous collection originating in the renal cortex. Renal cysts can be single or multiple (very rarely, more than 5 cysts in a kidney) and have variable sizes (between 1 and 10 cm). Cysts are mostly unilocular, but sometimes may have inner septa. The condition is benign, does not require medical or surgical treatment, only, possibly ultrasound surveillance (annual or biennial), and does not explain any back pain, whose most frequent cause is rheumatic. On ultrasound, they appear as transonic images, with fine walls (Fig. 7, Fig. 8, Fig. 9, Fig. 10), with variable locations in the kidney (they can be located in the cortex or around the pyelum - parapyelic). Very rarely, cysts may cause obstructive phenomena (hydronephrosis). Intracystic hemorrhage is rarely possible, and the cyst will change from transonic to partially or completely hypoechoic.

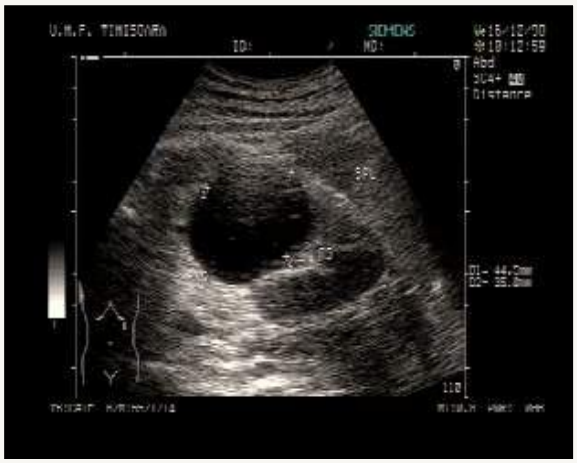


Fig. 7. Parapyelic renal cyst

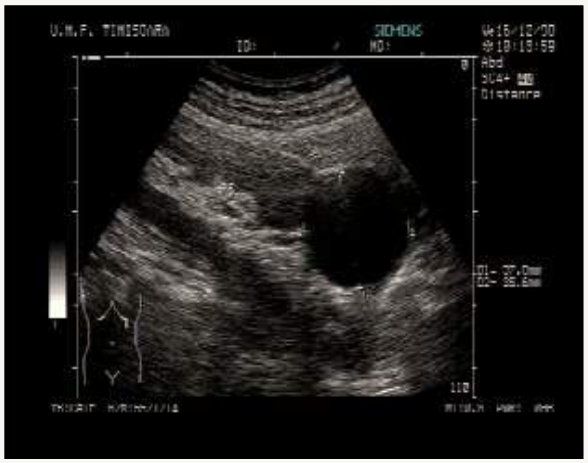


Fig. 8. Cortical renal cyst

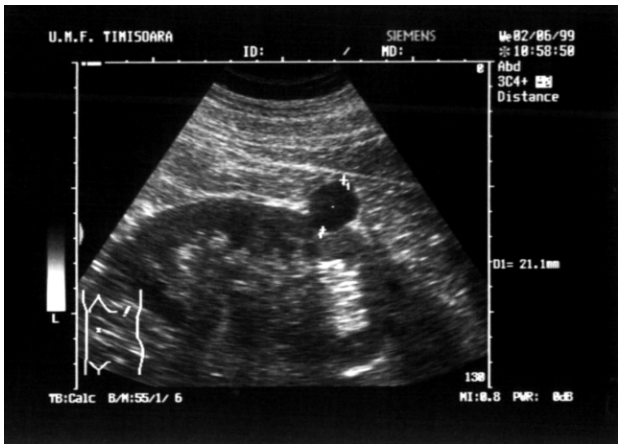


Fig. 9. Cortical renal cyst

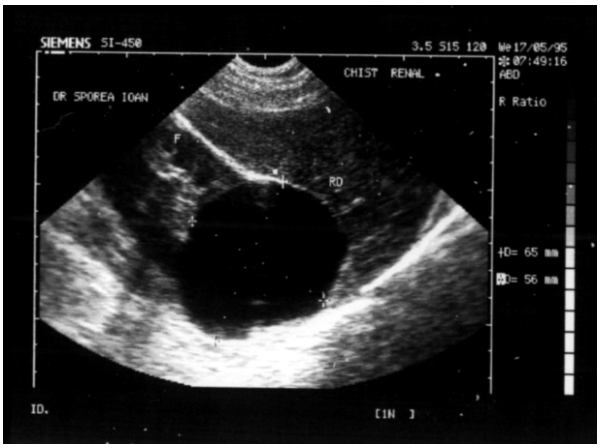


Fig. 10. Upper pole renal cyst

The ultrasound differential diagnosis of simple renal cyst is made with renal hydatid cyst (thick wall, inner septa), necrotizing renal tumors, renal polycystosis, the oligocystic form, hydronephrosis or hydropelvis. For unclear cases (complex cysts, according to the Bosniak classification), CEUS can clarify diagnosis, because tumors will enhance following ultrasound contrast agent, while cysts, even complex, will not capture SonoVue.

b) Polycystic kidney is a disease with genetic autosomal inheritance, characterized by the presence of multiple bilateral renal cysts that significantly enlarge the kidneys (the diagnosis is frequently made starting from an abdominal palpable mass) and thus generate an unclear ultrasonographic demarcation of the organ. Renal polycystosis always affects both kidneys, and is sometimes associated with hepatic polycystosis (hepato-renal polycystosis), and much more rarely, with pancreatic or splenic polycystosis. The prognosis is given by the kidney involvement, since the disease evolves into chronic renal failure, requiring hemodialysis. If polycystic kidney disease is diagnosed in a person, his or hers offspring should be examined by ultrasound in order to search for the disease. It is considered that if until the age of 20 no renal cysts have developed in an offspring, he or she has not inherited the disease.

The ultrasound appearance of polycystic kidneys is typical: bilateral involvement, large, poorly circumscribed kidneys, with the presence of tens of renal cysts of variable sizes (in general 1-8 cm). The ultrasound appearance suggests grape clusters (Fig. 11, Fig. 12). The pyelum is not visible, the whole kidney is changed into a cystic mass. Polycystic kidneys are quite frequently complicated by renal lithiasis (sometimes difficult to diagnose by ultrasound), intracystic hemorrhage (one or several cysts change from transonic to hypoechoic) (Fig. 13), or renal abscess (an appearance similar to that of intracystic hemorrhage, but in a patient with fever, with a septic state).

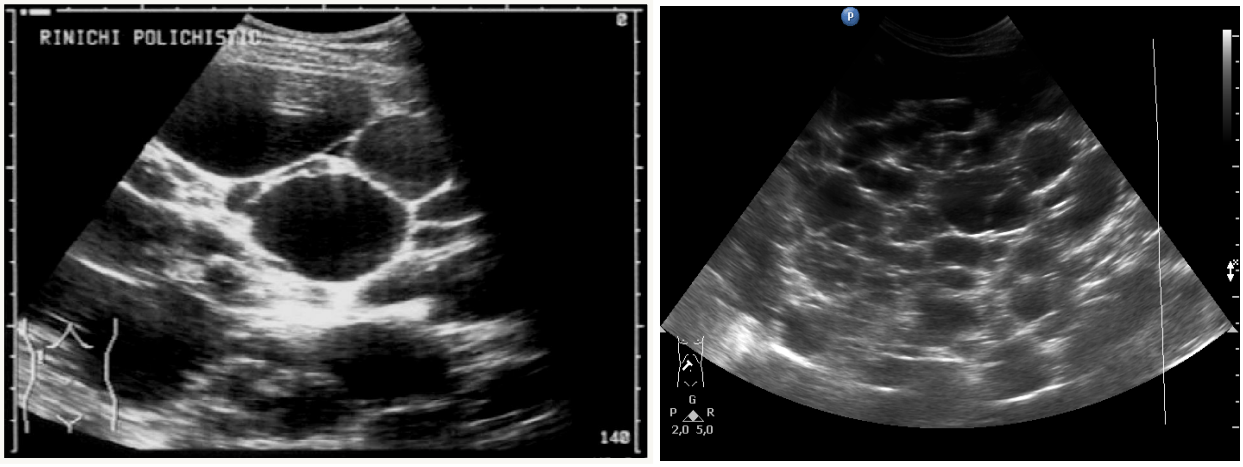


Fig. 11, Fig. 12. Polycystic kidney

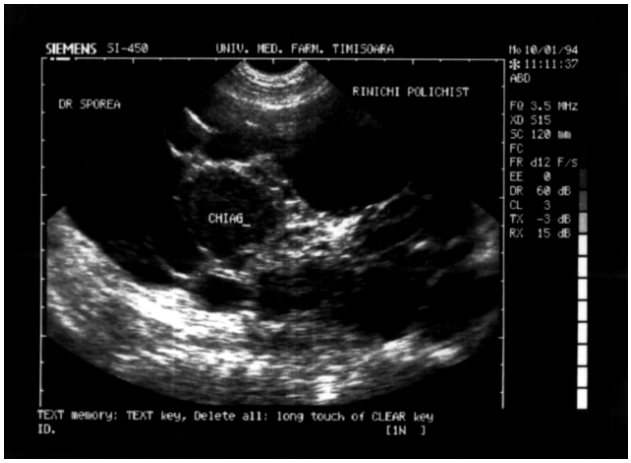


Fig. 13. Polycystic kidney with intracystic hemorrhage

The ultrasound differential diagnosis does not pose particular problems, because the imaging aspect is typical. Differential diagnosis can be made with multiple simple renal cysts (usually maximum 5-10 in a kidney), hydronephrosis, or renal hydatid cyst with daughter vesicles (single cyst image with inner septa). Very rarely these renal disorders are bilateral, as is renal polycystosis.

c) Medullar cystic kidney disease is characterized by the presence of multiple cystic dilations of the collecting ducts from the medullar area. The cause of the disease is not known, it develops in adults, usually bilaterally. In general, it is an incidental finding. Renal function is usually normal. The ultrasound appearance is of multiple anechoic areas (usually < 1cm) situated in the pyelum.

4. Renal lithiasis (kidney stones)

Definition: formation of concretions in the collecting renal system. It is a frequent condition. Renal calculi can be formed by: calcium oxalate, calcium phosphate, ammonium-magnesium phosphate, uric acid or cysteine. The formation of calculi is influenced by the family or personal predisposition, urinary salt concentration, change of urinary pH, presence of urinary infections, urinary tract anomalies.

The clinical picture of renal lithiasis is typical renal colic (intense colicky pain in the lumbar region radiating to the pelvis, with the presence of pollakiuria, dysuria), hematuria, recurrent urinary infections. In some cases, renal lithiasis can be completely asymptomatic and is detected incidentally on ultrasound. Very rarely, renal lithiasis can generate anuria (bilateral obstructive renal lithiasis).

The ultrasound appearance of renal lithiasis is of hyperechoic images with a posterior shadow (Fig. 14, Fig. 15). Unlike radiology that will only evidence radiopaque calculi, on ultrasound calculi appear as hyperechoic regardless of their chemical structure, being in fact obstacles that reflect ultrasounds.

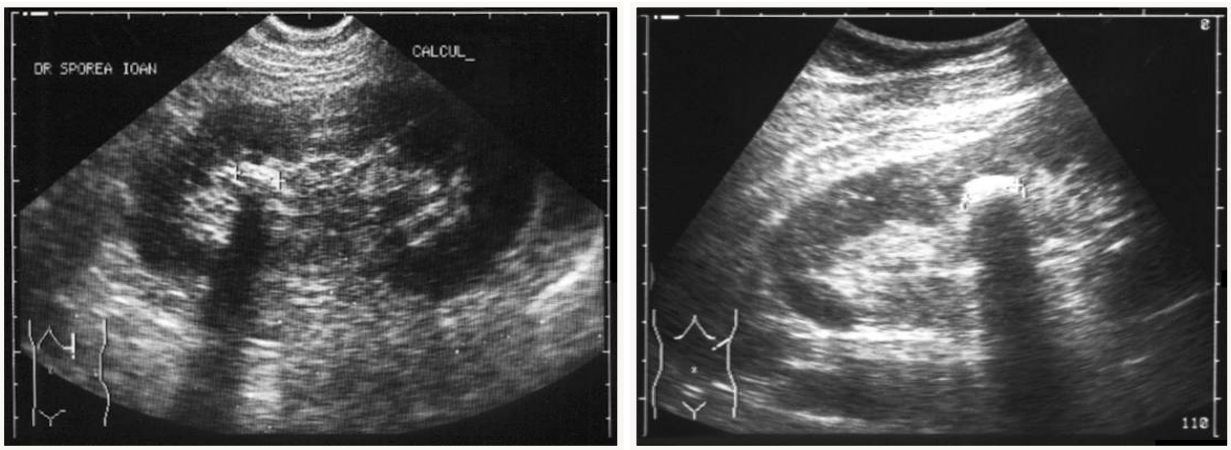


Fig. 14, Fig. 15. Kidney stone: hyperechoic image with posterior shadowing

The diagnosis of renal lithiasis is somewhat more difficult compared to that of cholelithiasis. In the case of gall-bladder stones, there is the benefit of the anechoic bile environment, with a very obvious contrast between the bile and the calculus. In renal lithiasis, ultrasound diagnostic difficulties may appear because the hyperechoic calculus is situated inside the pyelum (also hyperechoic). The posterior shadow behind the calculus is a defining element in the diagnosis of renal calculus, in its absence, this diagnosis of kidney stone cannot be sustained (Fig. 16). The presence of hyperechoic images without a posterior shadow in the kidney raises doubts about the diagnosis of renal lithiasis. Fibrous tissue, collagen or renal calcifications are the most frequent causes of this appearance.

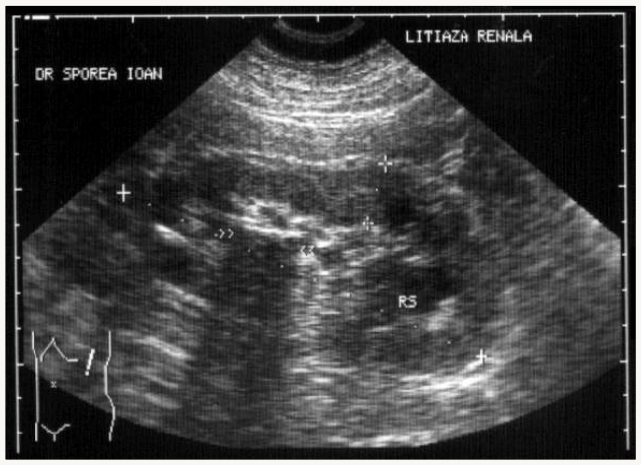


Fig.16. In this kidney stone case, the posterior shadow is much more clearly visible than the hyperechoic image in the pyelum

We wish to clarify an extremely widely circulated entity, ”renal sand”. Starting from a urine rich in urinary salts (which is normal if the patient is not sufficiently hydrated), very many ultrasonographers yield to the temptation to describe ”renal sand” (a non-existing ultrasound entity). If ultrasound is performed for uni- or bilateral lumbar pain and no hyperechoic images with a posterior shadow are detected, the rheumatic etiology of pain is common. The description of false ”stones” or "sand" will lead to inadequate therapy.

The ultrasound detection of a kidney stone (ultrasound image with a posterior shadow) will be followed by the assessment of the stone's size (for therapy: calculi smaller than 5-7 mm can usually be eliminated by natural routes, larger calculi will need to be treated – usually by extracorporeal lithotripsy). It will also be determined if the calculus is in the renal pelvis or in the calyces and whether it is obstructive or not (generating hydronephrosis or not). The ultrasound diagnosis of a coraliform renal calculus is often difficult, even if hyperechoic images with a posterior shadow are easy to visualize, but its coraliform extensions into the calices are difficult to assess (Fig. 17).

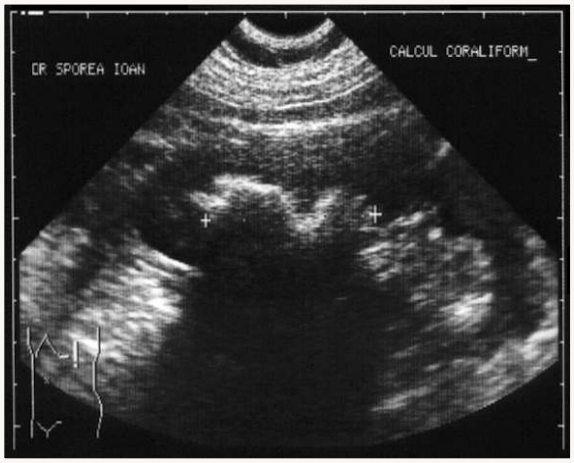


Fig. 17. Coraliform kidney stone

In conclusion, the diagnosis of renal lithiasis (kidney stones) is not always easy. Renal calculi can be visualized as hyperechoic images with a posterior shadow and sizes larger than 2-3 mm (evaluation is performed through multiple longitudinal and transverse sections, which must demonstrate the presence of a hyperechoic image with a posterior shadow in at least two incidences).

5. Hydronephrosis

Definition: dilation of the urinary tracts (calyx, renal pelvis and pyelo-ureteral junction) generated by an obstructive cause. The main causes of hydronephrosis are: renal lithiasis, renal tumors, prostate adenoma, blood clot, obstructive renal cyst. Obstruction can be due to the ureter's compression or invasion by retroperitoneal or genital tumors, colonic cancer, etc.

The ultrasound appearance is typical: a triangular anechoic image (Fig. 18) situated in the renal pelvis. Hydronephrosis can be compared to a ”palm” or a ”goose foot”. Acording to its severity, hydronephrosis can be classified into: mild (dilation of the renal pelvis, with a normal size cortex - Fig. 18); moderate (important dilation of the renal pelvis with narrowing of the cortex - Fig. 19); severe (severe dilation of the renal pelvis, with the presence of a significantly thinned cortex - Fig. 20).

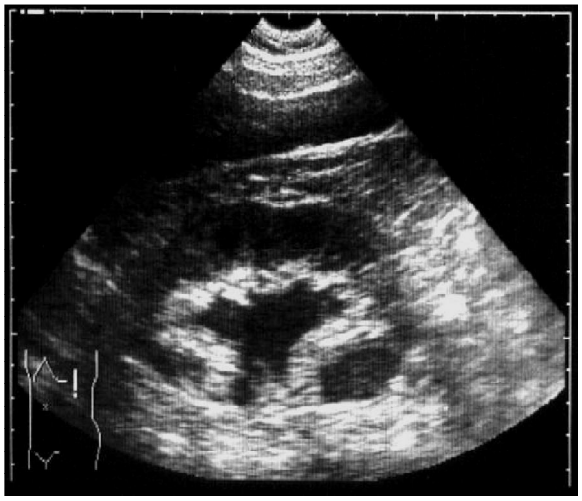


Fig. 18. Mild hydronephrosis

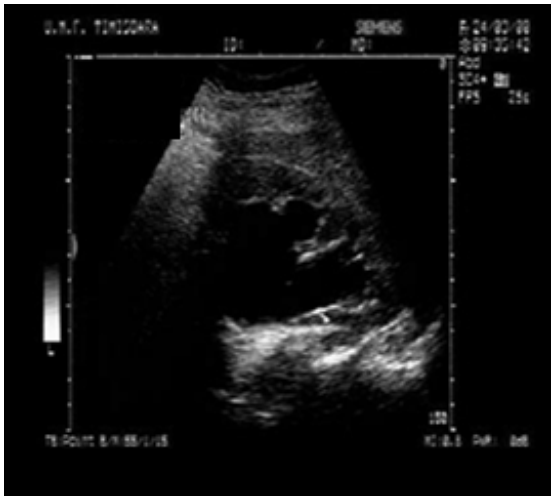


Fig. 19. Moderate hydronephrosis

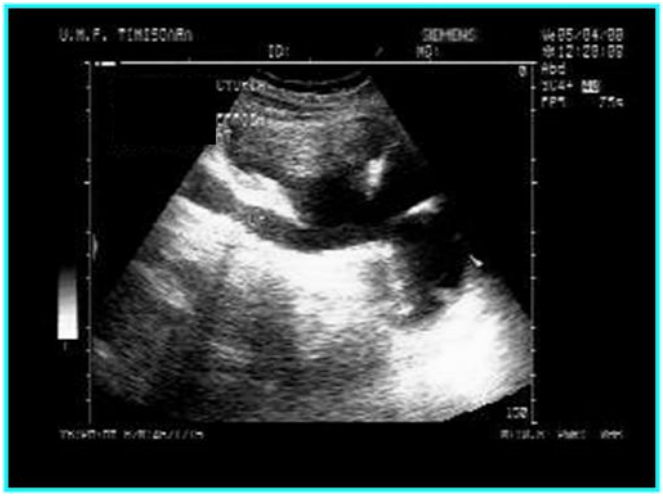


Fig. 20. Severe hydronephrosis with dilated ureter

Subsequently, the cause of hydronephrosis will be searched for. This can be a calculus impacted at the pyelo-ureteral junction (Fig. 21) or a calculus that has migrated to the ureter. The ultrasound diagnosis of ureteral calculus is often difficult. The transducer will be moved along the dilated ureter (visible as a duct with a transonic appearance) until the hyperechoic calculus that blocks the lumen is evidenced (Fig. 22). In the case of bilateral hydronephrosis, a low obstruction can be considered: pelvic tumors, urethral stenosis, obstructive prostate adenoma, etc.

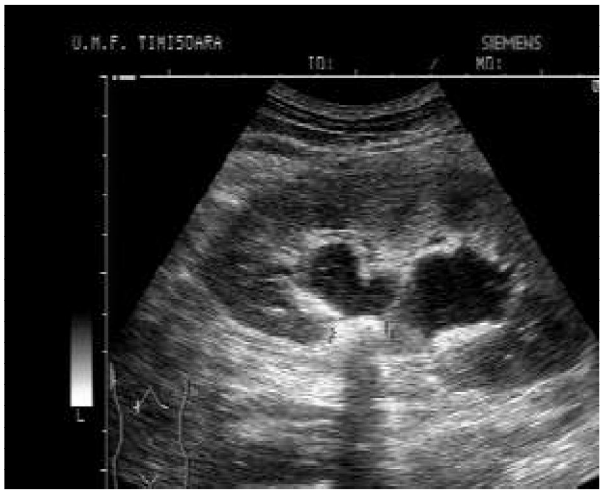


Fig. 21. Pyelo-ureteral junction kidney stone

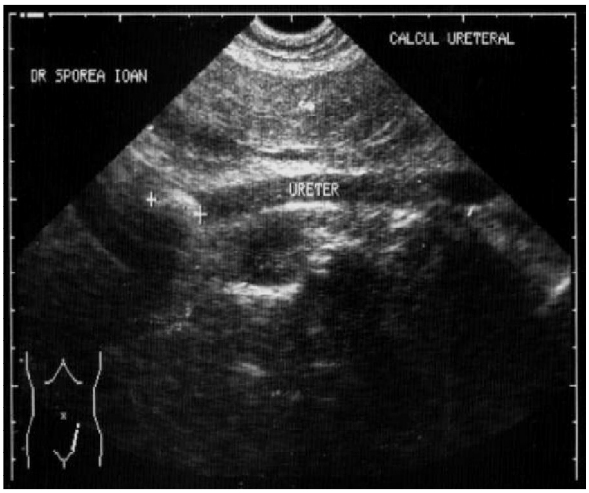


Fig. 22. Ureteral stone

The ultrasound differential diagnosis of hydronephrosis is made with: simple juxtapyelic cysts (the cysts are not contiguous, they are separated by septa); renal vascular ectasia (differentiation is made using power Doppler); renal sinus lipomatosis; papillary necrosis; bladder overloading (the patient drinks too much liquid before examination and is asked not to urinate; dilation is bilateral) - after the patient urinates, bilateral ”hydronephrosis” disappears; urothelial tumors (usually hypoechoic - Fig. 23); acute pyelonephritis (suggestive clinical symptoms, which can occur in a patient with hydronephrosis).

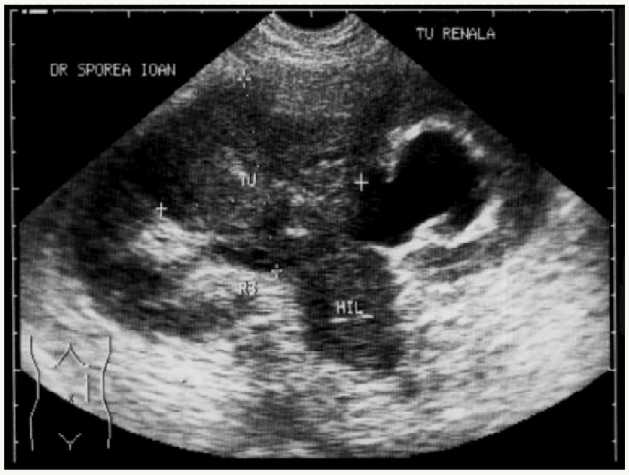


Fig. 23. Urotelium tumor with secondary hydronephrosis

In cases in which the diagnosis of hydronephrosis is not clear, urography can be used (the kidney will usually show no excretion in severe hydronephrosis). Other diagnostic techniques: CT (for tumors, papillary necrosis, renal sinus lipomatosis), power Doppler ultrasound (for renal vascular ectasia).

6. Renal tumors

a) Malignant tumors. Renal carcinoma originates in renal tubular epithelium. It represents 1-3% of visceral cancers, with a male to female ratio of 3:1. It is more frequent in persons aged between 50-70 years.

The clinical picture that leads to the diagnosis of renal cancer can be: capricious hematuria, unilateral lumbar pain and/or palpation of a tumor mass. The tumor has a tendency to vascular invasion (renal vein thrombosis) or lymphatic invasion. Metastases occur in locoregional lymph nodes, lungs, bone, liver. A renal tumor can be an incidental finding, discovered on routine ultrasound.

The ultrasound appearance of a renal tumor is most frequently that of a hypoechoic (Fig. 24), more rarely isoechoic (Fig. 25), or even hyperechoic mass (Fig. 26). The size of the tumor at the time of detection varies from 1-2 cm to giant sizes (10 cm or more). Large tumors are mostly inhomogeneous, due to necrosis and intratumoral hemorrhage.

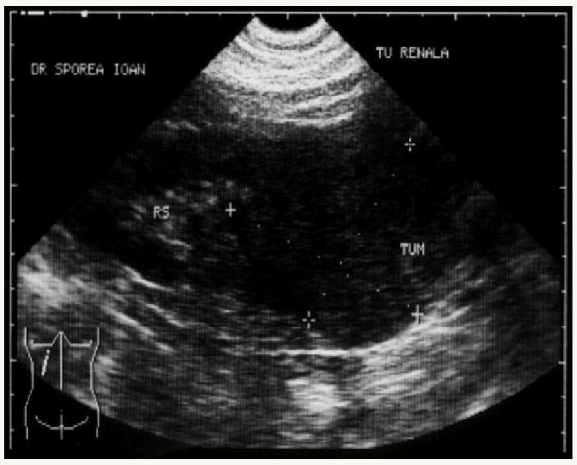


Fig. 24. Hypoechoic renal tumor

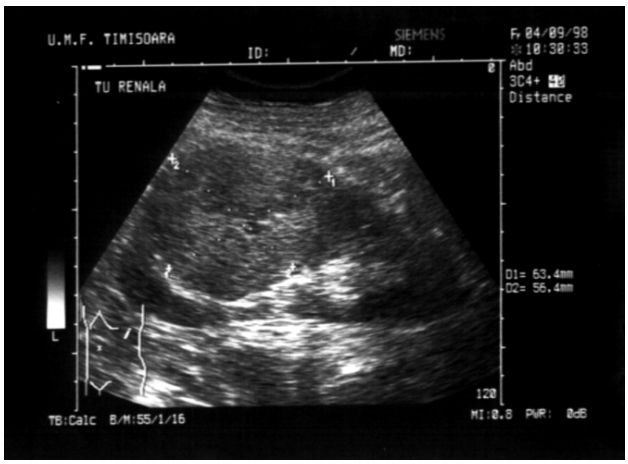


Fig. 25. Isoechoic renal tumor

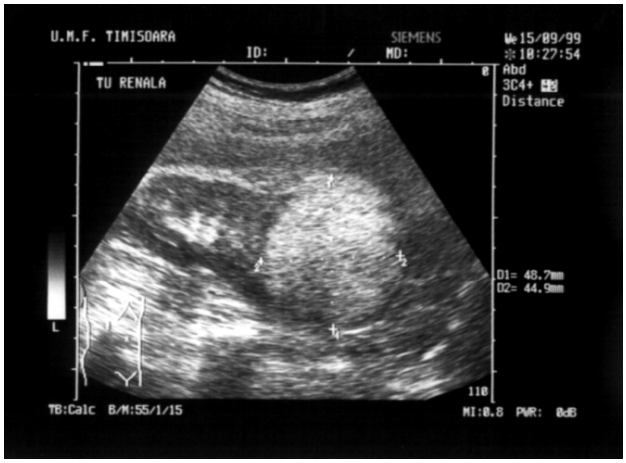


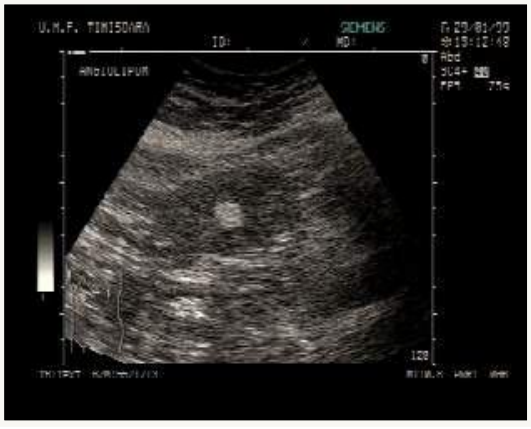
Fig. 26. Hyperechoic renal tumor

The detection of a renal tumor by ultrasound should be followed by the assessment of tumor invasion into the renal vein, into the inferior vena cava, and search for potential hepatic metastases. The diagnosis of renal tumor is confirmed by intravenous urography, CEUS, CT, MRI and possibly ultrasound guided fine needle biopsy.

Other types of malignant renal tumors are: urothelial carcinoma of the renal pelvis, Wilms tumor (pediatric nephroblastoma), renal lymphoma.

The ultrasound differential diagnosis of renal cancer can be made with: renal or perirenal hematoma, hemorrhagic renal cysts, renal metastases, congenital renal bulges, Bertin's column hypertrophy, renal angiolipoma.

b) Benign renal tumors. A particular ultrasound appearance is that of **renal angiolipoma (angiomyolipoma)**. This is a benign renal tumor, composed of fat tissue, smooth muscle fibers and vascular structures. It appears as a well circumscribed hyperechoic mass (fig. 27, fig.28), 1-3 cm in size, situated in the cortex. From an ultrasound point of view, angiolipoma is highly similar to hepatic hemangioma.



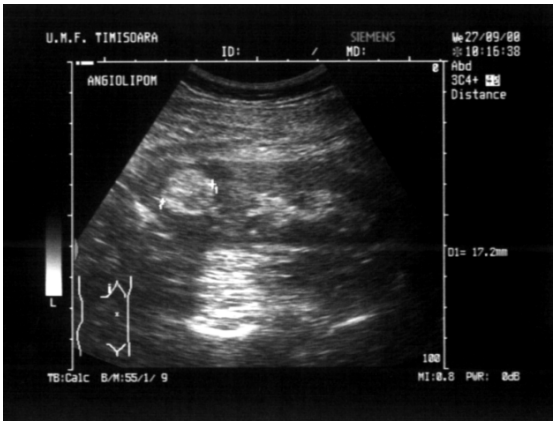


Fig. 27. Fig. 28. Renal angiolipoma

6. Renal hematoma

Whenever we face a patient who suffered an lumbar trauma (following an accident or following a fall) we must consider the possibility of a kidney hematoma, since in these cases there is the possibility of a "two-steps" severe internal bleeding. Clinically, these patients will have suggestive context (abdominal or lumbar trauma), loin pain on palpation, while hematuria is a big warning sign.

Ultrasound is useful for evaluating these patients because it is inexpensive, repetitive and is not irradiating, so that these patients can be followed-up whenever necessary. It must be considered that when there is a suspicion of renal hematoma, but we're not sure we have a good acoustic window and that we had a good visualization of the kidney (uncooperative patient with severe pain when attempting examination), we should always recommend a CT scan with contrast.

The ultrasonographic appearance of a subcapsular renal hematoma is that of a hypoechoic "eyebrow" imediately under the capsule (Fig. 29), while in intraparenchymal hematoma that of an intrarenal hypoechoic area. The integrity of the renal capsule must be examined as well as the presence of a possible haemoperitoneum (which will appear as a transonic area surrounding the kidney).

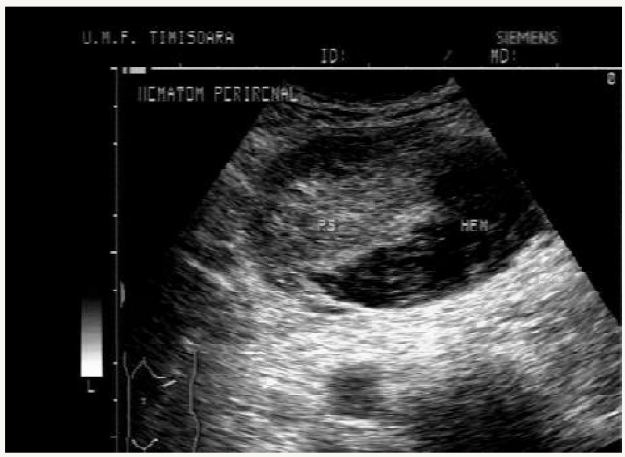
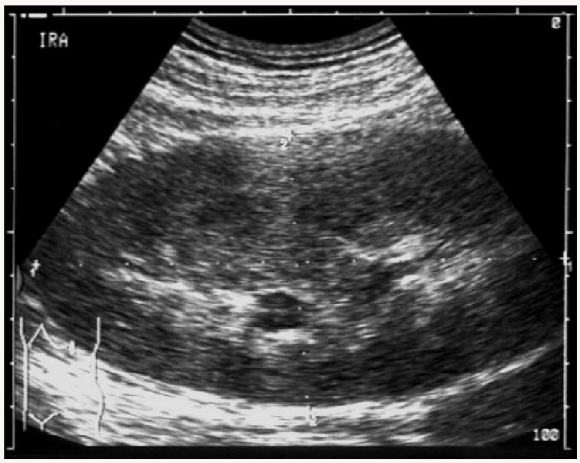


Fig. 29. Subcapsular hematoma - hypoechoic subcapsular "eyebrow"

7. Renal failure

***Definition**:* the incapacity of the kidneys to eliminate toxic metabolites from the blood. Renal failure can be either acute (ARF) or chronic (CRF). In the presence of a biological picture of renal failure (increased urea and creatinine values), ultrasound is a particularly useful method for differentiating ARF from CRF. In acute renal failure, the kidneys are large, while in chronic renal failure, they are most frequently small.

Acute renal failure (ARF) is ultrasonographically characterized by large kidneys (more than 12 cm along the long axis), hypoechoic cortex (Fig. 30, Fig. 31) (due to edema). If renal failure has a postrenal cause, an obstructive appearance with bilateral hydronephrosis or single kidney hydronephrosis (congenital or surgical) can be found.



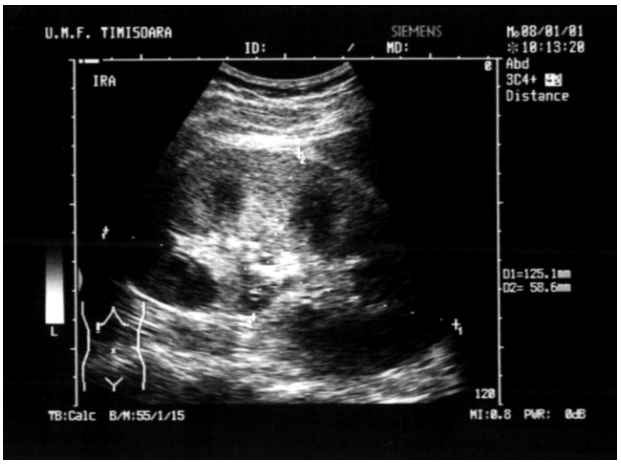


Fig. 30, Fig. 31. Large kidney with thick cortex, in ARF

Chronic renal failure (CRF) is generally characterized by small kidneys (fig. 32) with increased cortex echogenicity and erased differentiation between the cortical area and the basinet (fig. 33). There are situations in which large or normal kidneys are associated to CRF, such as amyloidosis or diabetic nephropathy. In advanced CRF stages, the kidneys are difficult to distinguish from the adjacent structures.

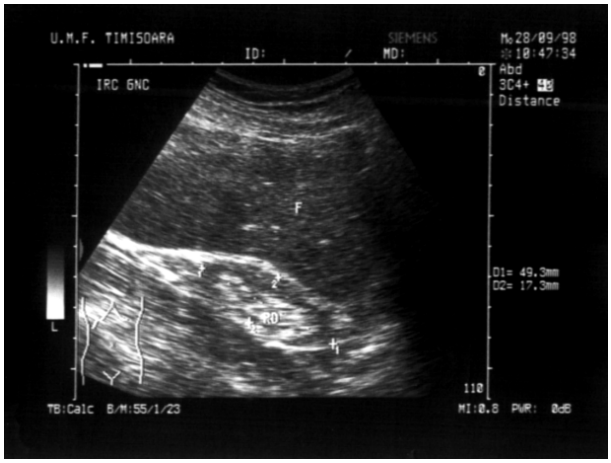


Fig. 32. CRF - small kidney

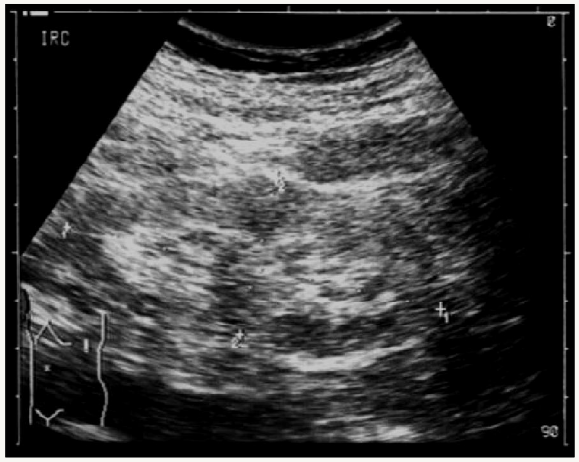


Fig. 33. CRF - erased differentiation between the cortical area and the basinet

THE ADRENAL GLANDS

The adrenal glands are pyramid shaped retroperitoneal organs, situated in the adipose tissue adjacent to the upper renal pole. The right adrenal gland is situated between the right renal pole, the right hepatic lobe, the right diaphragmatic crus and the inferior vena cava. The left adrenal gland lies between the left upper renal pole, the aorta and the left diaphragmatic crus.

Ultrasound visualization of the normal adrenal glands is generally difficult. The right adrenal gland is easier to visualize because the liver plays the role of an ultrasound window. To see the right adrenal gland, the area between the right hepatic lobe and the inferior vena cava, at the level of the upper renal pole, is scanned. The examination of the left adrenal gland is more difficult (except in the presence of splenomegaly). The left adrenal gland will be located between the upper renal pole and the aorta.

The method of choice for the evaluation of the adrenal glands is CT or echoendoscopy (left adrenal gland).

Adrenal tumors can be primary or secondary. They mostly appear as hypoechoic masses (Fig. 34), situated in the adrenal region. Sometimes, the tumor can appear as inhomogeneous, because of tumor degeneration and necrosis (Fig. 35). Tumor sizes are variable (2-6 cm) but sizes up to 10 cm can also be found. It is impossible to differentiate primary vs. metastatic adrenal tumors only by standard US.

The ultrasound differential diagnosis of adrenal tumors is made with other retroperitoneal tumors, periaortic or caval adenopathies, renal tumors, upper pole renal cysts.

For the evaluation of the adrenal glands in case of suspicion of adrenal hyperplasia, CT is recommended, which assesses the gland's size relatively easily. For the suspicion of pheochromocytoma, ultrasound is a good screening method because it can detect even small size tumors. When ultrasound is inconclusive, computed tomography should be performed.

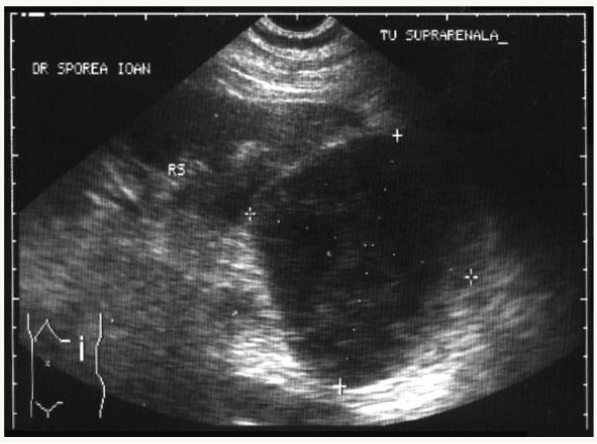


Fig. 34. Hypoechoic left adrenal gland tumor.

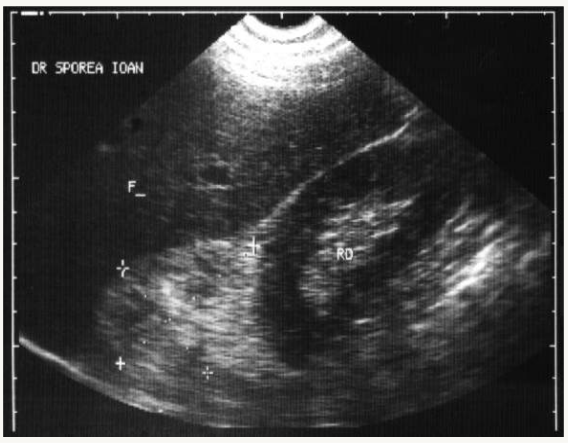


Fig. 35. Inhomogeneous right adrenal gland tumor

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