White Paper

Ultrasound examination of the Liver in dogs and cats

esaote

A practical guide to the ultrasonographic study of the liver in domestic animals with a discussion of echostructural features, pathological aspects and vascular and biliary tract abnormalities.

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Introduction

The anatomy of the liver is quite similar in the dog and cat. It is composed of several lobes, which are barely distinguishable from each other except where peritoneal effusion is present. The lobes are the left lateral and medial, the quadrate, the caudate, and the right lateral and medial. (Fig. 1)

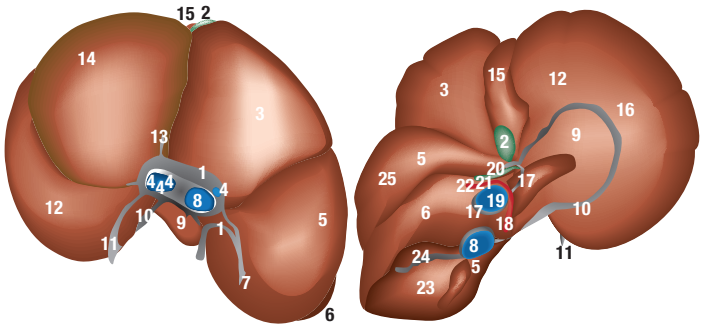

Fig. 1 Liver anatomy of the diaphragmatic surface (left) and visceral surface (right)[2]

1 *Coronary ligament*
2 *Gallbladder*
3 *Right medial lobe*
4 *Hepatic vein*
5 *Right lateral lobe*
6 *Caudate process of caudate lobe*
7 *Right triangular ligament*
8 *Caudal vena cava*
9 *Papillary process of caudate lobe*
10 *Lesser omentum*
11 *Left triangular ligament*
12 *Left lateral lobe*
13 *Falciform ligament*
14 *Left medial lobe*
15 *Quadrate lobe*
16 *Gastric impression*
17 *Hepatic branches*
18 *Hepatic artery*
19 *Portal vein*
20 *Bile duct*
21 *Right gastric artery*
22 *Gastroduodenal artery*
23 *Renal impression*
24 *Hepatorenal ligament*
25 *Duodenal impression*

Ultrasound examination is performed using medium and low frequency probes, although in the cat it is often possible to scan the liver with high frequency linear probes that provide excellent image resolution.

To examine the liver as a whole, transverse and longitudinal scans are performed using a retrosternal approach. However, in large, deep-chested dogs an intercostal approach is often necessary (Fig. 2).

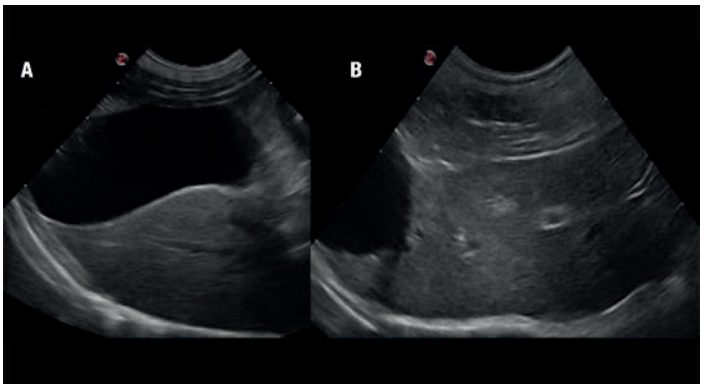

Fig. 2 Normal liver in sagittal (A) and transverse (B) section

Under normal conditions, the liver extends to the right until it comes into contact with the cranial pole of the right kidney and to the left until it comes into contact with the most cranial portion of the spleen. It is more hypoechogenic than both organs. (Fig. 3).

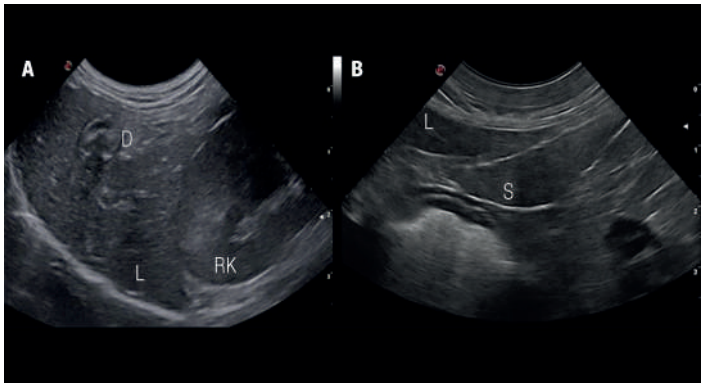

Fig. 3 A-Sagittal section of the right hepatic lobe (L) in contact with the cranial pole of the right kidney (RK), D duodenum; B-Tip of the left hepatic lobe (L) near the spleen (S)

Changes in liver volume

The size of the liver is very difficult to assess objectively. Since ultrasound allows a sectoral view, the last pair of ribs, the costal arch, the relationships with the surrounding organs, and the shape of the tips of the hepatic lobes are usually taken as a reference. An enlarged liver will extend significantly caudally to the last pair of ribs and the costal arch, may be in contact or close to the bladder, and will have rounded edges (Fig. 4). It is also usually possible to draw up a list of several possible differential diagnoses for increased or reduced liver volume, but a definitive diagnosis cannot be formed on the basis of ultrasound examination alone.[1] (Fig. 5)

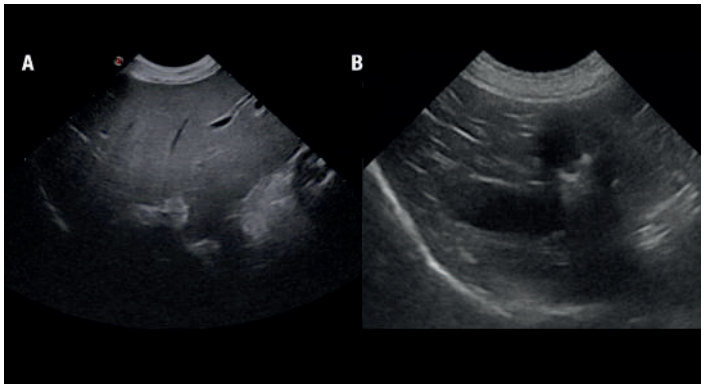

Fig. 4 Examples of increased (A) and reduced (B) liver volume

Diagnostic differentials for alterations in hepatic volume

Diffuse hepatomegaly	Focal or asymmetrical hepatomegaly	Small liver
Steroid hepatopathy	Primary or metastatic neoplasia	Congenital portosystemic shunting
Lipidosis	Abscess	Microvascular dysplasia or primary portal vein hypoplasia
Hepatitis or cholangiohepatitis	Cyst(s)	Cirrhosis
Passive congestion	Granuloma	Fibrosis
Round-cell neoplasia: lymphoma, malignant histiocytosis, and mast cells	Thrombosis	Severe hypovolemia
Massive hepatocellular carcinoma or metastases	Lobar torsion	
Amyloidosis	Hematoma	

Fig. 5 Possible differential diagnoses for the various changes in liver volume[1]

Changes in echogenicity and echostructure

Alterations in the echogenicity and echostructure of the parenchyma can be divided into diffuse and focal depending on how the organ is affected. In both cases, ultrasound is highly sensitive but poorly specific, making a diagnosis impossible.[3–5]

Diffuse liver disease

Diffuse disease may result in a homogeneous increase in echogenicity, a decrease in echogenicity, or evidence of inhomogeneous echogenicity, which is difficult to attribute to the primary cause but allows a list of possible etiologies to be drawn up. (Fig. 6).

Diagnostic differentials for diffuse alterations in hepatic parenchymal echogenicity		
Diffuse hyperechogenicity	**Diffuse hypoechogenicity**	**Mixed echogenicity**
Steroid hepatopathy	Passive congestion	Steroid hepatopathy associated with benign hyperplasia, or other combinations of processes
Lipidosis	Acute hepatitis or cholangiohepatitis	
Other vascular hepatopathies	Lymphoma	
Chronic hepatitis	Histiocytic neoplasm	
Fibrosis	Amyloidosis	
Cirrhosis		
Lymphoma		
Mast cell tumor		

Fig. 6 Possible differential diagnoses for the various alterations in liver echogenicity[1]

Some examples of hyperechogenicity and hypoechogenicity of the liver parenchyma are shown below, diagnosed by comparing them with the echogenicity of the splenic parenchyma and renal cortex (Fig. 7, 8, 9).

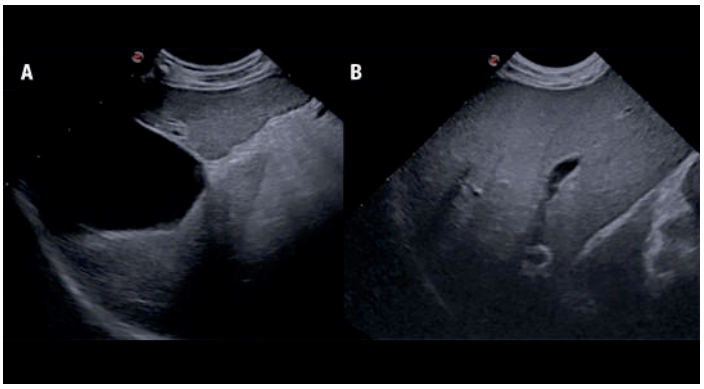

Fig. 7 A-Hyperecogenic liver, with rounded edges due to steroid-induced hepatopathy; B-Hyperecogenic liver following neoplastic infiltration (lymphoma), note the attenuation of distal echoes secondary to the increase in organ consistency

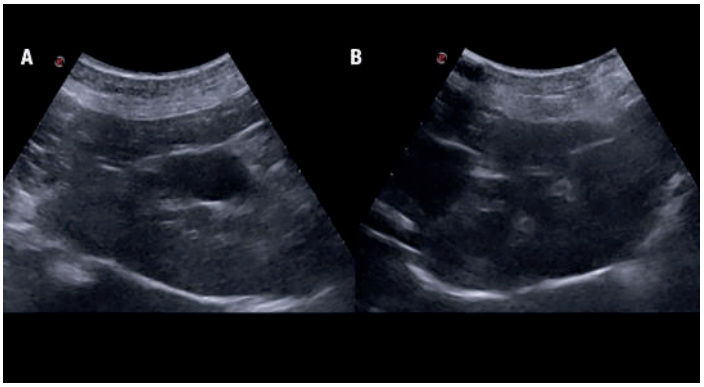

Fig. 8 Hypoechogenic liver due to amyloidosis in sagittal (A) and transverse (B) scan

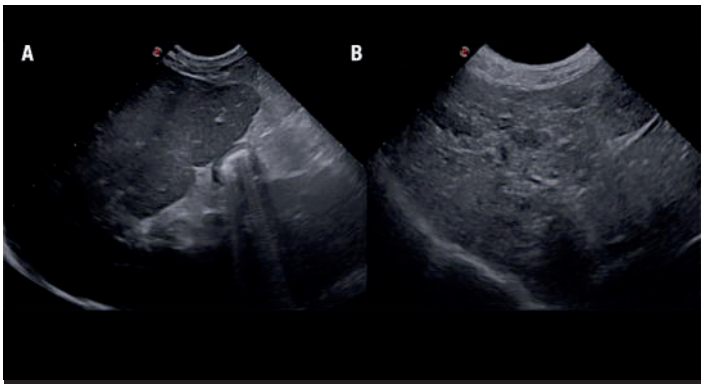

Fig. 9 A-Hypoechogenic liver with increased volume in a patient with hepatitis; B-Liver with parenchyma showing inhomogeneous echogenicity due to degenerative liver disease associated with nodular hyperplasia

Given the poor reliability of ultrasound examination in distinguishing between different liver diseases, it is always advisable to take all the ultrasound signs into consideration, to examine all the abdominal structures as a whole and to assess the ultrasound findings in relation to the hematochemical tests and, whenever possible, to take samples.[6]

The first test to consider is undoubtedly cytology, which is minimally invasive and can be carried out with a non-sedated patient. Surgical preparation of the field and the use of 20-22G needles is recommended. The risks are generally low in non-thrombocytopenic patients and bleeding is the most common complication, also in view of the fact that non-normal tissues can be more friable and that hepatopathic patients may have coagulation deficits. Cytology exam results are generally diagnostic in cases of lipidosis, vacuolar hepatopathy, round cell tumors, and suppurative hepatitis.[1,7,8]

All other diagnoses require a biopsy, which can be performed ultrasound-guided with the patient under general anesthesia and after performing a coagulation test. Semi-automatic 14-18G Tru-cut needles are normally used to perform this procedure following surgical preparation of the patient.[1]

The procedure is strongly discouraged in cats because of the shock, possibly lethal, caused by the cutting needle.[9]

Focal hepatic lesions

Ultrasonography is a highly sensitive examination for the diagnosis of focal hepatic lesions, which can differ in appearance and size but unfortunately, as for diffuse parenchymal changes, it is poorly specific in characterizing them.[1,5]

For this reason, it is only possible to produce a list of potential differential diagnoses that can be linked to the characteristics of the lesion examined, as explained in Figure 10.

Diagnostic differentials for local hepatic lesions with ultrasonography			
Anechoic	**Hypoechoic**	**Hyperechoic**	**Mixed echogenicity**
Cyst	Nodular hyperplasia	Nodular hyperplasia	Nodular hyperplasia
Cystic tumor	Metastasis	Primary neoplasia	Primary neoplasia
Necrosis	Lymphoma	Metastasis	Metastasis
Abscess	Primary hepatic neoplasia	Mineralization or cholelithiasis	Abscess
Hematoma	Abscess	Abscess	Hematoma
	Necrosis	Fat or myelolipoma	
	Hematoma	Granuloma	
	Complex cyst	Gas	
		Metallic clip	

Fig. 10 Differential diagnoses of focal hepatic lesions1

However, there are some criteria associated with the possible malignancy of the lesion examined: its size is greater than 3 cm, the presence of peritoneal effusion, the target-like aspect, concomitant changes in other organs and the lymph nodes.[5,10]

Examples of focal liver lesions diagnosed by cytological or histological examination are provided below (Fig. 11 and 12).

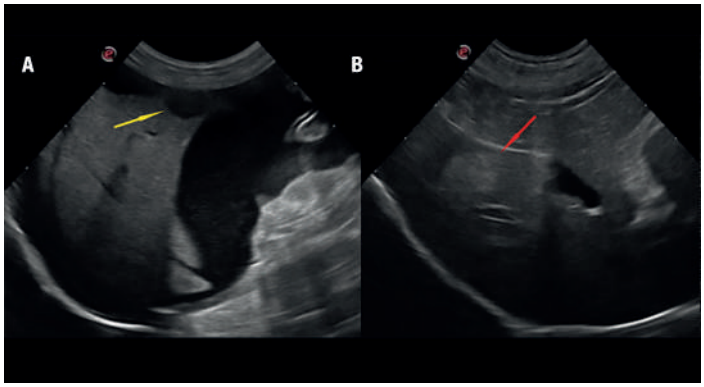

Fig. 11 A-Hypoechogenic focal lesion (yellow arrow); B-Hyperechogenic focal lesion (red arrow)

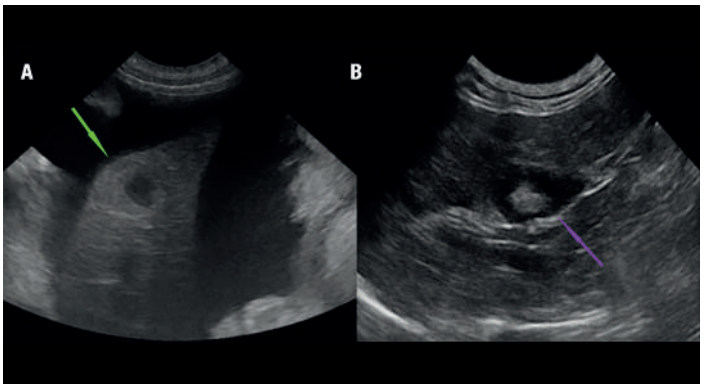

Fig. 12 A-Inhomogeneous focal lesion (green arrow) diagnosed as metastasis of splenic hemangiosarcoma; B-Target-like focal lesion (purple arrow) diagnosed as metastasis of carcinoma

For more specific characterization of focal hepatic lesions, in addition to cytological and histological sampling, contrast-enhanced ultrasound (CEUS) examinations can be performed, which, in human medicine, have a sensitivity comparable to contrast-enhanced CT scans and a higher specificity than cytology.[11]

Ultrasound contrast agents are microbubbles of gas encapsulated in a phospholipid membrane that are injected intravenously and remain almost completely within the vascular compartment, reaching and thus allowing the visualization of even the smallest capillaries. Their gaseous component is eliminated through respiration, while the capsule is eliminated through the biliary tract. Side effects are rare and mild, with vomiting and collapse occurring in 1% of canine and 0% of feline patients.

They require dedicated technologies and probes in order to be visualized. They can be administered without the need for sedation, generally in an amount ranging from 0.1 to 1 ml per patient, followed by a small volume of saline solution, inside a peripheral venous catheter.[1]

They are used primarily in the examination of the liver, for the detection and characterization of focal lesions, mainly due to the distinctive vascularization of this organ.

Malignant focal lesions have a faster wash-in and wash-out than the surrounding parenchyma because they draw their blood support primarily from the hepatic artery, and are therefore easily highlighted and characterized by the contrast agent. (Fig. 13 and 14).[11]

	Type	Arterial phase (10-20 sec)	Portal phase (30-45 sec)	Late phase (>120 sec)
Benign	Hemangioma	Globular enhancement	Centripetal filling	iso-/hyper-enhancement
	Focal nodular hyperplasia	Hyper-enhancement spoke wheel appearance centrifugal filling	Moderately hyper-/iso-enhancement	iso-enhancement scar (40%)
	Hepatocellular adenoma	Hyper-enhancement	Transition	iso-enhancement
Malignant	Hepatocellular carcinoma	Hyper-enhancement	Iso-/slightly hypo-enhancement	Hypo-enhancement
	Metastasis, hypervascular	Hyper-enhancement with/without central necrosis	Iso-/slightly hypo-enhancement	Strong hypo-enhancement
	Metastasis, hypovascular	No enhancement or peripheral rim	Iso-/slightly hypo-enhancement	Strong hypo-enhancement

Time ranges in the parentheses are the delayed time for each imaging after contrast agent injection

Fig. 13 Phases of acquisition of ultrasound contrast medium by the various focal lesions

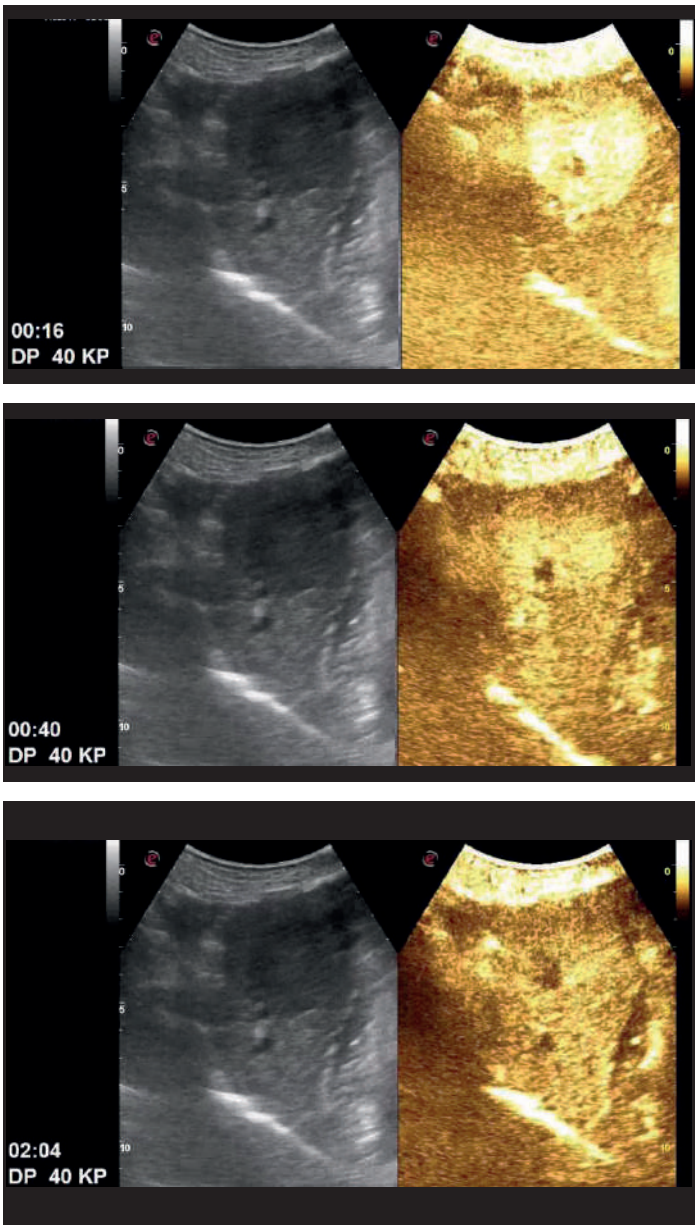

Fig. 14 Hepatic focal lesion larger than 3 cm, hypercaptant in early phase, with transition phase followed by isoechogenic captation with respect to the remaining parenchyma in delayed phase, diagnosed as adenoma.

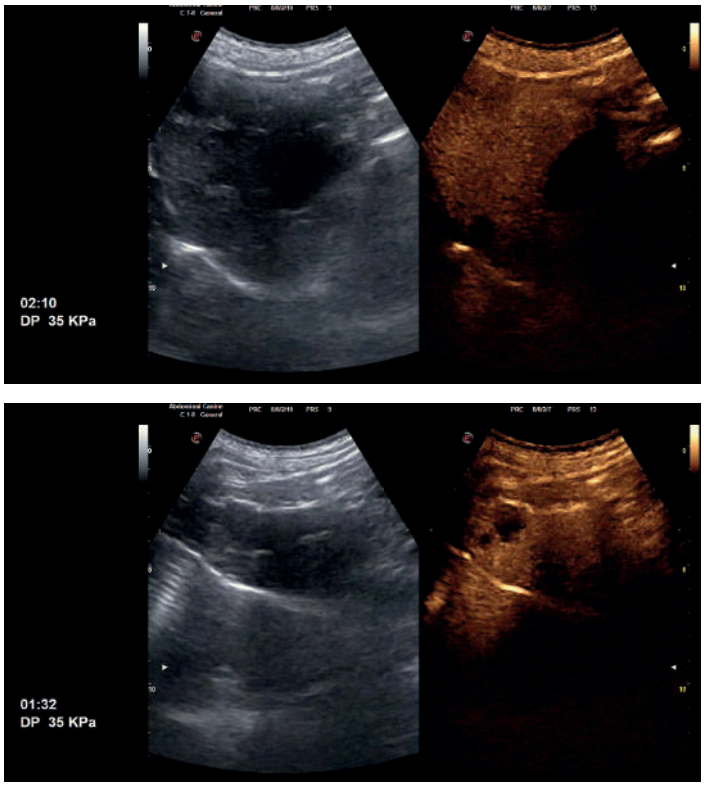

Fig. 14 Multiple focal hepatic lesions (arrows), inconspicuous or not evident in B-mode, hypocaptant with respect to the hepatic parenchyma in delayed phase and diagnosed as metastases of splenic hemangiosarcoma, G: gallbladder

Biliary tract diseases

The biliary system is relatively similar in the dog and cat, in which the gallbladder may appear bilobed. Under normal conditions the intrahepatic bile ducts are not visualized, nor is the common bile duct, except for its outlet at the level of the duodenal papilla.

The chief indications for ultrasound examination of the biliary tract are the search for sludge, stones, mucoceles, obstructions, inflammatory processes, and neoplasms.

Biliary sludge (Fig. 15) is a common finding in dogs, where it is found in a significant number of asymptomatic patients. In cats it may be associated with increased liver enzymes and bilirubin.[1]

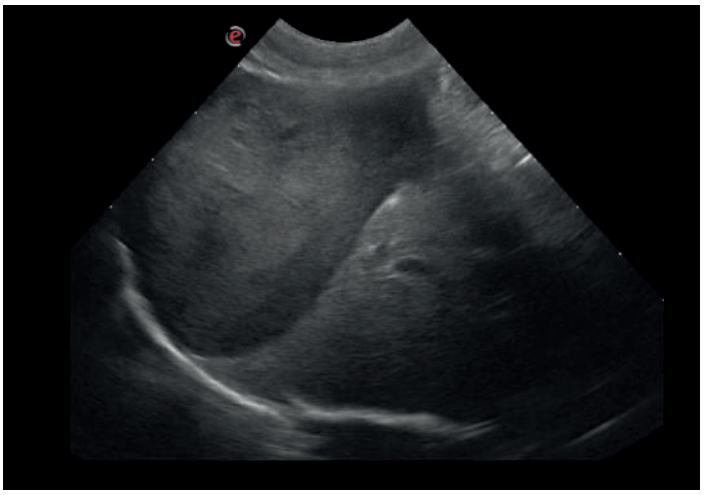

Fig. 15 Gallbladder of a canine patient filled with biliary sludge

Gallstones are generally an occasional finding, but may be associated with inflammation of the biliary system in feline patients. They are visualized as neoformations with a hyperechogenic and reflective surface and a posterior acoustic shadow. (Fig. 16)

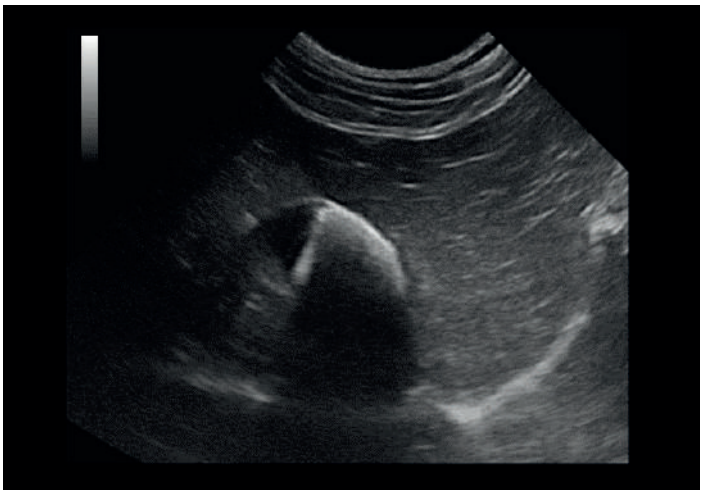

Fig. 16 Cholecystolithiasis

Biliary mucocele is a typical disease of small to medium-sized dogs in old age. It is the progressive accumulation of mucus associated with hyperplasia of the mucus-secreting epithelium (Fig. 17), which results in overdistension of the organ, with possible subsequent parietal necrosis. Blockages of mucus may detach, enter the common bile duct and cause obstruction. For these reasons, it is essential to assess the possible presence of biliary mucocele complications, such as rupture and subsequent biliary peritonitis or occlusion of the common bile duct.[1]

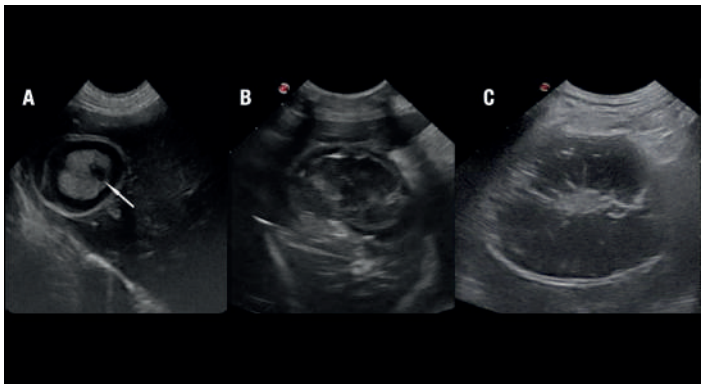

Fig. 17 Mucocele in different stages, note the detached mucus block (arrow) and the radiated aspect of the mature mucocele (C)

Obstruction of the biliary tract can also be caused by the presence of neoplasms, for example in the duodenal papilla, as can be seen in Fig. 18. The duodenal papilla appears severely thickened, hyperechogenic, with obvious presence of blood vessels on Doppler examination.

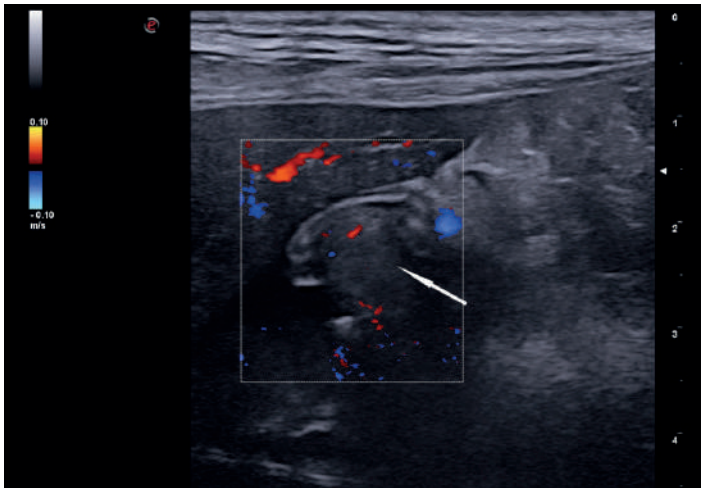

Fig. 18 Neoplastic duodenal papilla (arrow)

Inflammatory processes affecting the biliary tract usually cause thickening of the gallbladder wall (>1 mm) which, in acute cases, presents a double-track aspect or increased echogenicity (Fig. 19) Since it may be caused by an ascending infection from the gastrointestinal tract, cholecystocentesis has both diagnostic and therapeutic value in cases of overdistension of the organ. It is important to perform the cholecystocentesis passing through a portion of liver parenchyma to limit the damage caused by biliary extravasation and using 22G needles.[1]

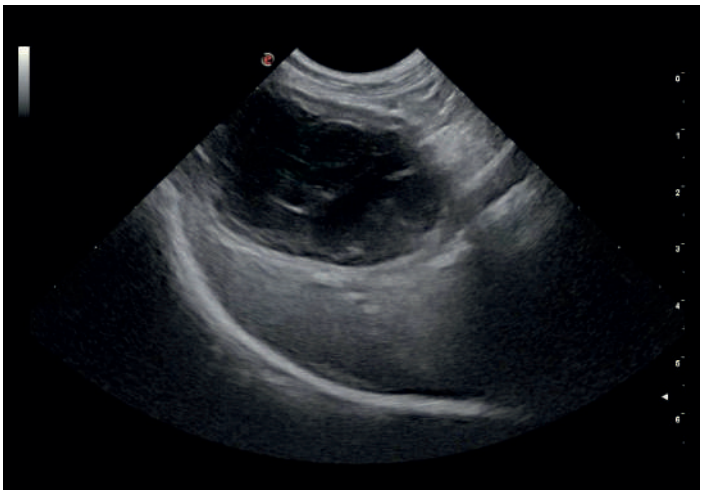

Fig. 19 Gallbladder with thickened and hyperechogenic wall

Abnormalities of the vascular system

The afferent hepatic vasculature gets 70% of its blood supply from the portal vein and the remaining 30% from the hepatic artery. The efferent system, on the other hand, carries waste blood through the hepatic veins to the caudal vena cava.

Within the liver parenchyma, the portal vessels can be distinguished from the hepatic vessels by their hyperechogenic walls. (Fig. 20)

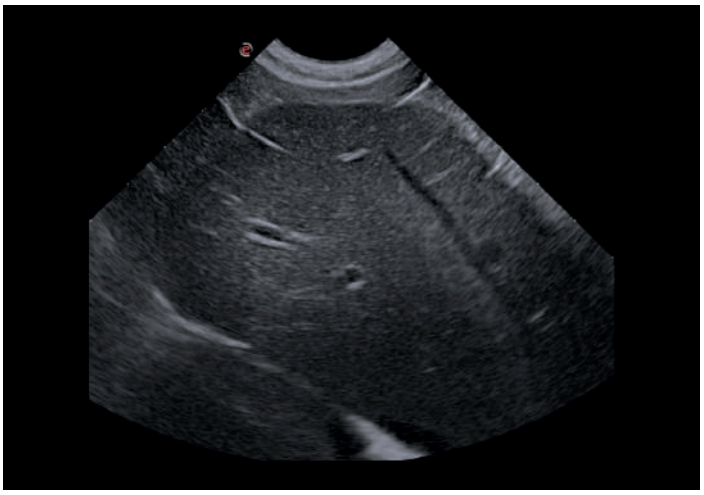

Fig. 20 Hepatic and portal vessels

The portal vein can be seen in the central abdominal portion, ventrally and laterally on the right side of the aorta. It has a relatively constant hepatic flow, with fluctuations due to respiration. A ratio of 0.71-1.25 between the transverse diameter of the portal vein and the aorta is generally considered normal. At the level of the porta hepatis, the portal flow velocity is measured using a sample volume of approximately

half the diameter of the vessel and a correction angle of no more than 60° (Fig. 21). The normal ranges of portal flow velocity are 15+/–3 to 18+/–8 cm/s in the dog and 10 to 18 cm/s in the cat. These ranges are quite wide because the flow velocity can easily be estimated inaccurately.[1]

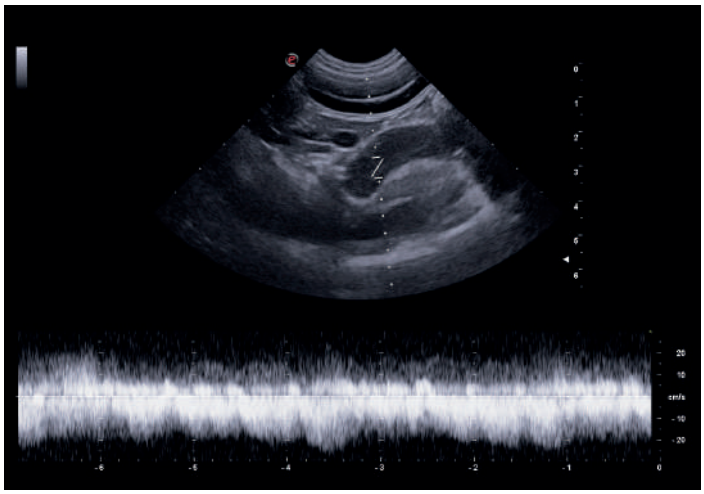

Fig. 21 Normal portal flow

Portal-systemic shunts (PSS) are abnormal vascular communications and are divided into congenital and acquired. The former are newly formed vessels that connect the portal system to the systemic circulation, via the caudal vena cava or azygos vein and are most often single. They are divided into extrahepatic and intrahepatic. Congenital extrahepatic PSS are more typical of small dog breeds and cats. They usually originate from the portal vein or one of its tributaries and flow into the caudal vena cava and are typically large vessels distinguished by hepatofugal flow. Congenital intrahepatic PSS are more typical of large dog breeds. They generally originate from a hepatic vein and flow into the caudal vena cava, and are classified as left-divisional, right-divisional, and central-divisional.

Acquired PSS, on the other hand, are pre-existing collateral vessels that connect the portal system to the systemic circulation and open up due to portal hypertension. Portal hypertension may result from chronic liver disease that reduces parenchyma compliance, reduced portal vein compliance, portal hypoplasia or portal thrombosis, the presence of an arterio-portal fistula or extraluminal compression of the portal vein. They appear as multiple tortuous vessels visible along the splenic profile, near the left renal vein, in the mesentery or around the caudal vena cava.[1]

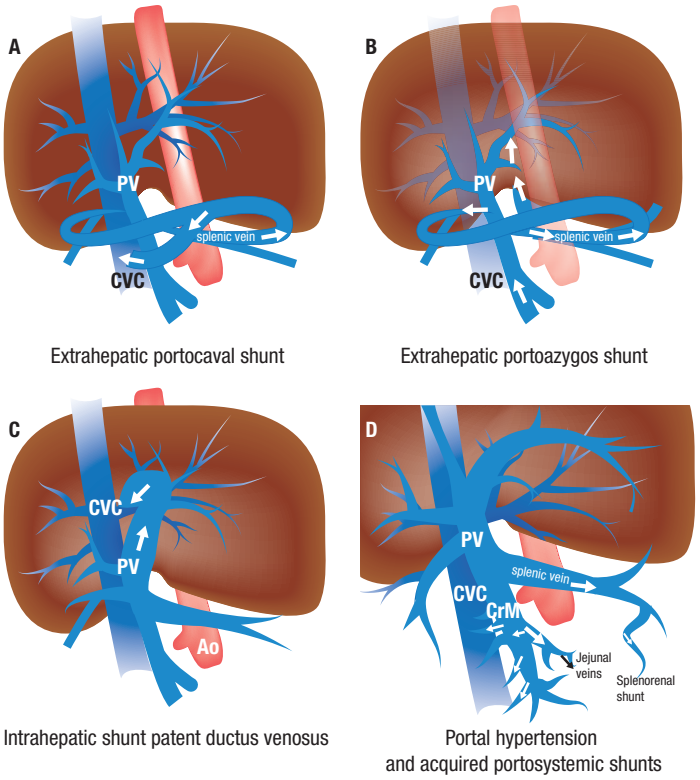

Fig. 22 Congenital extrahepatic shunts (A and B), congenital intrahepatic left-divisional shunt (C), multiple acquired shunts (D)[1]

Ultrasound-guided search for porto-systemic shunts has severe limitations and therefore it is essential to be systematic. This is why it is schematized in 10 steps.[13]

Ascites and edema

The presence of portal hypertension is suspected in the presence of hepatofugal flow or when the flow velocity is less than 10 cm/s and this may result in peritoneal effusion and/or edema of the gastric wall, gallbladder, and pancreas. (Fig. 23)

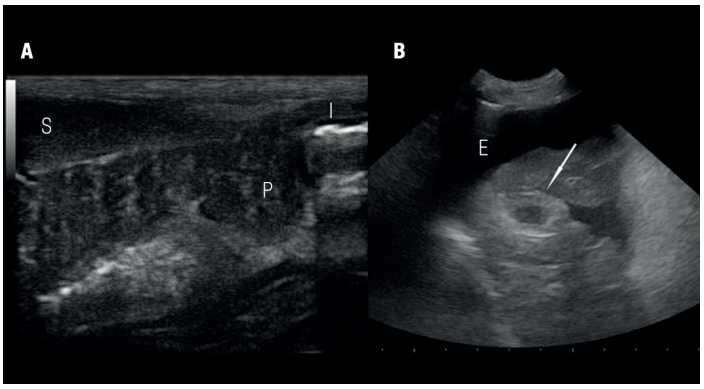

Fig. 23 A-ventrally to the spleen (S) and to a digiunal loop (I) we can observe the pancreas (P) hypoechogenic, with increased volume and a striated appearance; B-Edema of the wall of the gallbladder (arrow) - note the irregular profile of the liver and the presence of ascites (E)

Liver size

In the presence of PSS, liver volume appears reduced but with echogenicity and echostructure preserved. This contrasts with liver volume reductions secondary to fibrosis/cirrhosis, in which the liver also appears hyperechogenic/heterogeneous, irregular, or nodular. (Fig. 24)

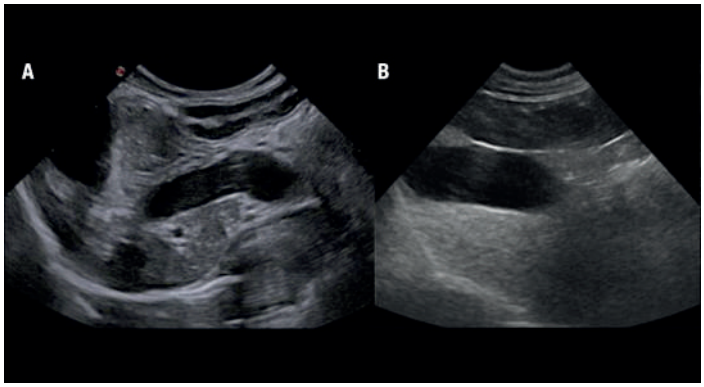

Fig. 24 Liver with reduced volume due to PSS (A) and hyperechogenic liver with reduced volume due to fibrosis (B)

Portal vessels and aberrant vessels

Intrahepatic portal vessels generally appear reduced in caliber in cases of PSS and portal vein hypoplasia, while in some cases the aberrant vessel can be seen within the liver parenchyma. (Fig. 25)

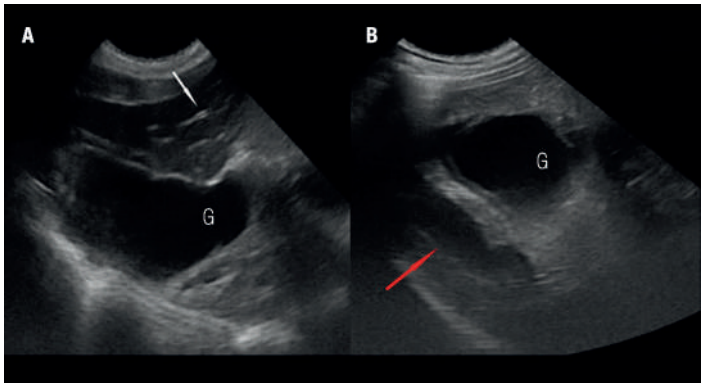

Fig. 25 Reduced intrahepatic portal vessels (white arrow) and intrahepatic shunt (red arrow), G: gallbladder

Portal vein diameter

As mentioned above, the ratio between the diameter of the portal vein and the aorta is normally between 0.71 and 1.25. If this is reduced, the presence of an extrahepatic shunt or portal hypoplasia is suspected (Fig. 26). Conversely, if it is increased, the presence of an intrahepatic shunt or portal hypertension is suspected.

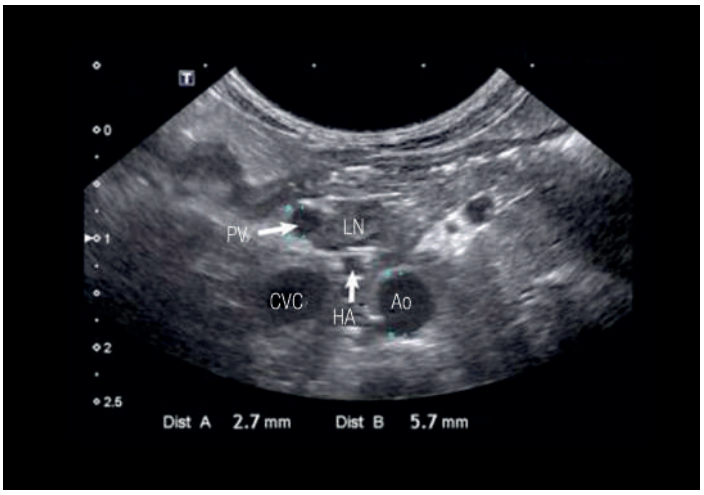

Fig. 26 PV: portal vein, CVC: caudal vena cava, Ao: aorta, HA: hepatic artery, LN: lymph node.[1]

Portal flow

As previously illustrated, portal flow is normally hepatopetal and has a velocity of approximately 15-20 cm/s in the dog and 10-18 cm/s in the cat. Reduction below 10 cm/s indicates the presence of portal hypertension, the causes of which must be thoroughly investigated.

Portal vein tributaries

Under normal conditions, all tributary veins of the portal vein should be smaller in diameter than the portal itself.

Turbulence in the caudal vena cava

Portocaval shunts cause an increase in the caliber of the caudal vena cava at their point of entry, associated with turbulence in the flow, which can be identified using color Doppler. (Fig. 27)

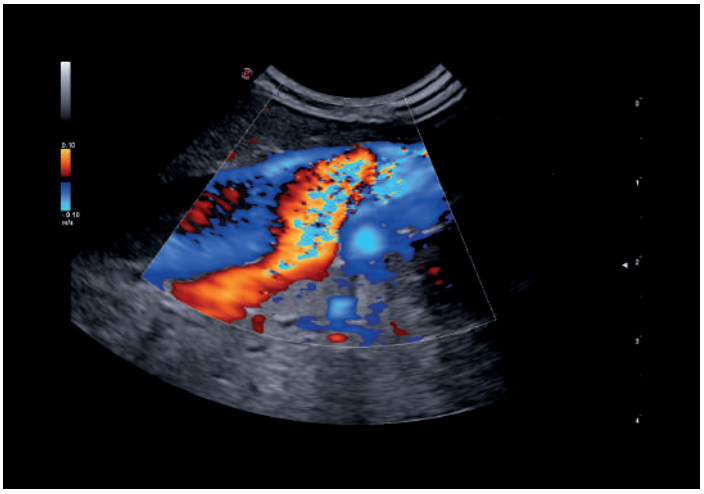

Fig. 27 Entry point of a portocaval shunt

Azygos vein

Under normal conditions, the azygos vein is not visible with ultrasound, but evidence of an additional vessel running along the aorta with cranially directed flow should prompt suspicion of a porto-azygos shunt.

Nephromegaly and urolithiasis

The existence of a PSS causes liver failure, which may result in a deficiency of the enzyme urate oxidase and subsequent formation of ammonium urate crystals. Another common finding is an increase in renal volume, for which several etiological hypothesis have been proposed.

Acquired portosystemic shunts

The presence of multiple tortuous vessels, most frequently evident caudally at the caudal pole of the left kidney, is an indication of the opening of PSS, the primary cause of which must be investigated. (Fig. 28)[14]

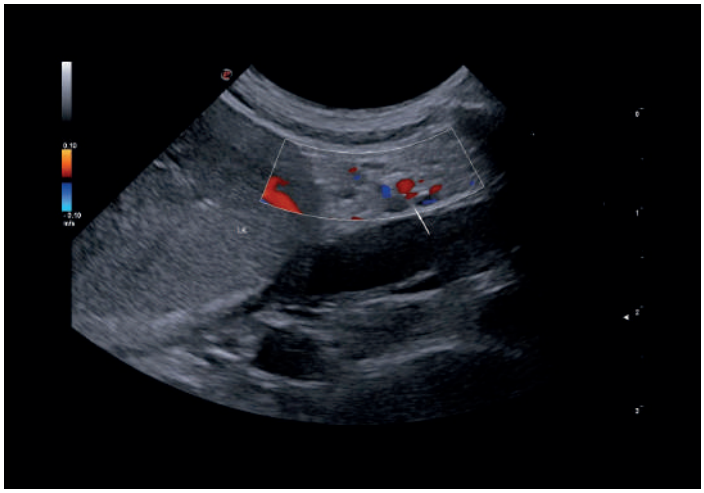

Fig. 28 Multiple tortuous vessels (arrow) caudal to the caudal pole of the left kidney (LK)

When examining a patient with suspected portosystemic shunt, the limitations of ultrasound technique must always be considered. Therefore, a second level diagnostic must always be taken into account, such as CT angiography, which allows highly accurate localization and characterization of the vascular anomaly, especially with a view to its surgical resolution.[15,16] (Fig. 29)

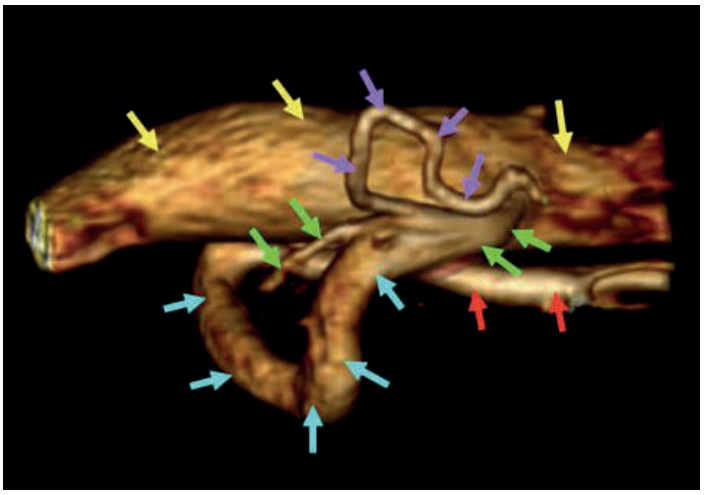

Fig. 29 Yellow arrows: caudal vena cava, red arrows: portal vein, purple arrows: cranial splenic vein, blue arrows: right gastric vein/loop, green arrows: left gastric vein/shunt.
Courtesy of Prof. Davide Zani, Department of Radiology, Lodi University Veterinary Hospital, University of Milan

Conclusions

Ultrasonography is an excellent method for examining the liver, allowing good visualization without the need for sedation of the patient, especially when interpreted with the background of hematochemical tests.

However, the limits of its specificity must be borne in mind and the possibility of taking samples or further investigation with the use of contrast media or second level imaging techniques must always be considered.

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