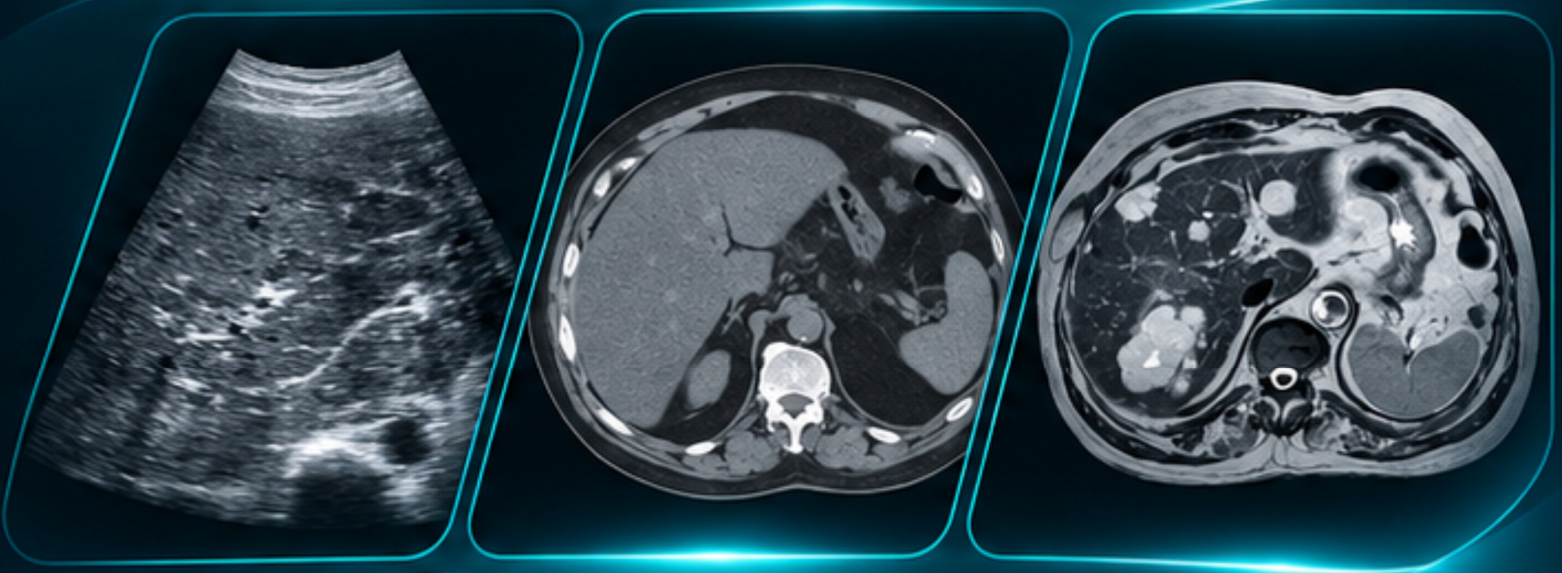
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Preface

Liver disease presents a wide range of diagnostic challenges. Similar clinical or laboratory findings may arise from different causes, while one disease can affect the hepatic parenchyma, vasculature, and biliary system in distinct ways. Imaging is central to recognizing these patterns, characterizing focal abnormalities, assessing disease extent, and guiding surveillance and treatment. Its value, however, depends on asking the right clinical question and selecting a technique suited to that question.

In this monograph, we bring together the anatomical and physiological foundations of hepatic imaging with the mechanisms and major manifestations of liver disease. We then examine the principal diagnostic methods, from conventional and Doppler ultrasound to elastography and contrast-enhanced ultrasound, computed tomography, magnetic resonance imaging, and nuclear medicine. Dedicated discussion of hybrid imaging, artificial intelligence, and radiomics places newer approaches within the broader clinical context. The comparative chapters consider where these methods complement one another and where their technical or practical limitations should guide interpretation.

The book is intended for radiologists, hepatologists, trainees, and other clinicians who seek a structured overview of imaging in liver disease. Our aim is to connect the strengths of each examination to its appropriate clinical role, while emphasizing that imaging findings are most meaningful when interpreted alongside the patient's history, laboratory results, and, when indicated, pathology and multidisciplinary review.

As imaging technology continues to evolve, careful clinical reasoning remains essential. We hope this monograph will serve as a useful reference for study and practice and encourage continued collaboration between imaging specialists and clinicians caring for patients with liver disease..

Acknowledgements

I would like to express my heartfelt gratitude to my family for their unwavering support throughout this demanding yet rewarding journey. Their reassurance, patience, and daily encouragement have been invaluable. I am also sincerely grateful to my collaborators for their support, dedication, and valuable contribution along the way.

Abstract

Liver disease encompasses a broad spectrum of inflammatory, metabolic, toxic, autoimmune, vascular, cholestatic, and neoplastic disorders. Imaging contributes to their detection, characterization, staging, and follow-up, but the usefulness of each modality varies according to the diagnostic objective. This monograph provides a narrative comparison of the principal techniques used in hepatic assessment, relating imaging findings to anatomical organization, pathophysiological mechanisms, and clinical and laboratory manifestations.

Conventional ultrasound remains widely used for initial assessment and surveillance because it is accessible, relatively inexpensive, and free of ionizing radiation. Doppler ultrasound adds hemodynamic information, while ultrasound elastography supports non-invasive assessment of fibrosis risk. Contrast-enhanced ultrasound provides continuous observation of lesion perfusion and is particularly useful for selected focal abnormalities. More recent Multiparametric Ultrasound (MPUS) adds value to the ultrasound examination. Multiphasic computed tomography offers rapid and comprehensive anatomical evaluation, with major applications in trauma, oncological staging, vascular disease, and acute complications. Magnetic resonance imaging combines multiple tissue contrast mechanisms with dynamic enhancement, diffusion imaging, quantitative fat and iron assessment, magnetic resonance elastography, and cholangiopancreatography. Nuclear medicine and hybrid techniques provide complementary functional and molecular information for selected clinical questions.

The comparison supports an indication-based, multimodal approach. No single examination provides an optimal solution for every liver disorder. Diagnostic performance must be interpreted in relation to lesion size, underlying liver disease, technical quality, contrast agent, and reference standard. Emerging computational methods may improve quantitative analysis and risk prediction, but their wider clinical use requires reproducibility, external validation, and evidence of benefit within real clinical workflows.

Keywords: ***Liver Disease; Ultrasound; Multiparametric Ultrasound (MPUS); Computed Tomography; Magnetic Resonance Imaging; Elastography; Contrast-Enhanced Ultrasound (CEUS); Nuclear Medicine; Radiomics.***

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Chapter 1. Anatomical and Physiological Foundations

1.1 Embryological development

The liver begins to develop during the fourth week of embryonic life through interactions between endodermal epithelial cells and the surrounding mesoderm. Its epithelial component originates in the hepatic diverticulum, a ventral outgrowth of the foregut that extends toward the septum transversum. Local developmental signals promote the differentiation and proliferation of hepatoblasts, which subsequently give rise to hepatocytes and the epithelial cells of the intrahepatic bile ducts. The surrounding tissues participate in the development of the supporting stroma and vascular environment.

The hepatic diverticulum separates into a larger cranial component, the pars hepatica, and a smaller caudal component, the pars cystica. The former develops into the principal hepatic mass; the latter contributes to the gallbladder and cystic duct. Remodeling of the vitelline and umbilical venous systems establishes the characteristic vascular organization of the developing liver and contributes to sinusoidal formation.

The fetal liver also serves as a major hematopoietic organ. Its rapid growth and close relationship to the septum transversum link hepatic development to that of the diaphragm. Disturbances of these processes can produce congenital biliary abnormalities, variations in hepatic configuration, and vascular anomalies. These developmental relationships are relevant to imaging because anatomical variants may alter the appearance of the liver, modify perfusion, or complicate surgical and interventional planning.

1.2 Gross anatomy and anatomical relationships

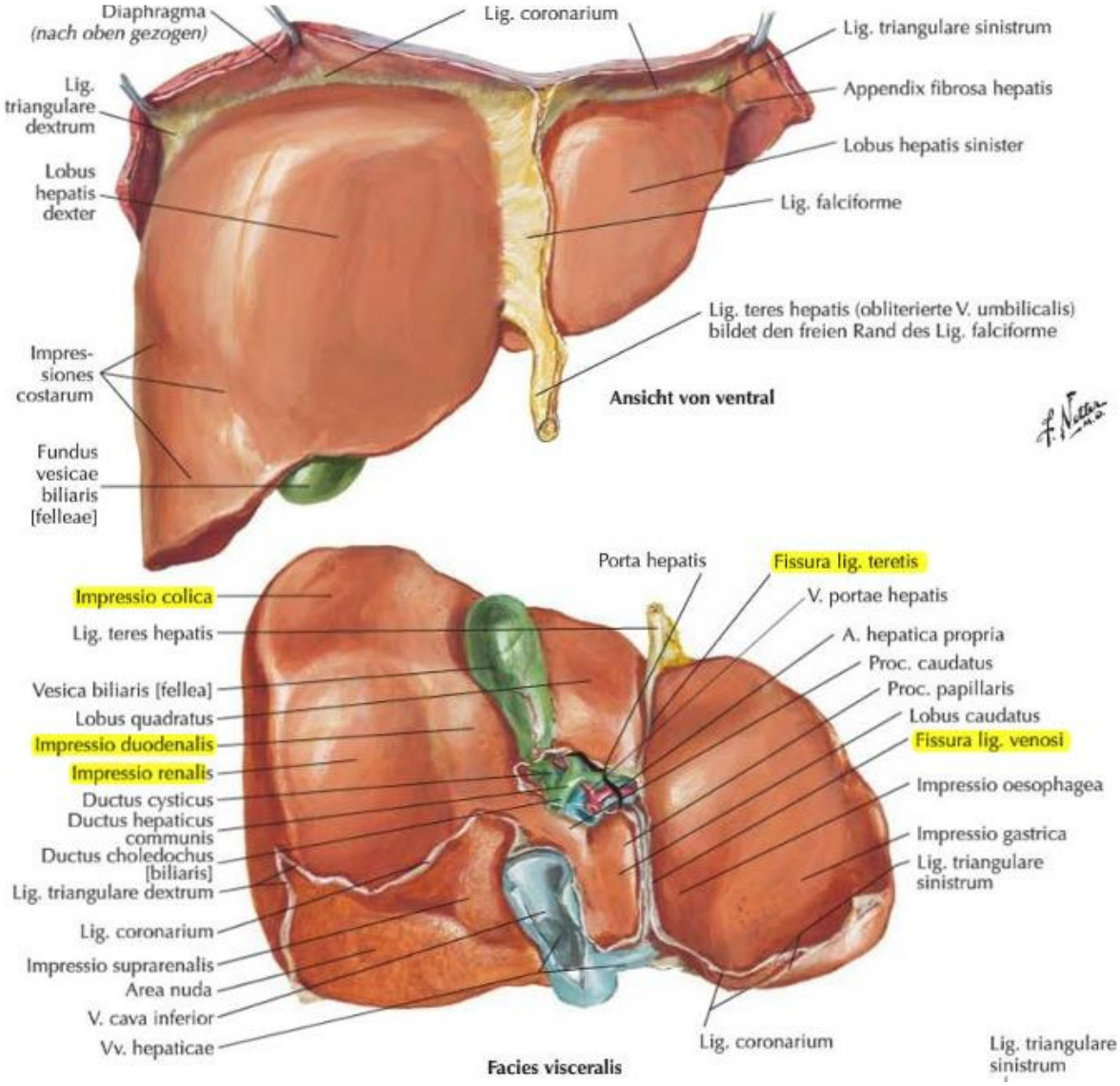
The liver is the largest solid visceral organ and a central regulator of metabolic homeostasis. In adults it commonly weighs approximately 1,200–1,500 g, although weight varies with body size, sex, and physiological or pathological conditions. It lies predominantly in the right upper quadrant, extends into the epigastrium, and may reach the left hypochondrium. Its reddish-brown appearance reflects its rich blood supply and sinusoidal organization.

The diaphragmatic surface is smooth and convex and follows the contour of the diaphragm. The visceral surface is more irregular, with impressions related to the stomach, duodenum, colon, right kidney, and right adrenal gland. These relationships influence the sonographic windows available for examination and help explain the routes by which inflammatory, neoplastic, or traumatic processes may involve adjacent structures.

Most of the organ is covered by visceral peritoneum. The principal exception is the bare area, where the liver is directly related to the diaphragm. A fibrous capsule, traditionally termed Glisson's capsule, encloses the parenchyma and continues along the intrahepatic vascular and biliary structures. Distension of the capsule contributes to the discomfort associated with hepatomegaly, congestion, and some acute inflammatory processes.

Morphologically, the liver is described as having right, left, quadrate, and caudate lobes. The right lobe is the largest. The quadrate lobe lies on the inferior surface between the gallbladder fossa and the fissure for the ligamentum teres. The caudate lobe is situated posteriorly, adjacent to the inferior vena cava, and has distinctive vascular relationships, including direct venous drainage through small hepatic veins.

Peritoneal reflections contribute to the stability of the organ. The falciform ligament marks a superficial division between the anatomical right and left lobes and contains the ligamentum teres, the remnant of the fetal umbilical vein. The coronary and triangular ligaments connect the liver to the diaphragm. These surface landmarks aid orientation, but they do not define the functional boundaries used in modern hepatic surgery.



Gross anatomy of the liver, showing its surfaces, lobes, ligaments, and visceral relationships (Netter).

1.3 The porta hepatis and dual blood supply

The porta hepatis is a transverse fissure on the visceral surface through which the major portal structures pass. These include the portal vein, hepatic artery, and hepatic ducts, accompanied by lymphatic vessels and autonomic nerves. Their arrangement and branching patterns are fundamental to the interpretation of hilar abnormalities and to the safety of hepatic and biliary interventions.

The liver receives blood from two sources. The *portal vein supplies approximately 70–75% of total hepatic blood flow* and delivers nutrient-rich venous blood from the gastrointestinal tract and associated organs. The hepatic arterial system supplies the remainder and contributes substantially to oxygen delivery. The relative arterial and portal contributions may change in disease, helping to explain the enhancement patterns used in multiphasic CT, MRI, and contrast-enhanced ultrasound (CEUS).

Venous outflow is mainly through the right, middle, and left hepatic veins into the inferior vena cava. The hepatic veins and portal branches provide important internal landmarks. Disruption of portal inflow, sinusoidal passage, or venous outflow produces different patterns of perfusion and congestion. Imaging interpretation should therefore distinguish disease of the portal circulation from hepatic venous obstruction and systemic venous congestion.

Vascular variants are common and may involve arterial origins, portal branching, or venous drainage. Their recognition is particularly important before transplantation, major resection, embolization, and other interventions in which the perfusion of the remaining liver must be preserved.

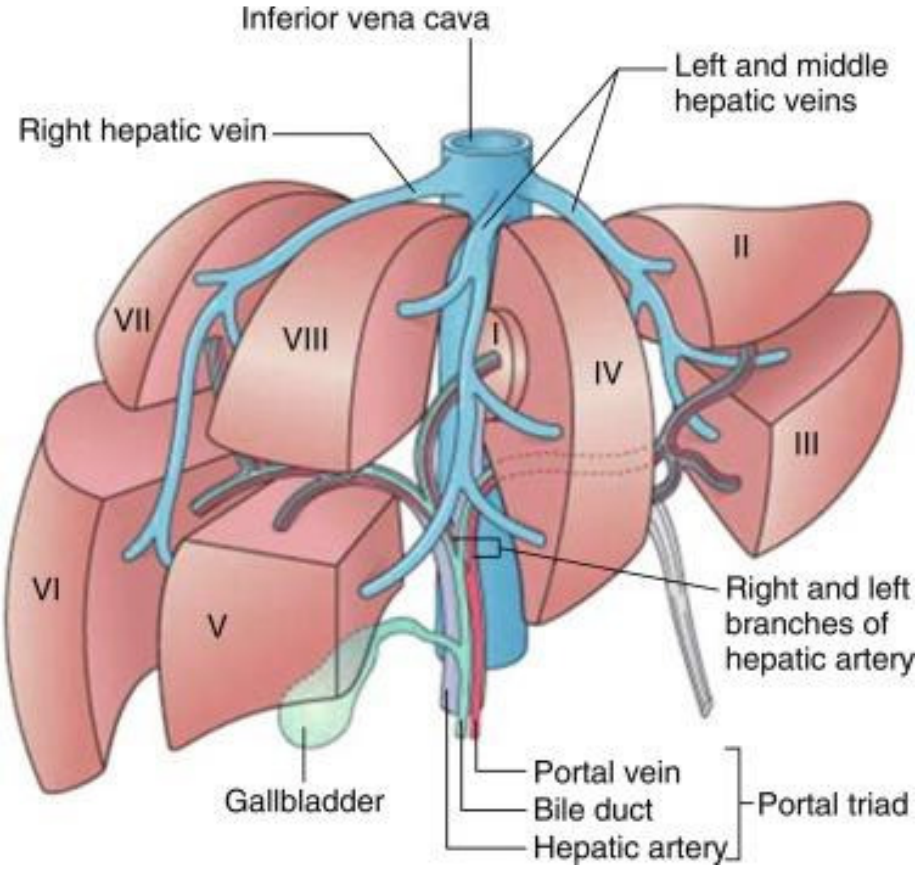
1.4 Functional segmentation

Couinaud's segmentation divides the liver into eight functionally defined segments according to the distribution of portal pedicles and the relationships of the hepatic veins. Each segment has its own portal venous and arterial inflow and biliary drainage, while the major hepatic veins principally occupy intersegmental planes. This distinction is important: functional segments are not simply miniature anatomical lobes with completely separate venous outflow systems.

Segment I correspond to the caudate lobe. Segments II and III constitute the left lateral section, and segment IV the left medial section. The right liver contains segments V–VIII. The functional division between right and left liver follows a plane related to the middle hepatic vein, rather than the falciform ligament alone.

Segmental localization allows the radiologist to communicate precisely with surgeons and interventional specialists. A report that identifies a lesion's segment, its relation to major vessels and bile ducts, and its proximity to the capsule is more useful than a general description of a right- or left-lobe mass. The same information supports segmentectomy, anatomical hepatectomy, ablation planning, and living-donor graft assessment.

Knowledge of segmentation also explains the regional distribution of some abnormalities. Portal obstruction may produce segmental perfusion changes; biliary obstruction may cause sectoral duct dilatation or atrophy; altered venous drainage may create focal congestion. A regional abnormality should therefore be interpreted in the context of the vascular and biliary territory it occupies.



Functional segmentation of the liver according to Couinaud, with the principal vascular and biliary landmarks.

1.5 Microscopic organization

The liver's microscopic architecture connects its metabolic activity to the circulation. Hepatocytes form plates separated by sinusoids through which mixed portal and arterial blood flows. Hepatocytes are polarized: their sinusoidal surface participates in exchange with blood, while their canalicular surface contributes to bile formation. Their abundant mitochondria, endoplasmic reticulum, and other organelles support synthesis, metabolism, and detoxification.

The classical hepatic lobule is conventionally represented as a hexagonal unit centered on a central vein, with portal tracts at its periphery. A portal tract contains branches of the portal vein and hepatic artery and a bile duct, together with lymphatic and neural elements. Blood passes from the portal region through the sinusoids toward the central vein; bile travels in the opposite direction toward the portal biliary system.

Sinusoidal endothelial cells are fenestrated and lack a continuous conventional basement membrane. The space of Disse separates the sinusoidal endothelium from the hepatocytes and provides a site for metabolic exchange. Hepatic stellate cells reside in this perisinusoidal compartment. In the healthy liver they store vitamin A; during persistent injury they become activated and contribute to extracellular matrix deposition and fibrosis.

Kupffer cells are resident hepatic macrophages associated with the sinusoids. They remove microorganisms, cellular debris, and other material delivered through the portal circulation and participate in immune surveillance. Their activity also contributes to inflammatory signaling and provides the biological basis for selected imaging techniques that depend on reticuloendothelial uptake.

The hepatic acinus offers a complementary functional model. It describes tissue according to its position along the perfusion gradient. Zone 1 is periportal and receives blood relatively rich in oxygen and nutrients. Zone 2 occupies an intermediate position. Zone 3 is pericentral, has lower oxygen availability, and contains important drug-metabolizing activity. These differences help explain the zonal distribution of ischemic injury, some toxic effects, and several metabolic processes.

The biliary canaliculi are formed between adjacent hepatocytes and are sealed by junctional complexes. They drain into the canals of Hering, which form the transition to the ductular system. Cholangiocytes line the progressively larger intrahepatic ducts. This organization clarifies why cholangiopathies may produce a pattern of injury different from primary hepatocellular disease.

1.6 Biliary anatomy and physiological function

The right and left hepatic ducts unite to form the common hepatic duct. The cystic duct connects the gallbladder to this system, and their junction forms the common bile duct. Bile reaches the duodenum through the distal biliary pathway. The gallbladder acts as a reservoir and concentrates bile between meals. Anatomical variants of this system are relevant to cholangiography, cholecystectomy, transplantation, and biliary intervention.

The physiological role of the liver extends across several interdependent processes. It regulates blood glucose through storage and release of carbohydrate substrates, participates in amino acid metabolism and urea synthesis, and coordinates lipid synthesis, oxidation, and export. It synthesizes albumin and many coagulation-related proteins, metabolizes endogenous and exogenous compounds, and produces bile for the elimination of bilirubin and the intestinal handling of lipids.

These functions explain why liver disease can have systemic consequences even when structural abnormalities appear modest. Conversely, marked anatomical change may coexist with sufficient functional reserve to maintain clinical compensation. Imaging, laboratory tests, and clinical assessment therefore describe related but non-identical aspects of hepatic health. A large liver is not necessarily a highly functional liver, and a normal-appearing liver does not exclude microscopic disease.

Chapter 2. Mechanisms of Liver Injury

2.1 Etiological framework

Liver injury may arise within the hepatocytes, bile ducts, vascular system, or supporting microenvironment. Its causes can be grouped as infectious, toxic, metabolic, autoimmune, vascular, obstructive, and neoplastic. The initial insult may be acute, recurrent, or persistent, while the subsequent course depends on its intensity and duration and on the regenerative and immune responses of the host.

Hepatotropic viruses include hepatitis A, B, C, D, and E. Their routes of transmission, propensity for chronic infection, and relationship to carcinogenesis differ. Alcohol and medications produce injury through a combination of direct toxicity, reactive metabolites, oxidative stress, immune responses, and interference with cellular or biliary transport. Drug-induced liver injury also includes injury caused by herbal products and dietary supplements.

Metabolic disease includes steatotic liver disease associated with cardiometabolic dysfunction and inherited disorders such as hemochromatosis, Wilson disease, and alpha-1 antitrypsin deficiency. These disorders do not share a single imaging signature. Some permit quantitative tissue assessment, particularly hepatic fat and iron measurement, while others are identified primarily through laboratory and genetic investigation, with imaging documenting structural consequences.

Autoimmune hepatitis primarily targets hepatic parenchyma, whereas primary biliary cholangitis and primary sclerosing cholangitis predominantly involve the biliary system. Vascular causes include portal vein thrombosis, hepatic venous outflow obstruction, and congestion related to cardiac disease. Biliary obstruction may result from stones, strictures, or tumors. Primary and secondary malignancies form a further major category, often interacting with underlying chronic liver disease.

This etiological framework is clinically useful because it prevents imaging findings from being interpreted without context. Steatosis, hepatomegaly, heterogeneous enhancement, or ascites can each arise from several mechanisms. A diagnosis becomes more reliable when the observed pattern is related to exposures, biochemical findings, serology, and the distribution of the abnormality.

2.2 Inflammation and immune signaling

Inflammation is a coordinated response to infection and tissue damage. Kupffer cells and other immune cells recognize microbial products and signals released by injured cells. Their activation promotes cytokine production, leukocyte recruitment, and changes in hepatocyte function. Mediators discussed in the source include tumor necrosis factor alpha and interleukins 1 beta and 6.

An acute inflammatory response may help eliminate the initiating insult. Persistent inflammation, however, maintains cellular injury and stimulates fibrogenic pathways. It links the original etiology to progressive architectural change. Imaging may show enlargement, edema, altered perfusion, or secondary biliary changes, but these manifestations are often non-specific and cannot establish the precise immune mechanism.

The severity of biochemical injury and the degree of established fibrosis must also be distinguished. A patient with acute inflammation may have markedly elevated aminotransferases, without advanced fibrosis. A patient with longstanding cirrhosis may have relatively modest aminotransferase elevation despite severely impaired reserve. The interpretation of elastography is especially dependent on this distinction because inflammation can increase measured stiffness independently of fibrosis.

2.3 Oxidative stress and metabolic dysfunction

Oxidative stress develops when reactive oxygen species exceed the capacity of cellular antioxidant systems. Mitochondrial metabolism, cytochrome P450 activity, and inflammatory cells contribute to their production. Reactive species can damage lipid membranes, proteins, and nucleic acids and can amplify inflammatory signaling.

Alcohol metabolism illustrates the interaction between toxic exposure and metabolic injury. Acetaldehyde formation and induction of CYP2E1 promote oxidative stress, while alterations in lipid handling favor steatosis. In metabolic steatotic liver disease, excess substrate delivery, insulin resistance, lipid intermediates, and mitochondrial stress can similarly contribute to hepatocellular injury.

Imaging can demonstrate fat accumulation and some consequences of inflammation, but does not directly establish the balance of individual biochemical pathways. Quantitative fat measurements should therefore be interpreted as measurements of tissue composition, rather than as complete measures of inflammatory activity or disease severity.

2.4 Necrosis and apoptosis

Necrosis involves loss of membrane integrity and release of cellular contents, which can provoke a substantial inflammatory response. Ischemia, severe toxic injury, and mitochondrial failure may lead to ATP depletion, disruption of ion transport, cellular swelling, and cell death. Extensive hepatic necrosis can produce acute liver failure.

Apoptosis is a regulated form of cell death initiated through receptor-mediated or mitochondrial pathways. Caspase activation leads to controlled cellular dismantling. It contributes to the removal of infected hepatocytes in viral hepatitis and may also occur in lipotoxic injury. Although apoptosis is organized at the cellular level, persistent apoptotic injury can still stimulate inflammation and fibrogenesis.

The imaging consequences depend on the scale and distribution of injury. Diffuse microscopic cell death may produce no specific structural abnormality, whereas extensive ischemia, infarction, or necrotic tumor can produce visible changes. Neither a normal CT nor a normal conventional MRI excludes biologically important hepatocellular injury.

2.5 Fibrogenesis and architectural remodeling

Fibrosis is the accumulation of extracellular matrix in response to sustained injury. Activated stellate cells and other fibrogenic cells acquire myofibroblastic characteristics and increase the production of collagen and associated matrix components. The balance between deposition and degradation determines whether fibrosis progresses, stabilizes, or regresses.

Progressive matrix accumulation distorts the sinusoidal environment and may form bridging septa. Regeneration within this altered architecture produces nodules and contributes to cirrhosis. Both fixed structural resistance and dynamic vascular changes increase resistance to portal flow. The resulting portal hypertension is therefore not explained by collagen deposition alone.

The imaging methods used to assess this process measure different consequences. Conventional ultrasound, CT, and MRI demonstrate morphological remodeling, especially in advanced disease. Elastography measures mechanical stiffness as an indirect marker. Laboratory indices incorporate biochemical and hematological features associated with fibrosis risk. Histology provides a tissue sample, but is subject to sampling and interpretation limitations. These approaches are complementary rather than interchangeable.

Fibrosis may partially regress after effective control of the cause, but regression of a stiffness measurement does not by itself prove complete histological reversal. Changes in inflammation and congestion may contribute to the observed reduction. Longitudinal interpretation should consider the treatment interval, clinical state, measurement technique, and persistence of complications.

Chapter 3. Major Clinical Patterns of Liver Disease

3.1 Clinical and biochemical classification

The initial evaluation of liver disease commonly distinguishes hepatocellular, cholestatic, and mixed patterns. Hepatocellular injury primarily affects hepatocytes and is usually associated with aminotransferase abnormalities. Cholestatic injury reflects impaired bile formation or flow and is associated with a different biochemical pattern. Mixed injury combines features of both.

Symptoms such as jaundice, fatigue, pruritus, right upper quadrant discomfort, anorexia, nausea, abdominal distension, and gastrointestinal bleeding can direct the investigation. Many patients, however, are diagnosed during investigation of incidental laboratory or imaging abnormalities. The absence of symptoms does not exclude chronic disease or clinically important fibrosis.

The diagnostic sequence should establish the likely category of injury, identify the cause, assess severity and stage, and detect complications that alter management. Imaging addresses structural and vascular questions within this sequence. It cannot independently identify every etiology, determine all aspects of functional reserve, or exclude disease when microscopic abnormalities predominate.

3.2 Hyperbilirubinemia and jaundice

Bilirubin fractionation helps distinguish disorders of production, uptake, conjugation, and excretion. Predominantly unconjugated hyperbilirubinemia may reflect hemolysis, ineffective erythropoiesis, impaired hepatic uptake, or reduced conjugation. In isolated hemolysis with otherwise preserved liver function, bilirubin elevation is usually modest; a disproportionately high concentration raises the possibility of an additional hepatic process.

Ineffective erythropoiesis can occur in thalassemia, megaloblastic anemia, and other hematological disorders. Resorption of a large hematoma may also transiently increase bilirubin production. Reduced conjugation is associated with neonatal physiological immaturity, selected medications, Gilbert syndrome, and Crigler–Najjar syndromes. Severe unconjugated hyperbilirubinemia in susceptible neonates can cause bilirubin encephalopathy.

Predominantly conjugated hyperbilirubinemia may arise from impaired canalicular secretion, hepatocellular injury, or obstruction. Dubin–Johnson syndrome is associated with impaired canalicular transport related to ABCC2/MRP2 and characteristic hepatic pigmentation. Rotor syndrome involves abnormalities of hepatic uptake transporters and does not produce the same pigmentation. Recurrent and progressive inherited cholestatic disorders represent a further group with different clinical trajectories.

In acquired hepatitis and biliary obstruction, both bilirubin fractions may rise. Their relative proportions do not reliably distinguish intrahepatic cholestasis from extrahepatic obstruction. Imaging is most helpful when the clinical question concerns duct dilatation, an obstructing lesion, a mass, or complications. Ultrasound usually provides the initial anatomical assessment, with MRCP or CT selected according to the unresolved question. Endoscopic Ultrasound (EUS) is the most sensitive method for the evaluation of an obstructive jaundice and can be followed by ERCP for therapy.

3.3 Acute viral hepatitis: shared features

The five principal hepatotropic viruses produce overlapping acute clinical syndromes despite substantial differences in virology and transmission. Hepatitis B is caused by a DNA virus that replicates through reverse transcription; the other principal agents contain RNA. The spectrum ranges from asymptomatic infection to symptomatic hepatitis and, less frequently, acute liver failure.

The common symptomatic course includes a prodromal period of fatigue, anorexia, nausea, and malaise, followed in some patients by jaundice, dark urine, and pale stools. Histological changes may include hepatocyte ballooning, lobular inflammation, apoptosis, and focal or confluent necrosis. None of these broad patterns identifies the viral agent without virological evidence.

Imaging findings are also generally non-specific. The principal diagnostic tests are serological and molecular assays interpreted with the clinical presentation. Ultrasound is useful for excluding biliary obstruction and documenting gross changes or complications. CT and MRI are selected when an alternative diagnosis, vascular abnormality, severe complication, or established chronic disease requires assessment.

3.4 Hepatitis A

Hepatitis A virus belongs to the Picornaviridae family and is transmitted mainly by the fecal–oral route through contaminated food or water and close personal contact. Infection is often acquired in childhood in areas with inadequate sanitation and may be clinically silent. In populations with lower childhood exposure, susceptible adults may experience more symptomatic disease.

After infection, the virus reaches the liver and is excreted in bile and feces. Hepatic injury is strongly related to the host immune response. The average incubation period is approximately four weeks. Symptomatic infection typically progresses from non-specific systemic symptoms to an icteric phase, with substantial aminotransferase elevation and eventual recovery.

Hepatitis A does not cause chronic infection. Prolonged cholestatic illness and relapsing courses may occur, while acute liver failure is uncommon but important, particularly in vulnerable patients or those with pre-existing liver disease. Recovery generally provides lasting immunity. The imaging contribution remains supportive rather than etiologically diagnostic: a normal scan does not exclude infection, and an abnormal scan does not establish hepatitis A.

3.5 Hepatitis B

Hepatitis B virus is transmitted through infected blood and body fluids, including perinatal, sexual, and parenteral exposure. The likelihood of chronic infection depends strongly on age at acquisition. Most immunocompetent adults recover from acute infection, whereas infection acquired in infancy or early childhood has a much greater tendency to persist.

The clinical outcome reflects the interaction between viral persistence and host immunity. Chronic disease can show changing levels of viral replication and inflammatory activity over time. Histology may demonstrate portal inflammation, interface hepatitis, and progressive fibrosis. Ground-glass hepatocytes reflect accumulation of viral surface antigen within the endoplasmic reticulum.

The major long-term consequences are cirrhosis, hepatic decompensation, and hepatocellular carcinoma. HCC can occur in chronic hepatitis B even without cirrhosis, an important distinction when determining surveillance eligibility. Risk assessment must therefore incorporate the underlying infection and clinical profile rather than relying solely on cirrhotic morphology.

Imaging cannot determine viral replication or immune phase. It assesses the consequences of disease, including fibrosis-related remodeling, portal hypertension, focal lesions, and vascular involvement. Surveillance and diagnostic examinations serve different purposes and should not be conflated.

3.6 Hepatitis C

Hepatitis C virus is an RNA virus of the Flaviviridae family. Transmission occurs predominantly through exposure to infected blood. Acute infection is often asymptomatic or only mildly symptomatic and may

consequently go unrecognized. Viral persistence is facilitated by genetic variability and evasion of host antiviral responses.

Chronic hepatitis C can remain clinically silent for many years. The rate of fibrosis progression varies with age, alcohol exposure, metabolic comorbidity, coinfection, and other host factors. Histological findings include portal lymphocytic inflammation, interface activity, and fibrosis; lymphoid aggregates or follicles may be present. Advanced disease may develop into cirrhosis with portal hypertension and increased HCC risk.

Extrahepatic manifestations, including mixed cryoglobulinemia and related renal or systemic disorders, reflect the broader biological effects of infection. These features reinforce the need to integrate hepatic imaging with clinical and laboratory evaluation.

Ultrasound and cross-sectional imaging assess structural consequences and detect complications. Elastography contributes to non-invasive staging and follow-up. After successful treatment, changes in inflammatory activity may affect liver stiffness, and imaging surveillance requirements depend on the residual risk profile rather than on viral clearance alone.

3.7 Hepatitis D

Hepatitis D virus requires hepatitis B surface antigen for the production of infectious enveloped particles. It may occur as simultaneous HBV–HDV coinfection or as superinfection in a person with established hepatitis B. These situations have different natural histories.

Coinfection may produce severe acute hepatitis but often resolves when both infections clear. Superinfection is more likely to establish persistent hepatitis D and is associated with aggressive necroinflammatory activity and relatively rapid progression toward advanced fibrosis and cirrhosis. Complications include acute liver failure, decompensation, portal hypertensive bleeding, and HCC in an appropriate chronic disease setting.

The imaging appearances reflect the resulting liver damage rather than the viral agent itself. Their importance lies in staging chronic structural disease, detecting focal lesions, and evaluating complications. A changing clinical course should prompt reassessment even if previous imaging was reassuring.

3.8 Hepatitis E

Hepatitis E virus is an RNA virus of the Herpesviridae family. Transmission includes contaminated water in endemic settings and zoonotic exposure, including inadequately cooked animal products, in other settings. The presentation can resemble hepatitis A, with a prodromal illness followed by jaundice and biochemical hepatocellular injury.

Most infections in immunocompetent individuals are self-limited. Chronic infection can occur in immunosuppressed patients, including transplant recipients, and may progress to fibrosis. Severe illness in pregnancy is particularly associated with some endemic epidemiological and viral genotype settings; this risk should not be generalized uniformly to all forms of HEV infection.

Histology may show features of acute hepatitis, sometimes with cholestasis, while severe cases can develop extensive necrosis. Neurological and other extrahepatic manifestations have also been described. Imaging helps exclude obstruction and evaluate complications, but infection is established through laboratory testing.

3.9 Drug-induced liver injury

Drug-induced liver injury encompasses injury caused by medications, herbal products, supplements, and other chemical exposures. Establishing causality requires a careful timeline of treatment, symptom onset,

biochemical abnormalities, withdrawal, and alternative causes. A plausible imaging appearance alone cannot prove that a particular drug is responsible.

Direct toxic injury is generally predictable and related to dose. Acetaminophen toxicity is the classical example. Idiosyncratic injury is less predictable and may involve reactive metabolites, immune responses, and genetic susceptibility. Its latency varies, and fever, rash, arthralgia, or eosinophilia may occur in selected cases.

Hepatic drug metabolism includes phase I reactions, often mediated by cytochrome P450, and phase II conjugation pathways. Reactive products may be neutralized through systems that include glutathione. Mitochondrial dysfunction, oxidative injury, and impaired canalicular transport can produce hepatocellular injury, steatosis, cholestasis, or mixed patterns.

Biochemical classification commonly uses the R ratio, calculated from the relative elevations of ALT and alkaline phosphatase above their respective upper limits of normal. Causality tools such as RUCAM can support structured evaluation, but clinical judgment and exclusion of competing explanations remain essential. Hepatocellular injury with jaundice in the appropriate Hy's law context signals increased concern for serious outcomes.

Imaging is particularly useful for excluding biliary obstruction, vascular disease, and focal pathology. It may document fatty change or other structural consequences but frequently remains non-specific. Histology is reserved for selected diagnostic questions rather than used automatically in every suspected case.

3.10 Chronic hepatitis

Chronic hepatitis describes persistent hepatic inflammation, conventionally lasting at least six months. It should be characterized by etiology, inflammatory activity, and fibrosis stage. These three dimensions answer different clinical questions: the cause determines the therapeutic target, activity reflects ongoing injury, and stage indicates accumulated structural damage and risk.

Causes include chronic viral infection, autoimmune hepatitis, persistent drug-related injury, and selected inherited or metabolic disorders. Histological assessment evaluates portal and lobular inflammation, interface hepatitis, cell death, and fibrosis. Advanced disease may develop bridging septa and regenerative nodules.

Many patients are asymptomatic. Others report fatigue or non-specific discomfort, while advanced disease produces ascites, jaundice, encephalopathy, or gastrointestinal bleeding. A normal ultrasound or conventional cross-sectional examination is compatible with early chronic hepatitis. Elastography and blood-based fibrosis tests are therefore often more informative for non-invasive staging than morphological imaging alone.

Biopsy remains useful when the diagnosis, activity, or competing etiologies cannot be resolved non-invasively and when the result will influence management. It should be understood as one component of evaluation rather than an inevitable prerequisite for every patient with chronic liver disease.

3.11 Alcohol-related liver disease

Alcohol-related liver disease includes steatosis, inflammatory hepatocellular injury, fibrosis, and cirrhosis. Alcohol-associated hepatitis is a distinct clinical syndrome that may develop on a background of chronic disease and can be associated with substantial short-term risk.

Ethanol is metabolized to acetaldehyde and then acetate. Chronic exposure can induce CYP2E1, increase oxidative stress, alter lipid oxidation and export, and promote inflammation. Intestinal dysbiosis and

increased permeability may augment hepatic immune stimulation. Susceptibility varies with exposure pattern, genetic factors, sex, metabolic comorbidity, and other liver disease.

Simple steatosis may be asymptomatic, whereas severe alcohol-associated hepatitis commonly presents with rapidly developing jaundice and systemic illness. Histological features can include macrovesicular steatosis, hepatocyte ballooning, Mallory–Denk bodies, neutrophilic inflammation, and pericellular or perivenular fibrosis. AST often exceeds ALT, but biochemical patterns require clinical interpretation and are not independently diagnostic.

Ultrasound, CT, and MRI identify steatosis and advanced structural complications. MRI-PDFF quantifies hepatic fat, and elastography supports fibrosis assessment. During active inflammation, elevated stiffness can overstate the contribution of fixed fibrosis. Imaging cannot reliably separate alcohol-related from metabolically associated steatosis without the history and other evidence.

3.12 Metabolic dysfunction-associated steatotic liver disease (MASLD)

The source thesis predominantly uses the historical terms non-alcoholic fatty liver disease and non-alcoholic steatohepatitis. Contemporary terminology includes metabolic dysfunction-associated steatotic liver disease (MASLD) and metabolic dysfunction-associated steatohepatitis (MASH). The newer framework is based on steatosis and cardiometabolic criteria and should not be treated as a purely linguistic substitution for every historical NAFLD cohort.

The biological spectrum includes fat accumulation, inflammatory hepatocellular injury, progressive fibrosis, and cirrhosis. Obesity, insulin resistance, hypertriglyceridemia and type 2 diabetes are important clinical associations. Genetic variation, including variants affecting lipid handling, contributes to individual susceptibility. Excess fatty acid delivery, lipogenesis, lipotoxicity, mitochondrial stress, inflammatory signaling, and altered gut–liver interactions participate in progression.

Aminotransferases may remain normal, and their concentrations do not reliably establish fibrosis stage. Conventional ultrasound commonly detects moderate or marked steatosis but may miss mild disease. MRI-PDFF offers quantitative fat assessment, while elastography evaluates stiffness. Fat content and fibrosis risk must be considered separately: a high fat fraction does not automatically imply advanced fibrosis, and advanced disease may not retain a conspicuously fatty appearance.

Non-invasive risk assessment combines blood-based scores (FIB4, APRI) with imaging techniques in an appropriate sequence. Fibrosis stage is a major determinant of liver-related risk. A selected biopsy may be necessary when inflammatory activity or competing diagnoses remain unresolved and the result will change management. Established imaging measurements of fat and stiffness do not independently reproduce every histological component of MASH.

3.13 Cirrhosis and portal hypertension

Cirrhosis represents advanced architectural remodeling characterized by diffuse fibrosis and regenerative nodules. Its major consequences are impaired hepatocellular function and increased resistance to portal blood flow. The etiological spectrum includes viral, alcohol-related, metabolic, autoimmune, cholestatic, and inherited disease, as well as persistent congestion and cryptogenic cases.

Compensated cirrhosis may remain clinically silent. Decompensation is marked by events such as ascites, hyperbilirubinemia, variceal bleeding, or hepatic encephalopathy and substantially changes prognosis. Laboratory findings can include increased bilirubin and INR, reduced albumin, and thrombocytopenia, although individual patterns vary with stage and context.

Portal hypertension reflects increased intrahepatic resistance together with circulatory changes, including increased splanchnic flow. A hepatic venous pressure gradient of at least 10 mmHg identifies clinically significant portal hypertension in the settings where this measurement is applicable. Imaging demonstrates consequences rather than directly replacing pressure measurement in every disease type.

Ascites develops through interacting hemodynamic, neurohormonal, and renal mechanisms. Portosystemic collateral vessels and splenomegaly may be present. Hepatic encephalopathy relates to impaired detoxification and shunting. Hepato-renal dysfunction and bacterial infection of ascitic fluid are important clinical complications whose diagnosis requires clinical and laboratory assessment.

Ultrasound may demonstrate surface nodularity, coarse or heterogeneous echotexture, altered hepatic volume, signs of portal hypertension, splenomegaly, and ascites. Doppler evaluates portal and hepatic venous flow. CT and MRI provide a broader assessment of segmental remodeling, collateral vessels, thrombosis, and focal lesions. Early compensated disease may be less conspicuous than advanced cirrhosis.

The cirrhotic liver also creates a difficult background for lesion detection. Regenerative nodules, dysplastic nodules, perfusion abnormalities, and HCC may overlap in appearance. Multiphasic examinations and standardized interpretive systems support lesion assessment. Surveillance of patients with advanced liver fibrosis remains a distinct process designed to identify disease before symptoms or advanced tumor burden develop.

3.14 Hepatobiliary, vascular, and neoplastic disease

The source section labeled hepatobiliary disease largely repeats its preceding discussion of cirrhosis. That duplicate material has been incorporated into Section 3.13. The biliary and vascular disorders described elsewhere in the thesis are brought together here to clarify their relationship to imaging.

Primary biliary cholangitis involves the small intrahepatic ducts, and early disease may not produce visible duct dilatation. Primary sclerosing cholangitis can involve both intrahepatic and extrahepatic ducts, with multifocal strictures and intervening dilatation. Obstruction caused by stones, benign strictures, or malignancy may produce upstream duct dilatation, but the absence of dilatation does not categorically exclude early or intermittent obstruction.

Portal vein thrombosis and hepatic venous outflow obstruction require separate assessment of the affected vascular territory. Congestive hepatopathy must be considered in relation to cardiac disease and systemic venous pressure. Doppler provides flow information, while CT and MRI establish anatomy, extent, associated perfusion changes, and potential causes.

HCC usually arises in a high-risk hepatic environment, although cirrhosis is not an absolute prerequisite. Intrahepatic cholangiocarcinoma and other primary tumors have different biological and enhancement patterns. Metastases are an important and frequent cause of malignant hepatic masses. The interpretation of a focal lesion must therefore take account of both the liver background and any known extrahepatic malignancy.

Benign masses and non-neoplastic lesions are equally important to recognize. Simple cysts, hemangiomas, focal nodular hyperplasia, adenomas, abscesses, and parasitic cysts require different management. Imaging is most valuable when it distinguishes an observation that can be safely characterized from one that requires additional imaging, tissue diagnosis, or intervention.

Chapter 4. Conventional and Doppler Ultrasound

4.1 Physical principles

Ultrasound forms images from high-frequency mechanical waves transmitted into the body and reflected by tissue interfaces. Medical diagnostic systems operate over a broad frequency range, commonly about 2–20 MHz, with lower frequencies favored for deeper abdominal structures and higher frequencies for superficial detail. The examination does not use ionizing radiation.

The transducer contains piezoelectric elements that convert electrical excitation into mechanical vibration and returning echoes into electrical signals. The system calculates depth from the interval between transmission and echo reception, using an assumed average propagation speed of approximately 1,540 m/s in soft tissue. The brightness assigned to each echo depends on its amplitude and on the processing settings.

Reflection is influenced by differences in acoustic impedance. Strong reflection at tissue–air and tissue–bone interfaces restrict visualization beyond bowel gas or ribs. Attenuation increases with depth and frequency, creating a fundamental compromise: a higher frequency improves potential spatial detail, but reduces penetration. The appropriate frequency is therefore the highest that still adequately visualizes the structure of interest.

B-mode imaging provides the principal anatomical display. A-mode represents amplitude as a function of depth and has limited routine relevance to hepatic imaging. M-mode displays movement over time and is mainly associated with cardiac applications. Tissue harmonic imaging can improve contrast and reduce selected artifacts, while time-gain compensation adjusts amplification at different depths.

Axial resolution depends on pulse length, and lateral resolution depends substantially on beam width and focusing. More focal zones can improve selected spatial detail, but may reduce frame rate. Gain, depth, focal position, and dynamic range should be adjusted to the diagnostic task, rather than left at arbitrary default values. A technically poor examination cannot be corrected simply by a more confident interpretation.

4.2 Examination technique and normal appearance

Liver ultrasound is commonly performed with a curvilinear abdominal transducer, often within a frequency range of approximately 3–5 MHz (multi frequency probe), adapted to patient habitus. Subcostal and intercostal approaches, changes in position, and respiratory maneuvers improve access to different segments. A higher-frequency linear transducer may help evaluate the anterior surface and subtle nodularity.

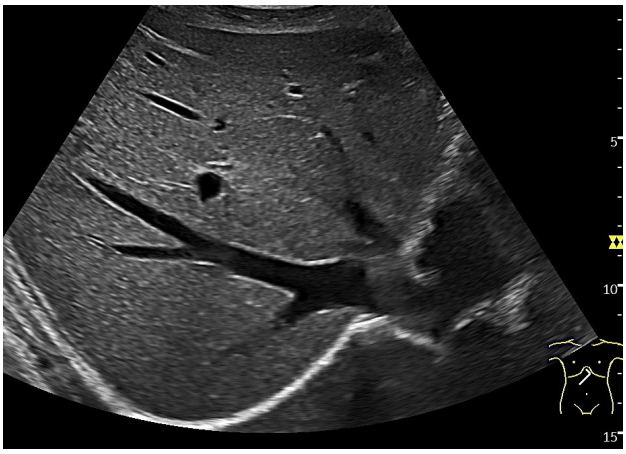
A complete examination assesses the liver's size, contour, echotexture, focal abnormalities, major vessels, and visible biliary system, together with relevant adjacent structures. The spleen, gallbladder, and presence of free fluid contribute to interpretation. If substantial areas are not adequately visualized, the limitation should be stated, because it affects confidence in excluding lesions. Experience plays a role for the assessment of all structures in the abdomen.



Normal liver and normal hepatic veins



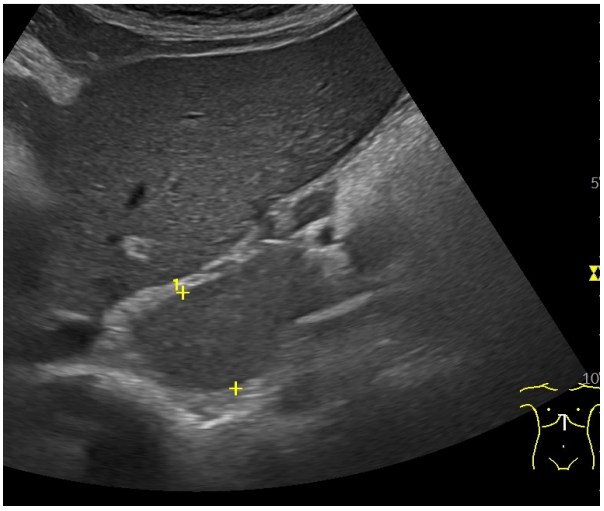
Normal liver and normal hepatic veins



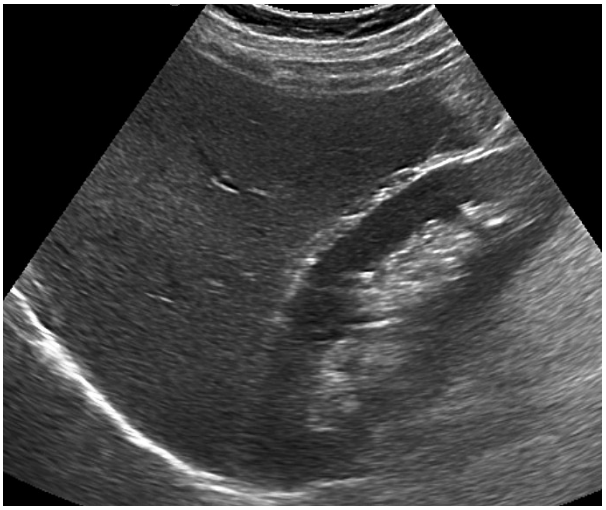
Normal liver with right hepatic vein



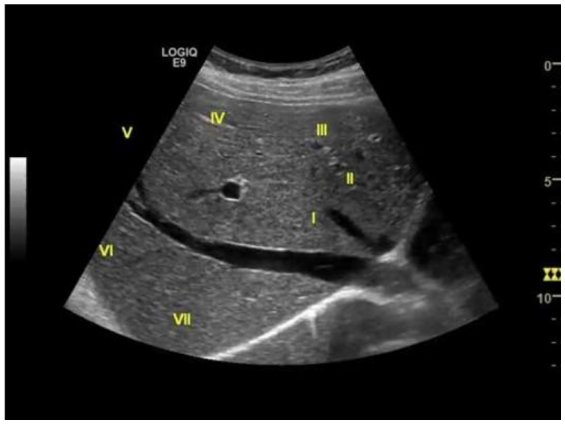
Normal caudate lobe (longitudinal section)

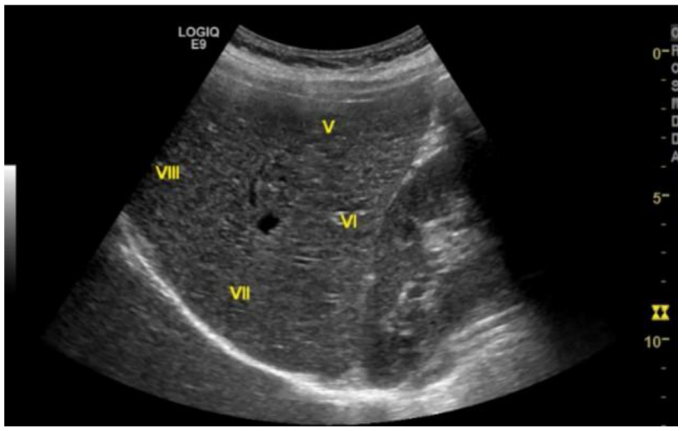


Normal caudate lobe (longitudinale section)



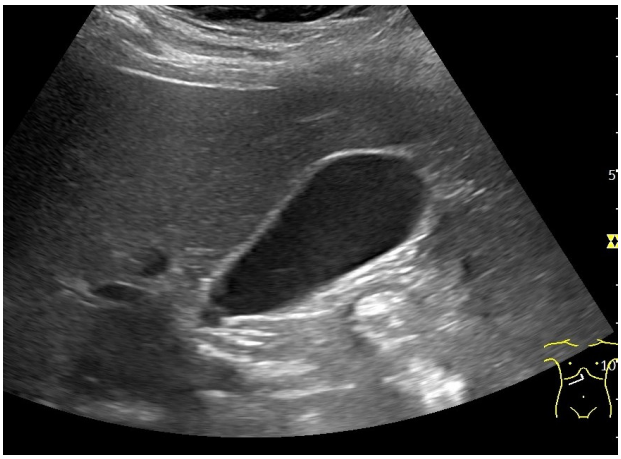
Liver and right kidney in the same section





Segmentation of the liver (oblique subcostal section)

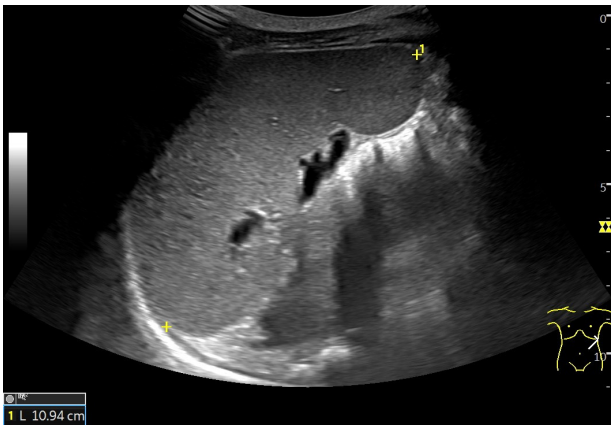
Segmentation of the liver (right intercostal aproach)





Normal gallbladder

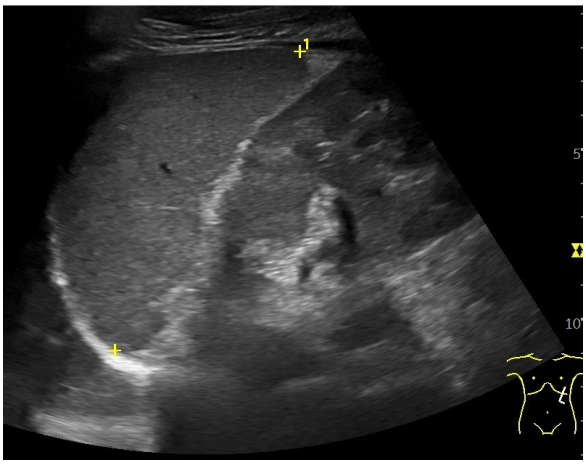
Normal gallbladder





Normal size of the spleen (<12 cm)

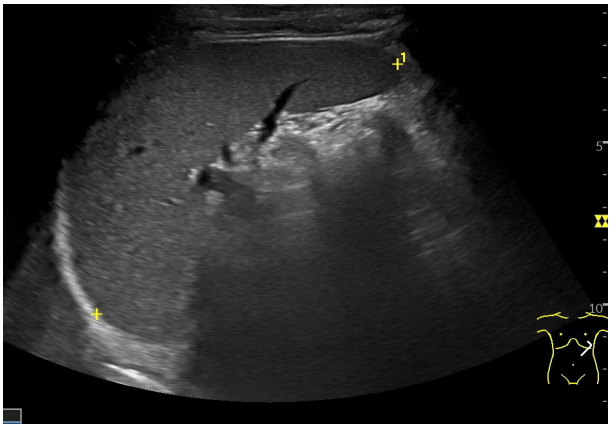
Normal aspect and size of the main biliary duct



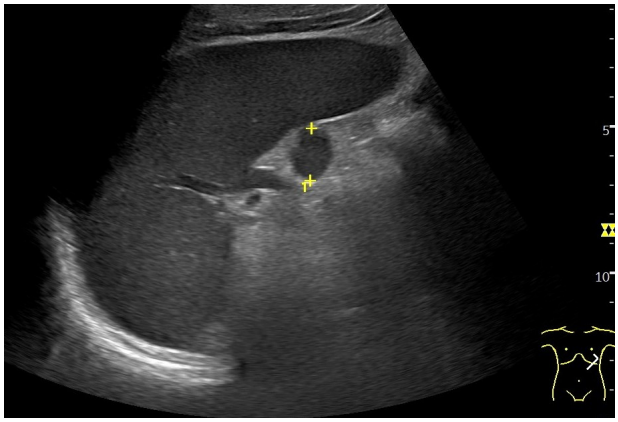
Normal spleen



Main biliary duct (in front of the vena porta)



Measurement of the spleen

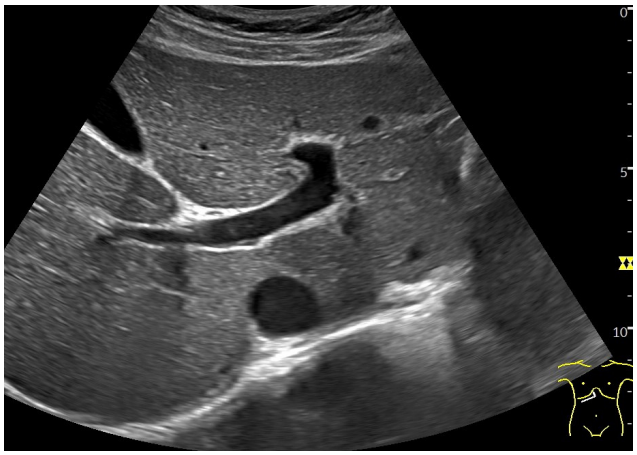


Normal spleen with accessory spleen

Normal parenchyma is relatively homogeneous. The portal branches have conspicuous echogenic walls, while hepatic veins converge toward the inferior vena cava and generally have less prominent walls. The liver can be compared with the adjacent renal cortex (for the appreciation of steatotic liver), when evaluating echogenicity, provided renal disease does not invalidate the comparison.



Porta in the hilum

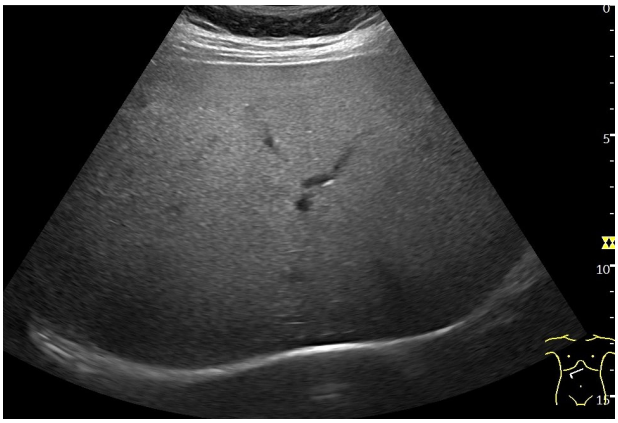


Portal bifurcation

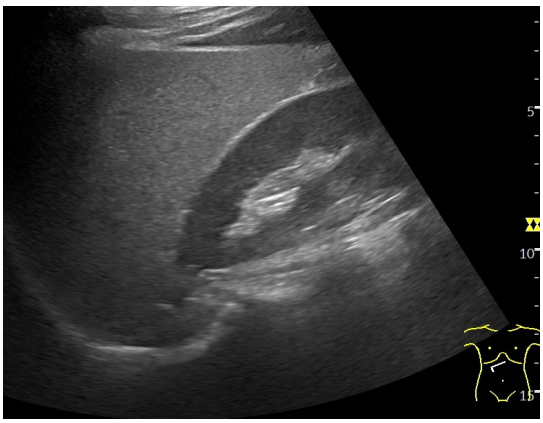
On Doppler examination, normal portal flow is directed toward the liver and is relatively continuous, with respiratory and cardiac variation. Hepatic venous waveforms reflect right atrial pressure changes and usually show phasicity. Hepatic arterial flow has a low-resistance pattern with sustained diastolic flow. Numerical measurements vary with the acquisition method and physiological state and should not be interpreted as universally fixed normal values.

4.3 Diffuse liver disease

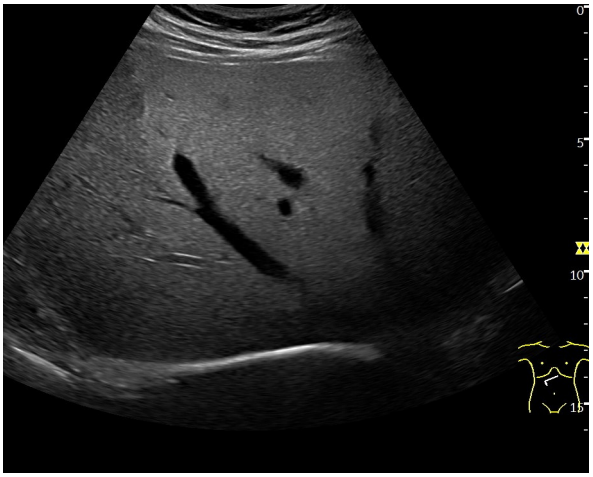
In steatosis, increased parenchymal echogenicity may be accompanied by reduced visibility of vessel walls, greater posterior attenuation, and poor visualization of the diaphragm. These findings are most conspicuous in more marked fat accumulation. Conventional ultrasound is less sensitive for mild steatosis and does not provide a precise, universally transferable measurement of fat fraction.



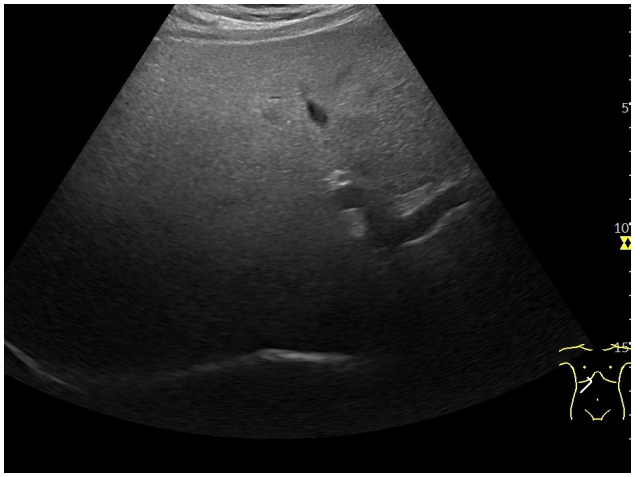
Moderate fatty liver (oblique section)



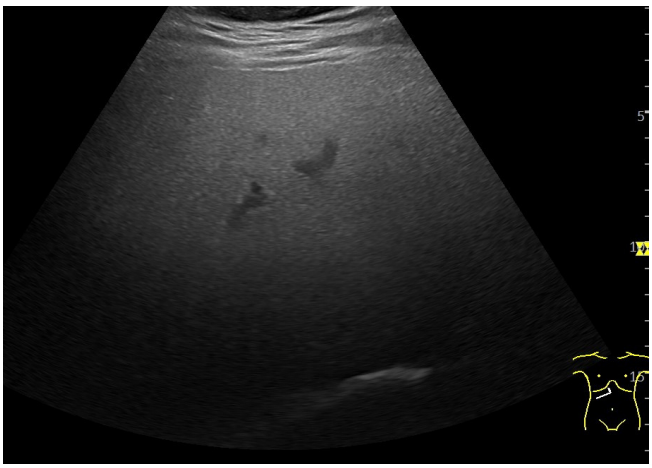
Fatty liver (comparison with the right kidney)



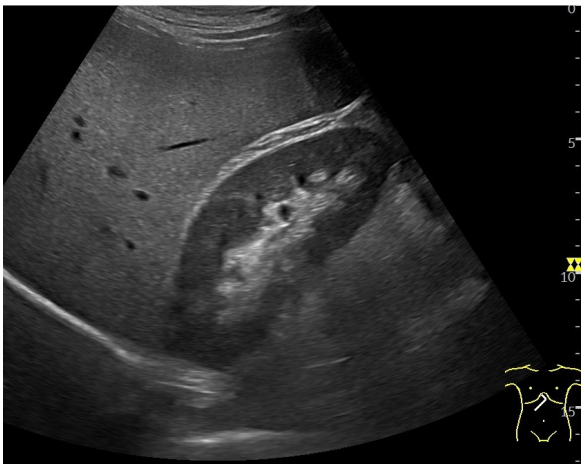
More severe liver steatosis



Severe liver steatosis (severe attenuation)

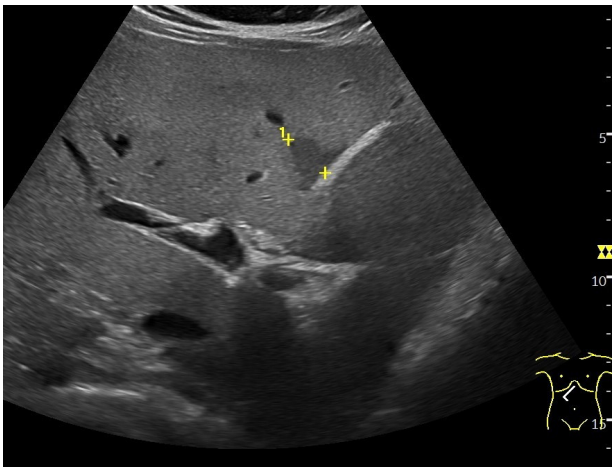


Very severe steatosis (strong posterior attenuation)

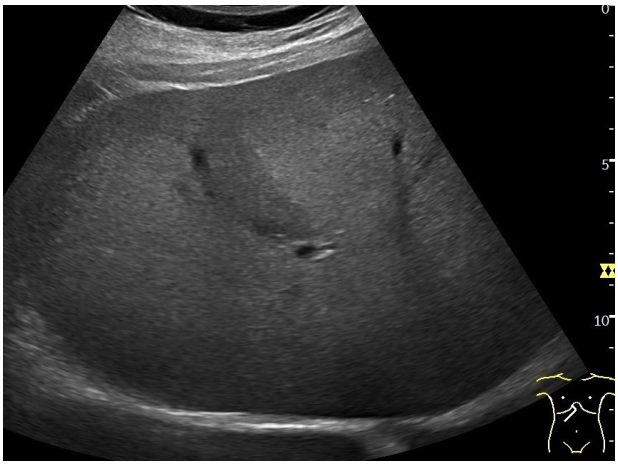


Liver steatosis (increased hepato-renal index)

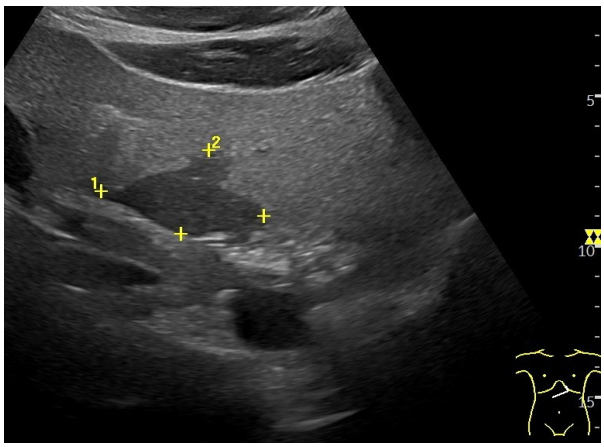
Fibrosis can also alter echotexture, so brightness alone does not establish the mechanism. A diffusely bright liver should be interpreted with the clinical and biochemical context. Focal fatty change or focal sparing may simulate a mass; typical location, lack of mass effect, and preservation of traversing vessels help identify these pseudo lesions. MRI can resolve uncertainty by demonstrating their fat-related signal behavior.



Fatty free area (between the signs)

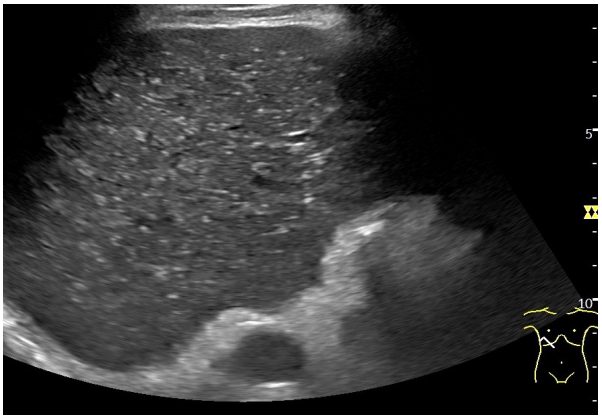


Fatty free area

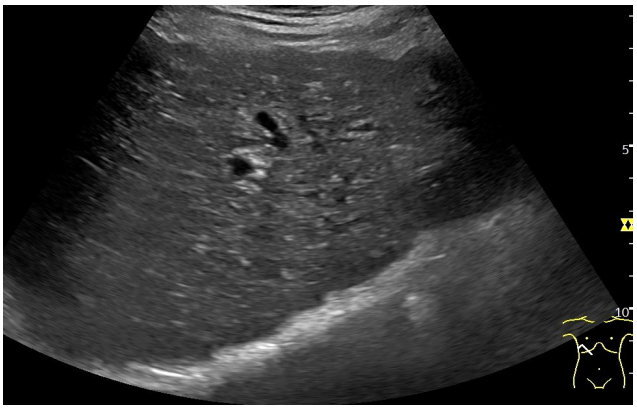


Fatty free area

Advanced cirrhosis may produce surface nodularity, heterogeneous parenchyma, segmental volume changes, and indirect evidence of portal hypertension. The assessment becomes more reliable when several concordant findings are present. A coarse liver alone is insufficient to confidently stage fibrosis, and a smooth contour does not exclude earlier chronic disease.



Cirrhotic liver (heterogenous liver structure)



Cirrhotic liver (heterogenous liver structure)



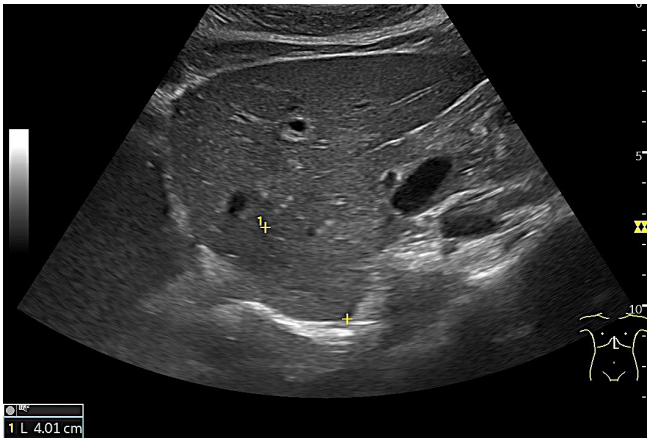
Cirrhotic liver (heterogenous liver structure)



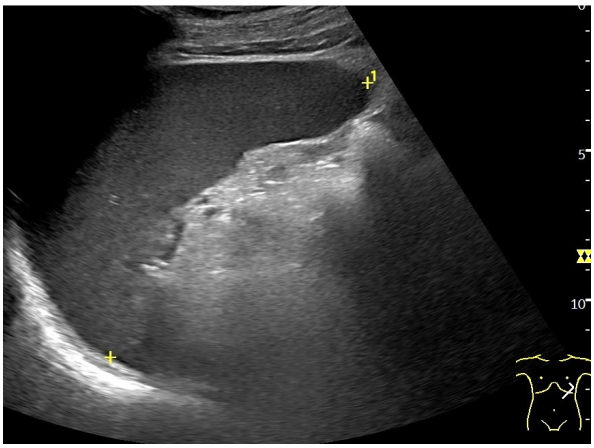
Cirrhotic liver



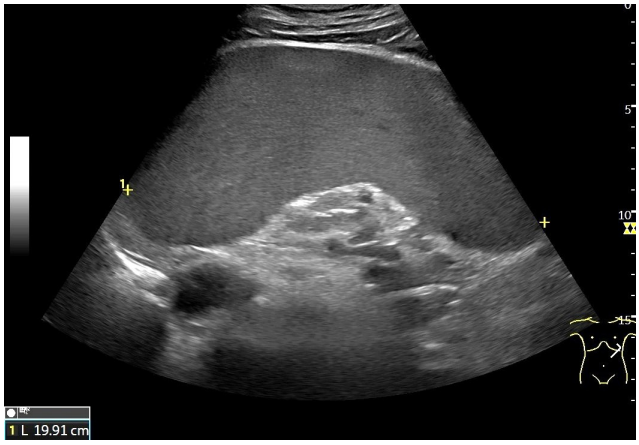
Cirrhotic liver



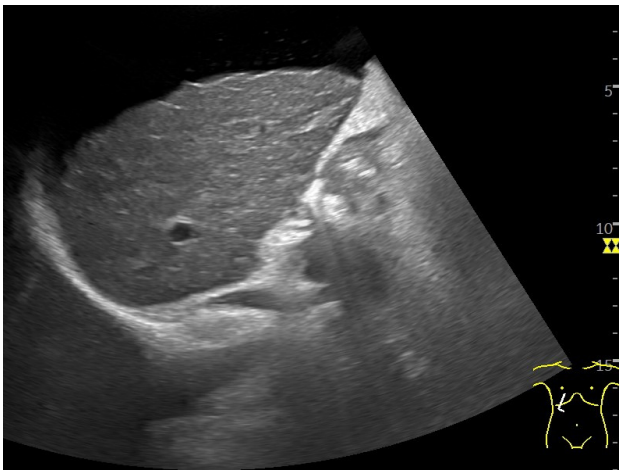
Increased size of caudate lobe (40 mm)



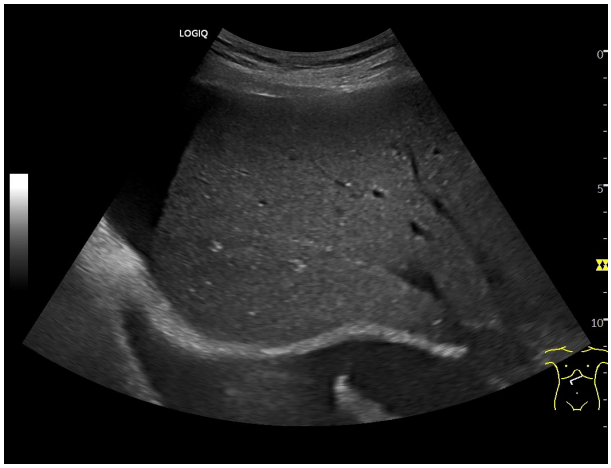
Splenomegaly in a patient with liver cirrhosis



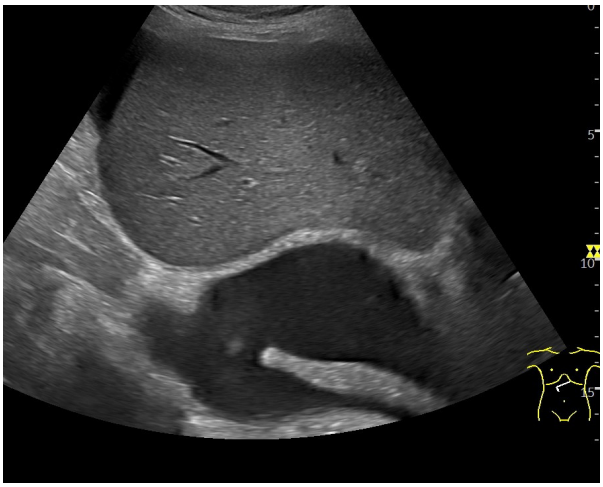
Important splenomegaly



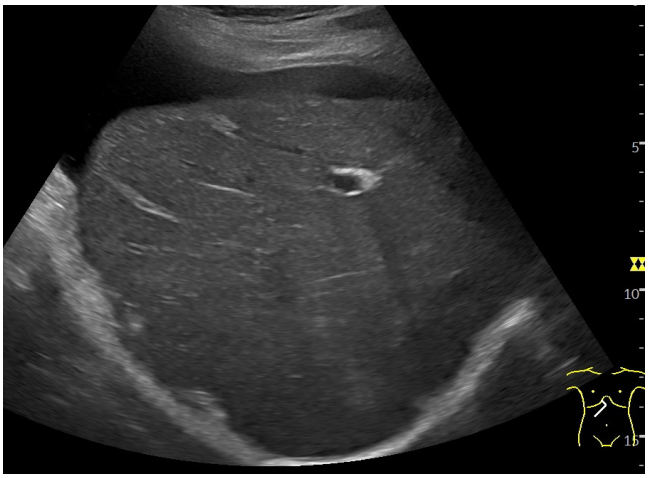
Large amount of perihepatic ascites



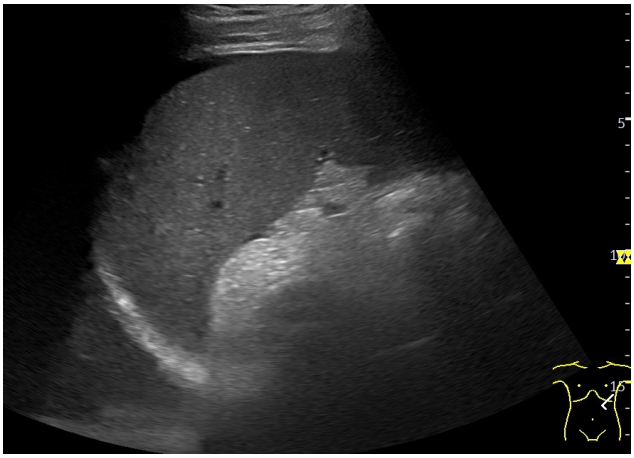
Ascites and pleural effusion



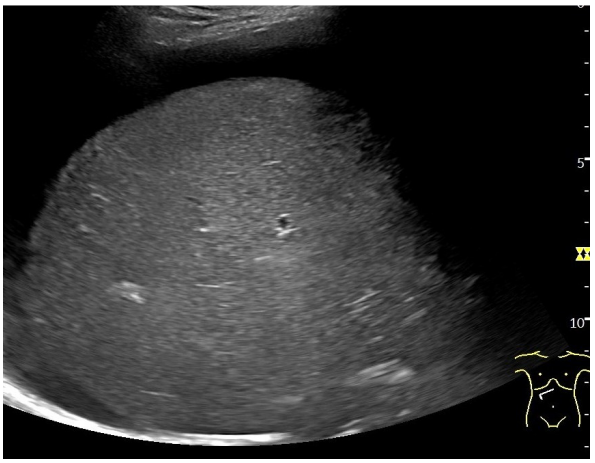
Small ascites and large pleural effusion



Small amount of perihepatic ascites



Small perisplenic ascites

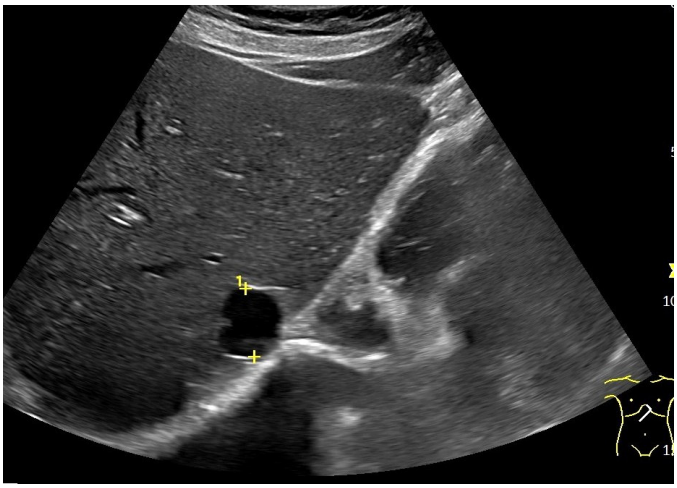


Perihepatic ascites

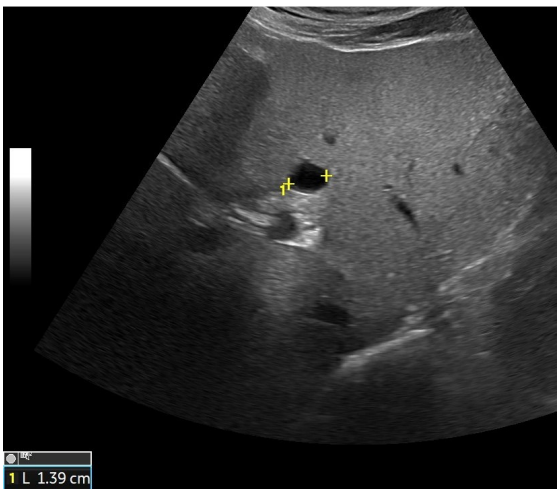
Ultrasound is especially useful for detecting ascites and guiding procedures. It supports surveillance because it is repeatable and widely accessible, but surveillance quality depends on adequate visualization. In patients with a repeatedly limited acoustic window, an alternative strategy may be required according to the risk profile and applicable guidance.

4.4 Focal, cystic, and infectious abnormalities

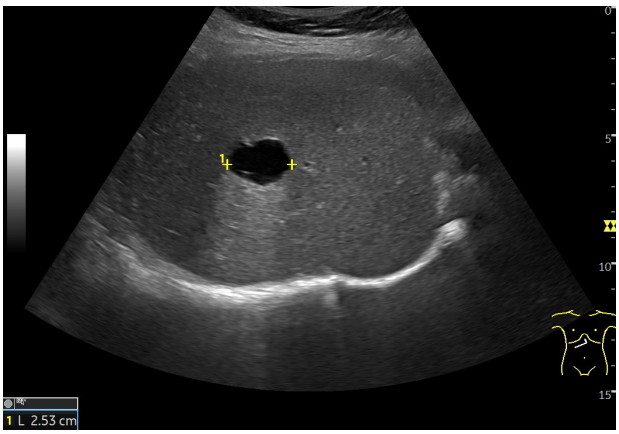
Ultrasound often detects a focal lesion incidentally or during surveillance. Its echogenicity, margins, posterior acoustic behavior, internal structure, and Doppler signal help formulate the differential diagnosis, but many benign and malignant lesions overlap in B-mode appearance.



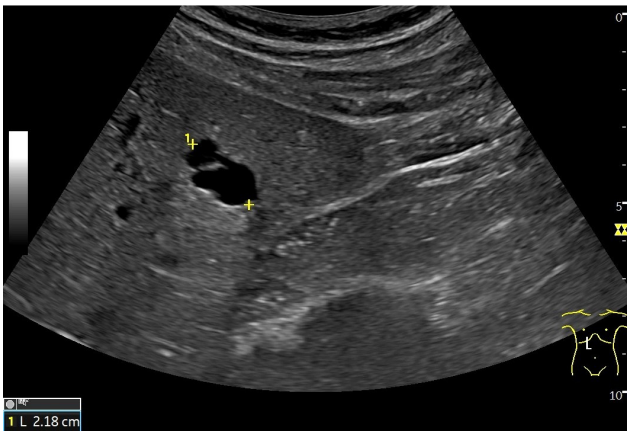
Biliary cyst



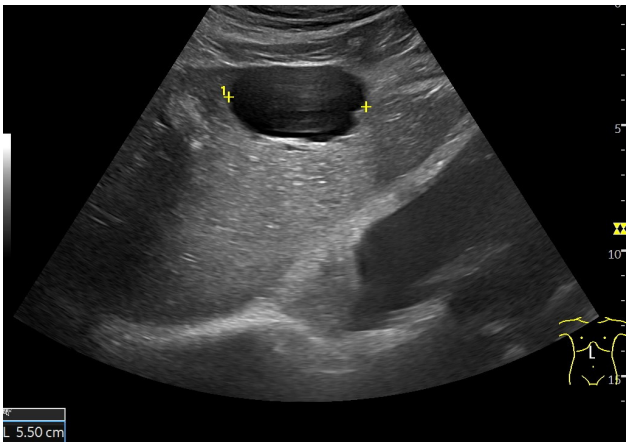
Small biliary cyst



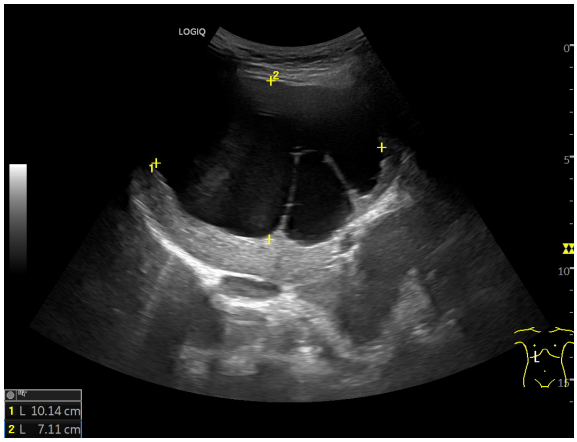
Biliary cyst



Biliary cyst with thin septa

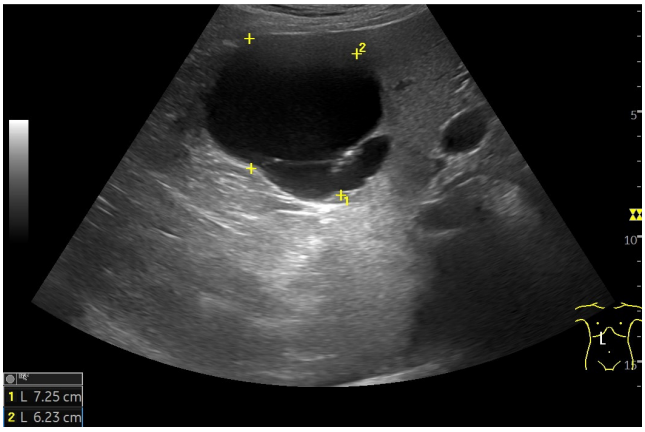


Biliary cyst

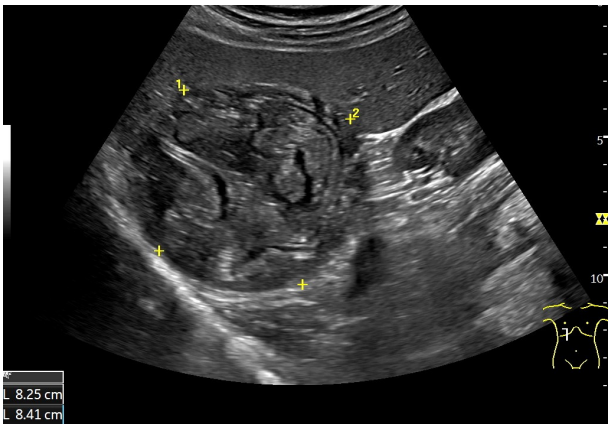


Large biliary cyst with septa

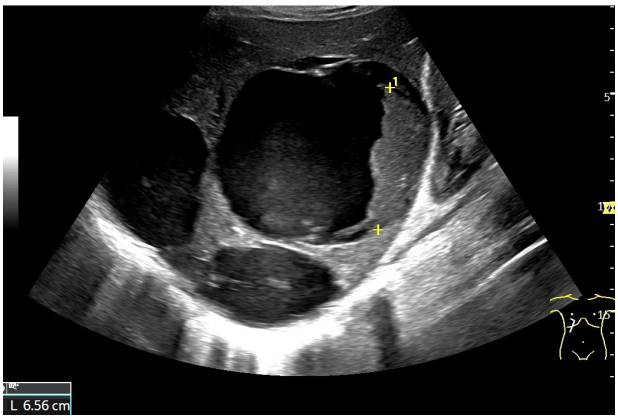
A simple cyst is typically anechoic, sharply defined, and thin-walled, with posterior acoustic enhancement and no internal vascularity. These features allow confident characterization in an appropriate setting. Internal debris, thick or irregular septa, mural nodules, or a solid component indicate a complex lesion requiring further assessment.



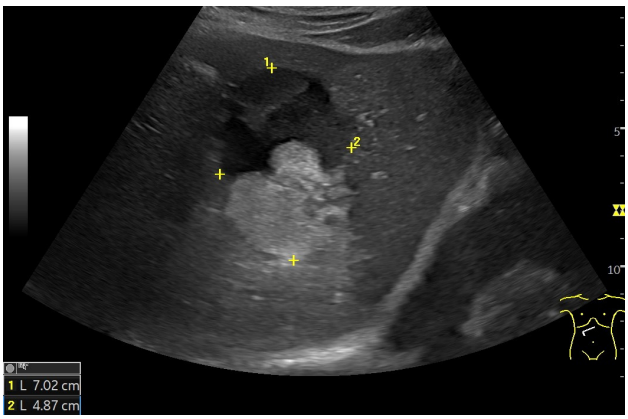
Biliary cyst with thin septa



Hydatic cyst



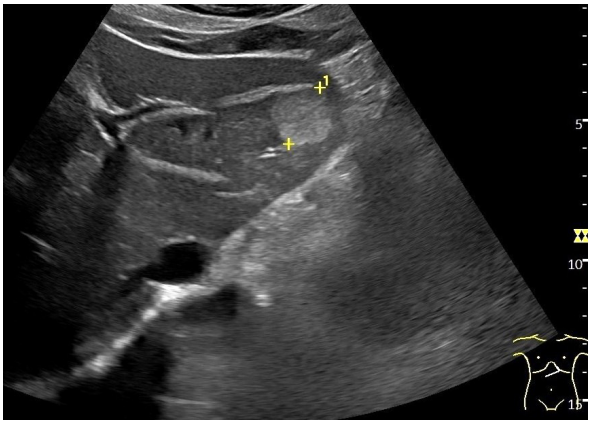
Difficult cystic lesions in the liver



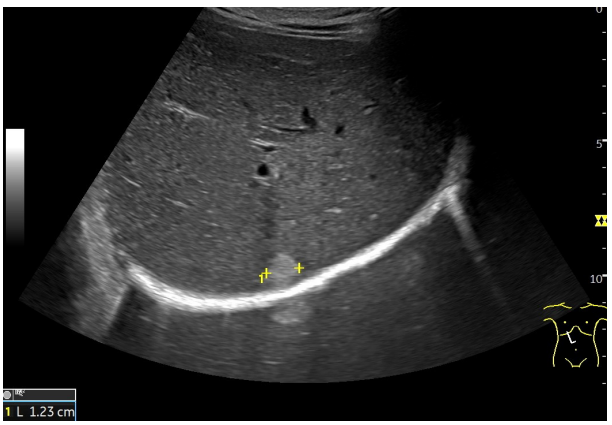
Complex cystic lesion

An abscess can evolve from an ill-defined, relatively solid-appearing focus into a complex collection with liquefied content. The appearance depends on its stage and organism. Ultrasound can guide aspiration or drainage when the lesion is accessible, while CT provides a more comprehensive map of complex infection or associated extrahepatic disease.

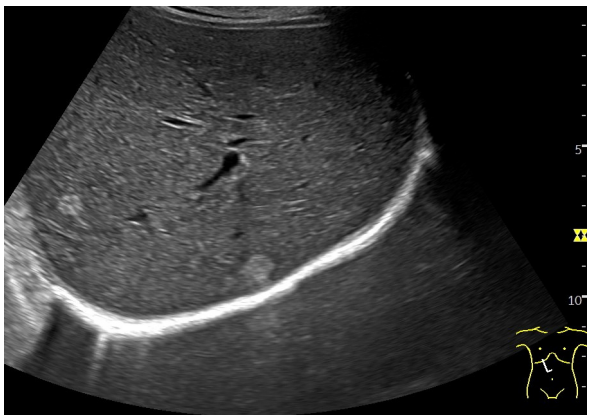
A hemangioma may appear as a well-defined echogenic lesion, but echogenicity is not specific. The significance of the finding changes in a patient with cirrhosis or known malignancy. Additional contrast-based ultrasound (CEUS) should be chosen when the appearance, size, or clinical setting leaves uncertainty. Ultrasound is a sensitive method for detection of focal liver lesions (FLL), but less specific. For this reason, Contrast Enhanced Ultrasound (CEUS) using second generation contrast agents (mainly SonoVue, at least in Europe) is very useful for lesion characterization. Many meta-analyses shown a value of CEUS near the value of CE-CT or CE-MRI.



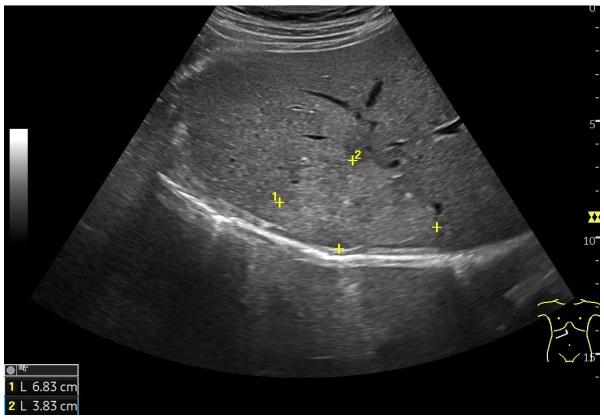
Small hemangioma in the left lobe



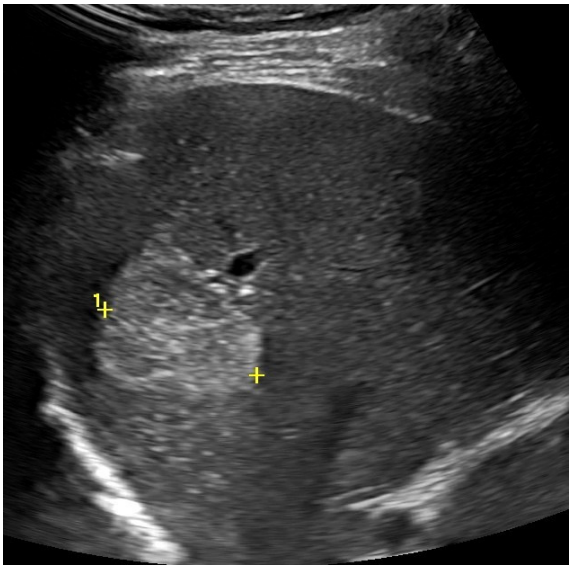
Hemangioma in the segm.7



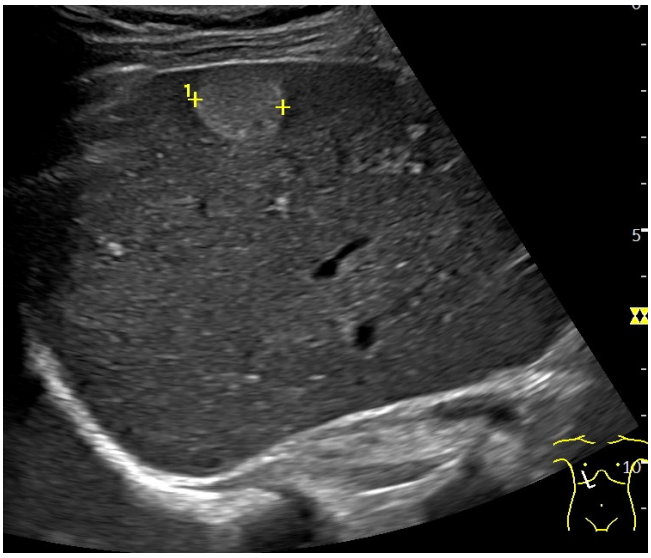
Two small hemangiomas



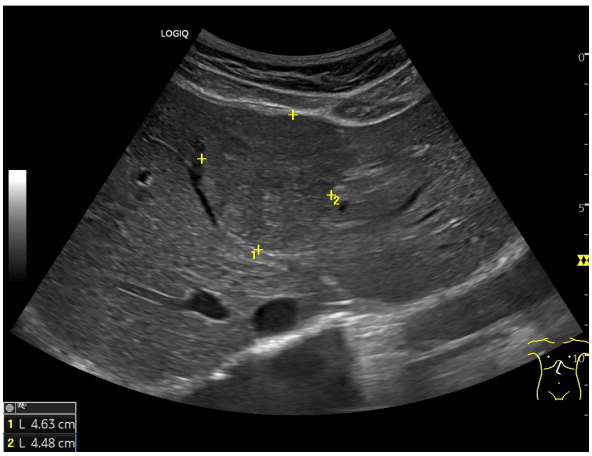
Large hemangioma in segm.7



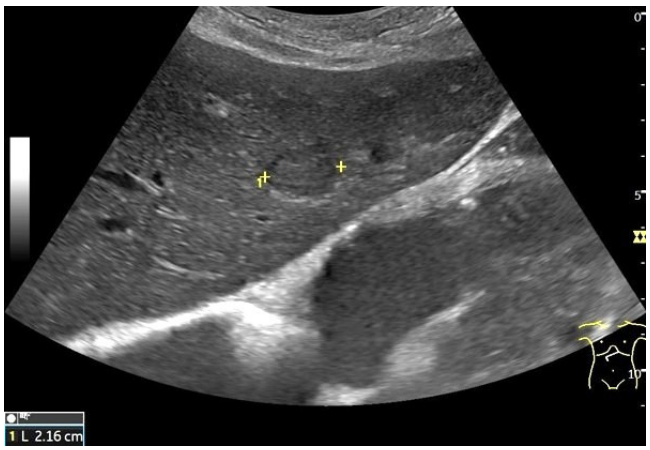
Large hemangioma



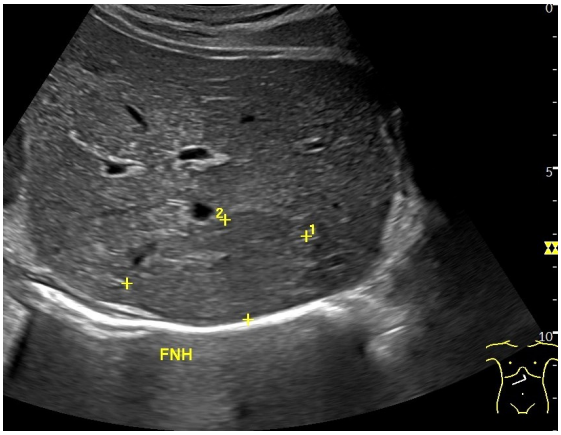
Hemangioma bellow the liver capsule



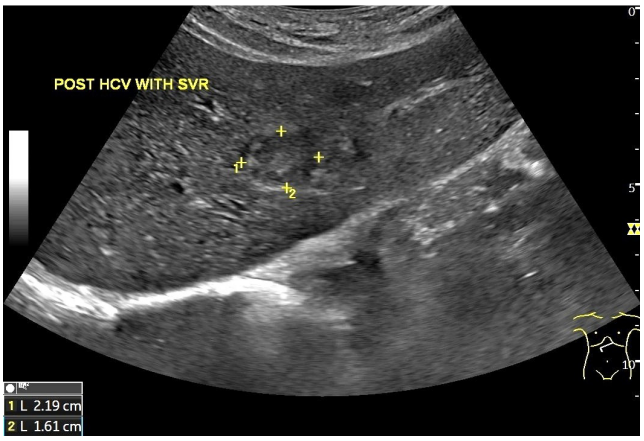
Isoechoic liver lesion (FNH at CEUS)



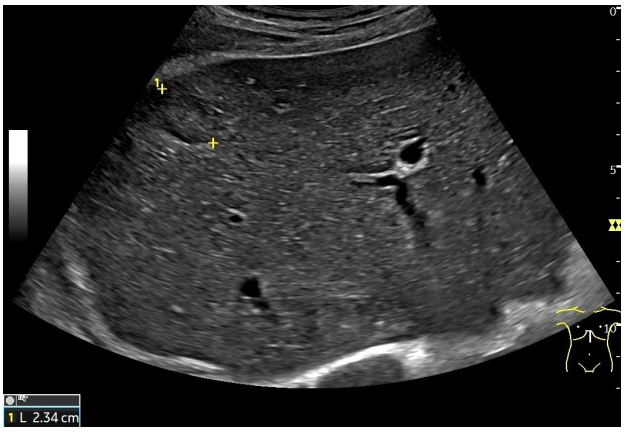
Isoechoic lesion (HCC at CEUS)



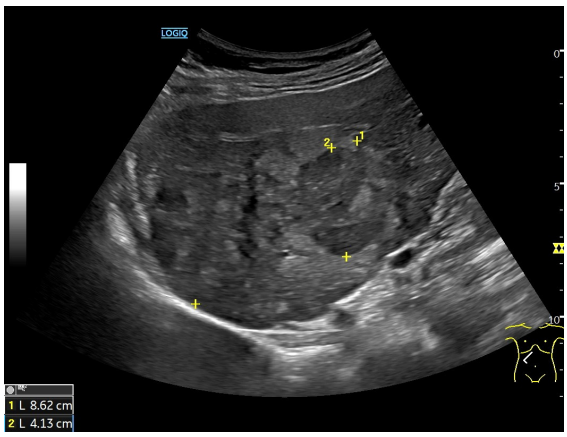
Hypoechoic lesion (FNH at CEUS)



Small HCC after HCV cure



Small subcapsular HCC

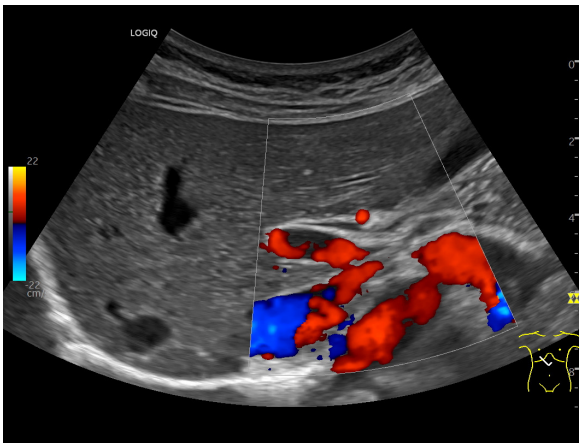


Large HCC (with “nodule in the nodule”)

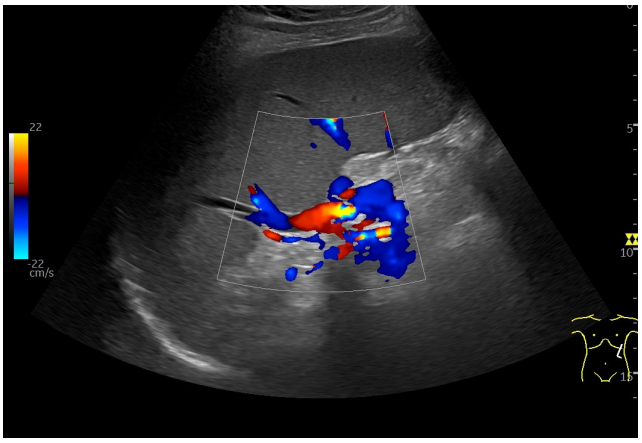
4.5 Doppler principles and measurement

The Doppler effect is the change in frequency of an ultrasound wave reflected by moving blood cells. Its magnitude depends on the transmitted frequency, blood velocity, and the angle between the beam and flow direction. At an angle of 90 degrees, no useful axial Doppler shift is generated. Velocity estimates become progressively less reliable as the angle approaches this value.

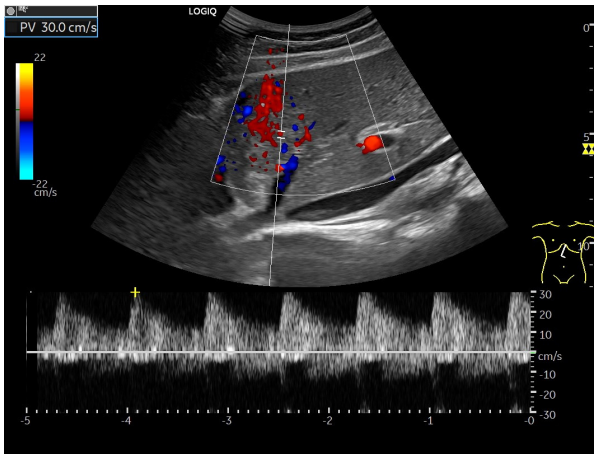
Pulsed-wave Doppler samples flow at a selected depth within a range gate. Spectral Doppler displays the distribution of frequency shifts, usually converted to velocities, over time. Color Doppler overlays directional flow information on the anatomical image. The conventional red and blue assignments indicate motion toward or away from the transducer according to the selected color map; they do not identify artery versus vein or inherently establish the anatomical direction of flow.



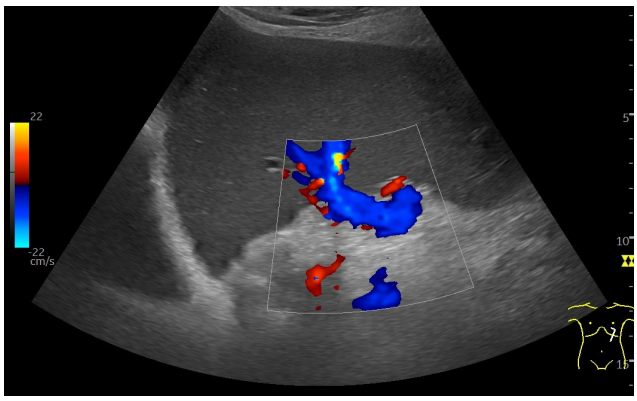
Color Doppler



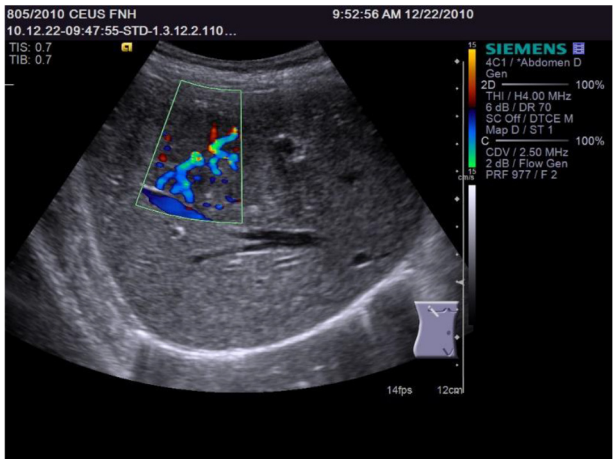
Color Doppler of the splenic hilum (portal hypertension)



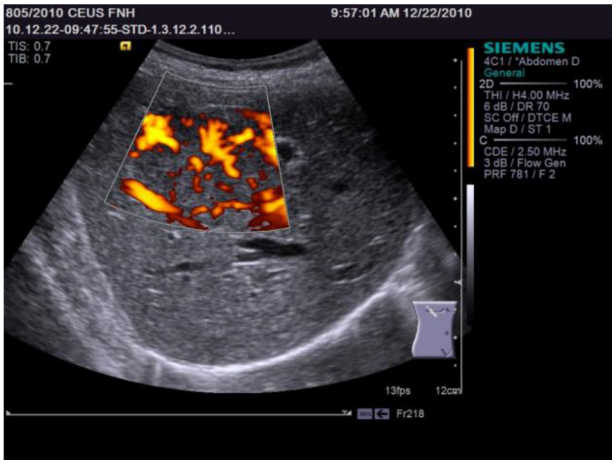
Doppler in a patient with liver FNH



Color Doppler of the spleen in portal hypertension



Color Doppler in a case of liver FNH



Power Doppler in a case of liver FNH

The size and placement of the sample volume affect the waveform. A poorly positioned gate can miss slow flow or capture adjacent vessels. Respiratory or pulsatile vessel movement can intermittently displace the lumen from the sampling region and produce a misleading trace. For velocity measurements, the correction cursor should follow the actual flow direction, with a suitable insonation angle, commonly no greater than about 60 degrees when feasible.

Pulsed Doppler is limited by the Nyquist frequency, equal to half the pulse repetition frequency. Shifts above this limit are displayed as aliasing. Adjusting the scale, transmitted frequency, baseline, or acquisition approach can improve interpretation, but a baseline shift alone does not remove the underlying sampling limit. Inappropriate settings can make aliasing resemble reversal of flow.

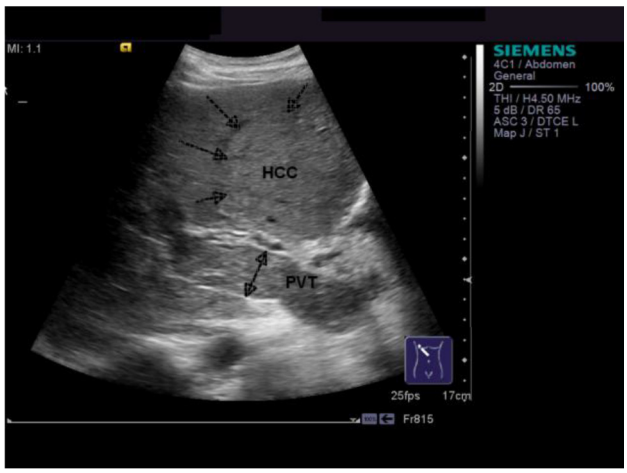
Wall filters suppress low-frequency signals from vessel motion. If set too high, they also remove genuine slow venous flow. The absence of color or spectral signal therefore does not establish thrombosis until gain, scale, filter, angle, and acoustic access have been optimized. This is particularly important in the portal system.

Measurements of peak velocity, mean velocity, and volumetric flow are not interchangeable. Flow calculations require reliable cross-sectional area and appropriate velocity averaging. Errors in vessel diameter and assumptions about the flow profile can materially affect the estimated volume.

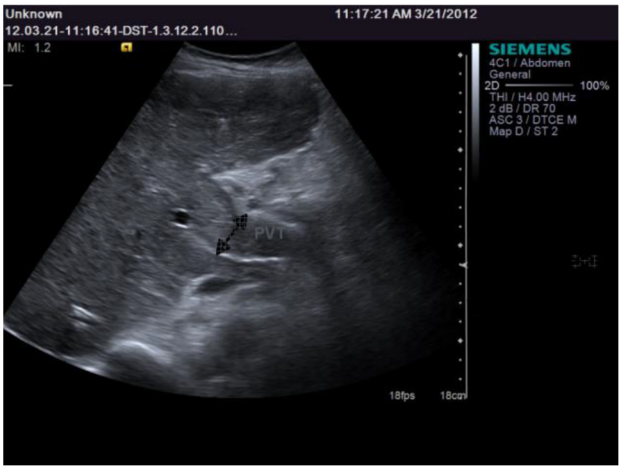
4.6 Vascular applications

Doppler ultrasound provides an initial assessment of portal vein patency and direction of flow. Portal hypertension may be associated with slow or reversed flow and collateral vessels, but isolated velocity or diameter findings are insufficient to establish its severity. The complete picture includes the liver, spleen, ascites, collaterals, and clinical findings.

Acute portal thrombosis may be difficult to see on grayscale imaging because the thrombus can be relatively hypoechoic. Chronic thrombosis may produce contraction of the native vein and cavernous transformation. Suspected thrombus should be confirmed through optimized flow assessment and, when necessary, contrast-enhanced cross-sectional imaging. Internal enhancement or arterialized flow can raise concern for tumor thrombus, interpreted with the surrounding tumor and vascular findings.



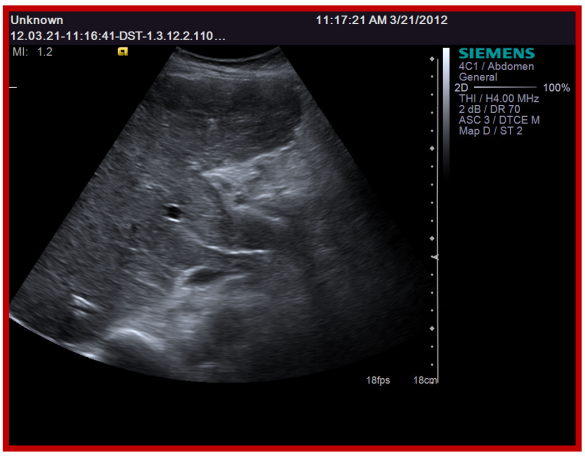
Portal thrombosis in a case of HCC



Hilar portal thrombosis



Thrombosis of the portal bifurcation



Thrombosis of the hilar porta

In Budd–Chiari syndrome, abnormal hepatic venous patency and flow, intrahepatic collateral channels, and changes in the inferior vena cava may be demonstrated. Non-visualization of a vein is not by itself sufficient, particularly if the examination is technically limited or cirrhosis is present. CT or MRI can establish the extent and associated parenchymal remodeling.

Hepatic venous waveform abnormalities can also occur in cardiac disease, congestion, and altered liver compliance. Their significance depends on the pattern and clinical setting. Doppler is particularly valuable because it measures dynamic information that static anatomical images do not directly provide.

Portal venous gas may appear as moving echogenic foci, while pneumobilia produces echogenic structures and associated artifacts in the biliary system. Their significance depends on the cause and accompanying findings. Portal venous gas can occur in severe bowel disease, but also in other settings, including after procedures; it should not be treated as an automatic diagnosis of intestinal necrosis.

4.7 Artifacts and limitations

Reverberation produces repeated echoes from strongly reflective interfaces. Mirror-image artifacts, particularly near the diaphragm, may duplicate hepatic structures in an apparent location beyond the reflecting surface. Refraction, side-lobe effects, acoustic shadowing, and enhancement can also alter the appearance of lesions. Some artifacts assist diagnosis, while others simulate disease.

The major practical limitations are operator dependence, restricted acoustic access, attenuation in obesity or severe steatosis, and interference from bowel gas. Deep posterior or subdiaphragmatic lesions may be difficult to assess. A satisfactory view of part of the liver does not guarantee a satisfactory evaluation of the whole organ. Good training and enough experience can overcome many of these limitations. Good ultrasound machines and good settings help a lot to improve the quality of the ultrasound examination. New high tech ultrasound systems use a lot of Artificial Intelligence (AI) tools to help practitioners obtain the best examinations.

Conventional ultrasound has limited ability to stage early fibrosis or definitively characterize many focal masses. Its role is strengthened by Doppler, elastography, and CEUS, but these additions answer specific questions and do not remove every anatomical limitation. Their use should follow the unresolved diagnostic need.

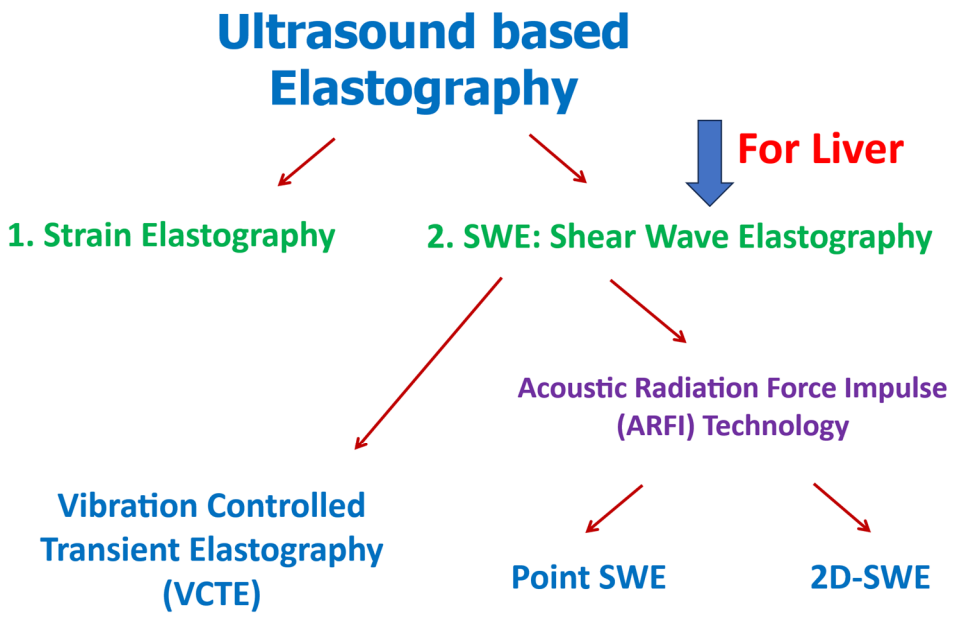
Chapter 5. Elastography, Fatty Quantification and Contrast-Enhanced Ultrasound

5.1 Mechanical tissue assessment

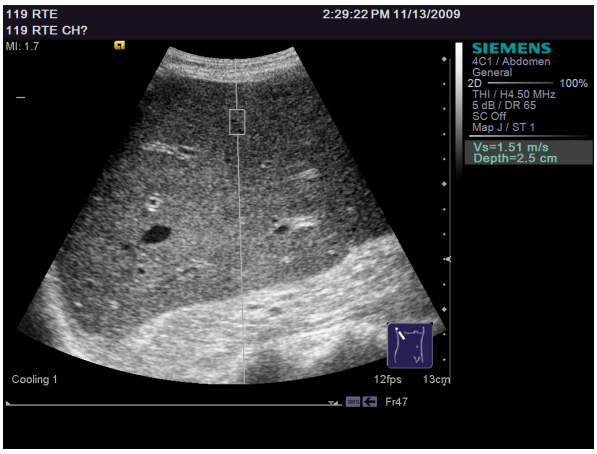
Elastography measures tissue response to mechanical stimulation. In shear-wave techniques, the propagation of a mechanical wave is assessed within the liver. Greater stiffness is generally associated with faster propagation, and results may be expressed in meters per second or converted to kilopascals using technique-dependent assumptions.

Liver stiffness is an indirect marker of fibrosis. It is not a direct measurement of collagen content and is not determined exclusively by fibrosis. Acute inflammation, cholestasis, congestion, food intake, and technical conditions can influence the result. The value should therefore be interpreted within a defined clinical setting and with method-specific quality criteria.

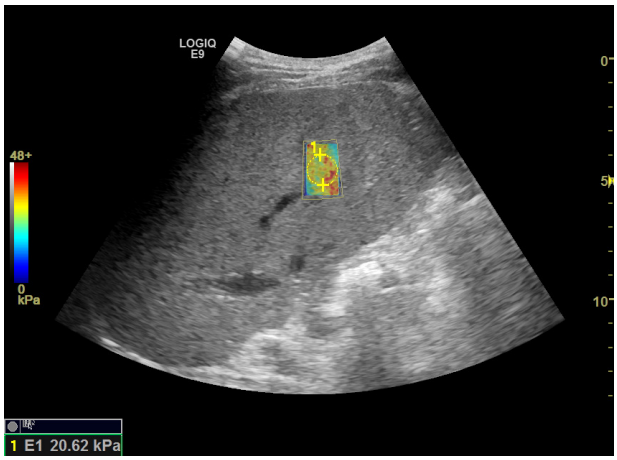
Vibration-controlled transient elastography (VCTE) uses a dedicated device and offers a rapid examination. Point shear-wave and two-dimensional shear-wave methods using Acoustic Radiation Force Impulse (ARFI) can be integrated into ultrasound equipment, allowing anatomical targeting of the measurement region. The techniques differ in excitation, sampling volume, output, and validated thresholds, so their measurements should not be assumed to be interchangeable.



Classification of ultrasound based elastography



pSWE (point shear wave elastography)



2D-SWE (numeric and color coded)

Magnetic resonance elastography assesses stiffness over a larger region and is discussed further in Chapter 7. Both ultrasound-based and MR methods support fibrosis risk assessment, but the choice depends on availability, technical reliability, patient characteristics, and the need for additional anatomical information.

Table 2. Advantages and disadvantages of the main non-invasive tests used to diagnose and stage liver fibrosis.

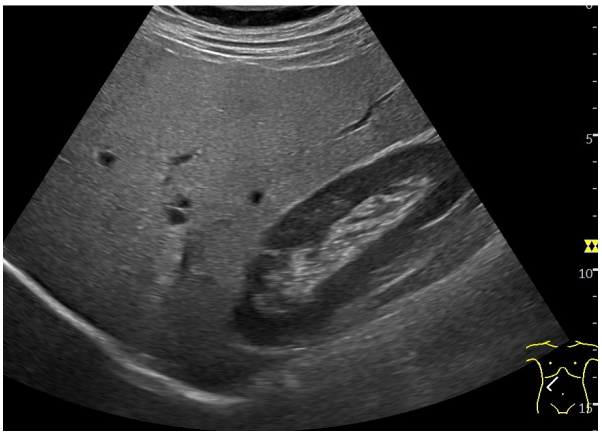
	Serum markers		Transient elastography	pSWE	2D-SWE	MRE
Advantages	Non-patented • Good reproducibility • High applicability (95%) • No cost and wide availability • Well validated • Can be performed in the outpatient clinic • Prognostic value of some has been validated for some aetiologies of chronic liver disease on population level	Patented Good reproducibility • High applicability (95%) • Well validated • Can be performed in the outpatient clinic • Prognostic value of some has been validated for some aetiologies of chronic liver disease	• Most widely used and validated technique • Point-of-care (bedside; rapid, easy to learn) • Quality criteria well defined • Good reproducibility • High performance for cirrhosis (AUROC >0.9) • Prognostic value in compensated cirrhosis well validated	• Can be performed in combination with regular ultrasound if the device is provided with adequate software • ROI smaller than TE and location chosen by the operator • Higher applicability than TE (ascites and obesity) • Performance equivalent to that of TE for advancedfibrosis and cirrhosis • Prognostic value in cirrhosis • High applicability for spleen stiffness measurement	• Can be performed in combination with regular ultrasound if the device is provided with adequate software • Large ROI that can be adjusted in size and location chosen by the operator • Measures liver stiffness in real time • Good applicability • High performance for the diagnosis of significant fibrosis and cirrhosis • Prognostic value in compensated cirrhosis	• Can be implemented on a regular MRI machine • Examination of the whole liver • Higher applicability than TE (ascites and obesity) • High performance for the earlier fibrosis stage and for diagnosis of cirrhosis
Disadvantages	• Non-liver-specific • Performance not as good as TE and patented serum markers • False positive results with FIB-4 and NFS in case of age>65 yrs	• Cost • Non-liver-specific • Performance not as good as TE for cirrhosis • False positive results in case of extrahepatic inflammatory conditions, profibrotic, extrahepatic disease and other (*e.g.* haemolysis, Gilbert syndrome)	• Requires a dedicated device • ROI cannot be chosen • Applicability (>95%) lower than serum biomarker: (obesity, ascites, operator experience) • False positive in case of acute hepatitis, extrahepatic cholestasis, liver congestion, food intake and excessive alcohol intake	• False positive in case of acute hepatitis, extrahepatic cholestasis, liver congestion, food intake and excessive alcohol intake	• False positive in case of acute hepatitis, extrahepatic cholestasis, liver congestion, food intake and excessive alcohol intake	• Not applicable in case of iron overload • Requires a MRI facility • Time consuming • Costly • No clear data on prognostic value

2D-SWE, bidimensional shear wave elastography; FIB-4, fibrosis-4; MRE, magnetic resonance elastography; MRI, magnetic resonance imaging; NFS, NAFLD fibrosis score; pSWE, point-shear wave elastography; ROI, region of interest; TE, transient elastography.

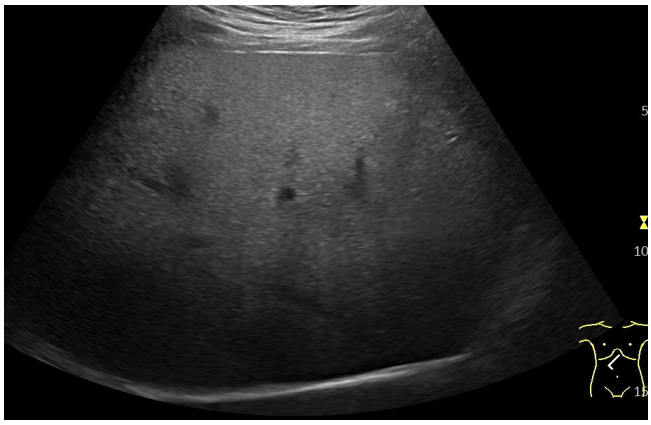
EASL Clinical Practice Guidelines on non-invasive tests for evaluation of liver disease severity and prognosis, 2021 update. J Hepatol. 2021 Sep;75(3):659-689.

5.2 Fatty Quantification

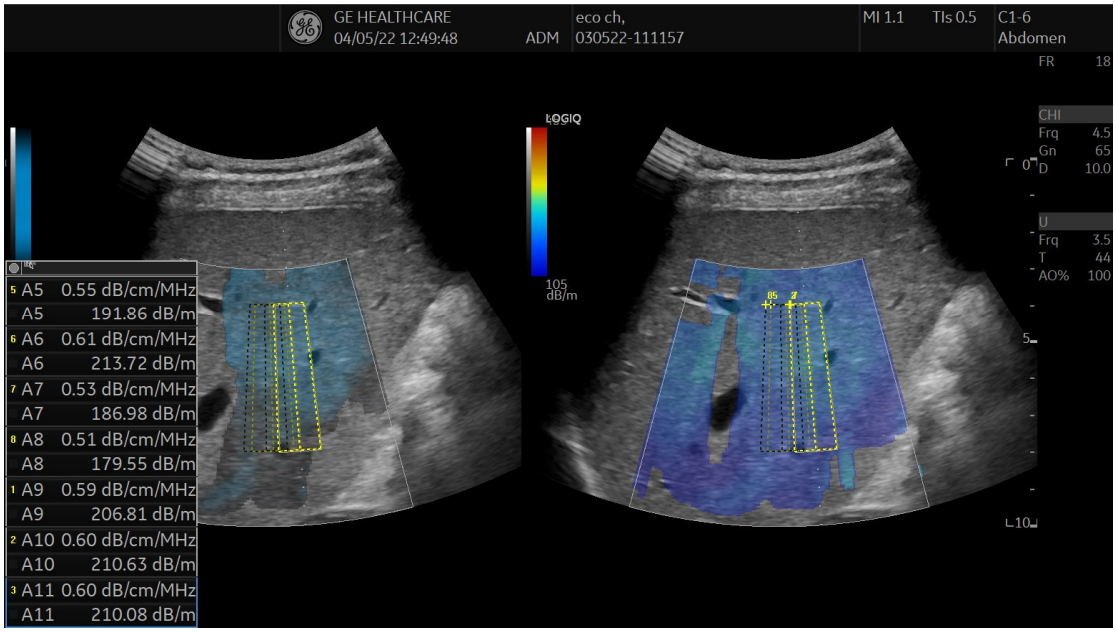
Ultrasound is a very good method for the quantification of the fat in the liver. Standard ultrasound is a subjective method. Quantitative Ultrasound (QUS) uses attenuation, backscattering or speed of sound (or combination of them) for more precise and quantitative estimation of liver fat. Examination is quick and the result is expressed in dB/cm/MHz or in dB/m. More recently some ultrasound systems express the fat in % (making easier the interpretation of the results). Usually, ten measurements are performed and then the median value of them express the severity of the liver steatosis. This evaluation is very useful in patients with MASLD (metabolic associated steatotic liver disease).



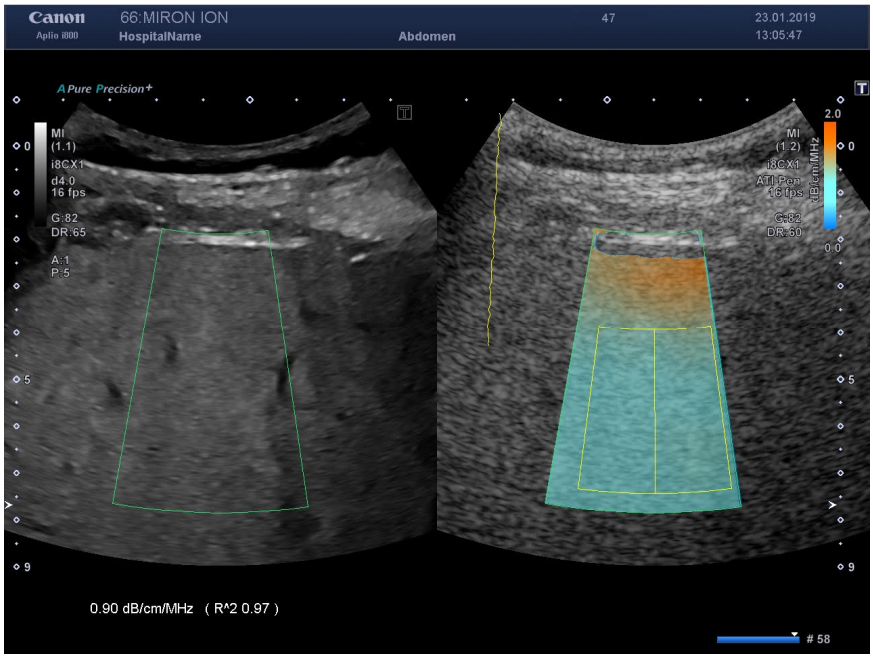
Typically steatotic liver in standard US



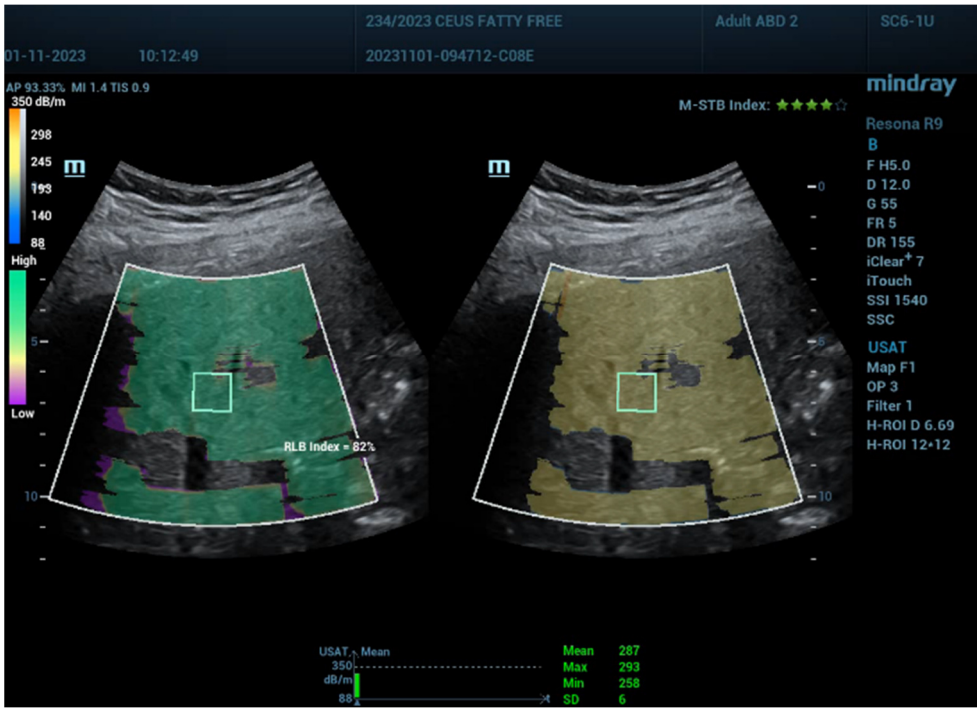
“Bright liver” with posterior attenuation



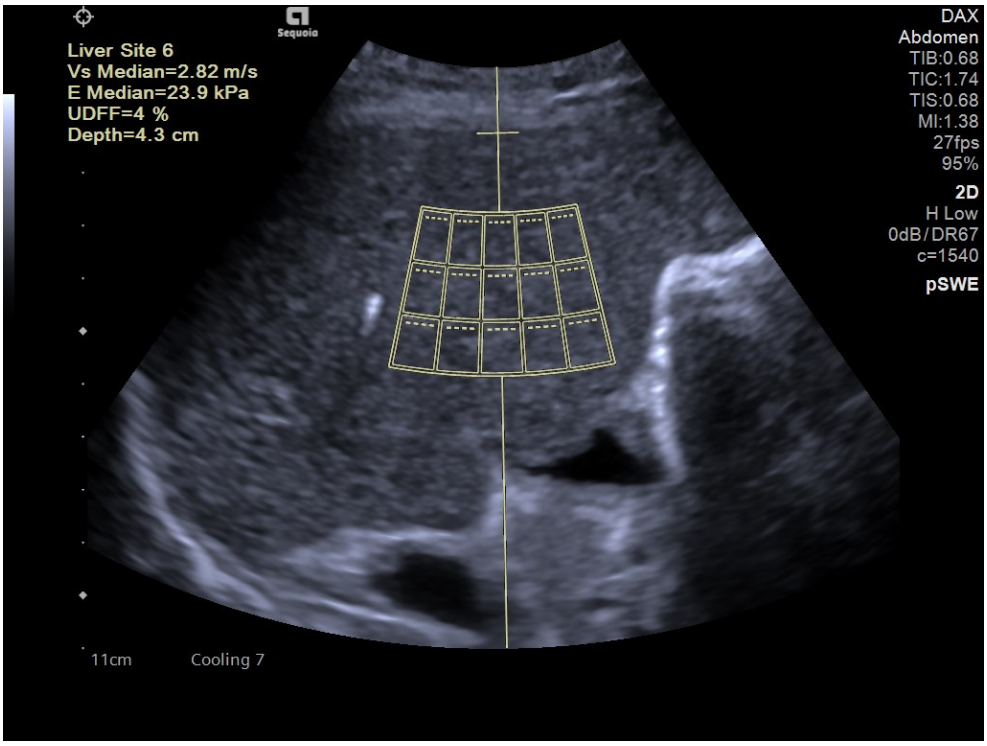
UGAP system for the quantification of liver fat



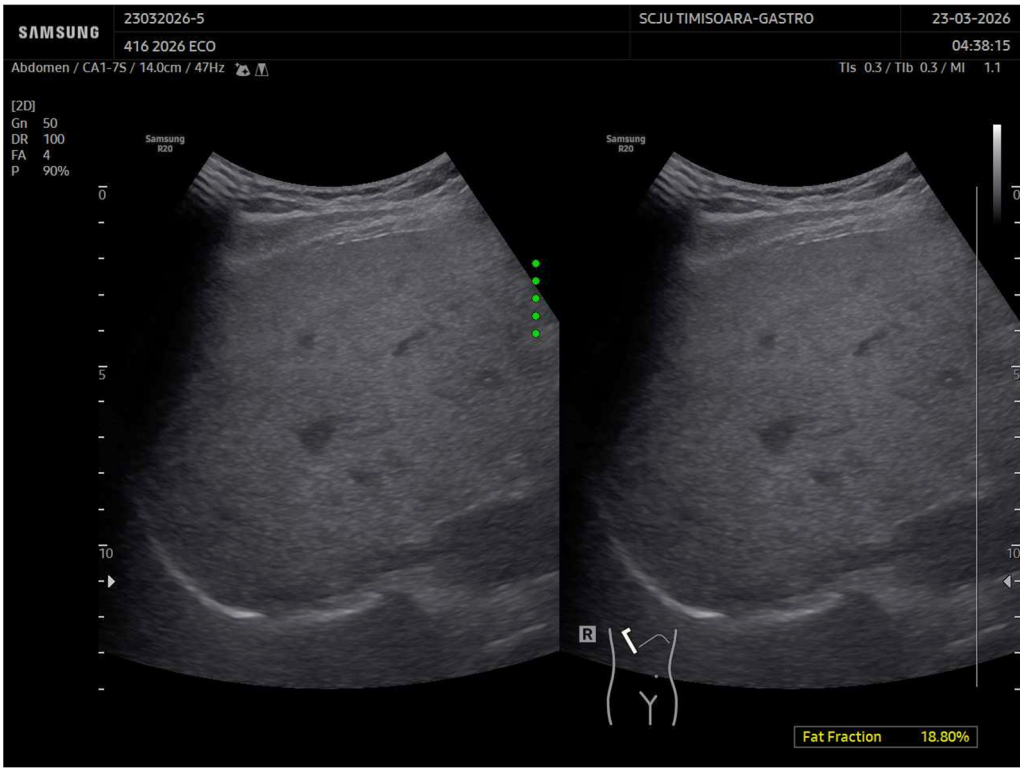
ATI for fatty quantification



USAT system for liver fat quantification



UDFF with results expressed in %



AI guided fat quantification (Deep USFF)

5.3 Clinical application and longitudinal interpretation

Elastography is valuable in chronic viral hepatitis, metabolic steatotic disease, alcohol-related disease, and other chronic conditions in which fibrosis affects prognosis. It supports identification of patients unlikely to have advanced fibrosis and those requiring further evaluation. In many pathways it is combined with blood-based risk scores rather than used in isolation.

A high-quality measurement requires appropriate preparation (fasting for 3-4 hours), a suitable sampling region, and attention to the reliability indicators specific to the technique (IQR/Median<30%). Measurements should avoid large vessels, focal lesions, and obvious artifacts. Failure to obtain a reliable measurement is a technical outcome that must be reported, rather than converted into an apparently precise clinical conclusion.

Serial measurements are useful when the technique and acquisition conditions remain reasonably comparable. A change may reflect fibrosis, inflammation, congestion, or their combination. An early reduction after treatment should not automatically be described as histological regression, just as an increase during acute hepatitis should not automatically be labeled progression to cirrhosis.

Stiffness can contribute to the assessment of portal hypertensive risk when used within validated algorithms. Stiffness for portal hypertension can be provided in liver and spleen. It does not replace every endoscopic or hemodynamic examination. Thresholds depend on etiology, clinical setting, and the particular measurement system; a single universal cutoff cannot serve all purposes.

5.4 Principles of Contrast-Enhanced Ultrasound

CEUS uses intravenously administered microbubbles to increase the ultrasound signal from the circulation. Contrast-specific imaging, generally at a low mechanical index, enables observation of enhancement while limiting premature bubble destruction. Most vascular-phase agents remain within the blood pool; selected

agents also have a later Kupffer-cell phase. Their behavior must be interpreted according to the product and protocol.

A defining advantage is continuous observation of contrast arrival and subsequent changes. CT and MRI acquire selected phases at predetermined moments, whereas CEUS can directly observe the arterial enhancement pattern and the onset and degree of washout. This temporal detail is useful when another examination has missed the optimal arterial phase.

CEUS assesses a target lesion in relation to the surrounding liver. It is particularly valuable when the lesion is visible and accessible on ultrasound. Its field of view and acoustic constraints mean that it is generally not a substitute for comprehensive whole-body staging or a complete cross-sectional map of numerous lesions.

Microbubble agents do not share the renal toxicity profile of iodinated or gadolinium-based agents. They can therefore offer a useful alternative for selected patients with impaired renal function. Adverse reactions remain possible, and use must follow the product's contraindications, local approvals, and an appropriate response protocol.

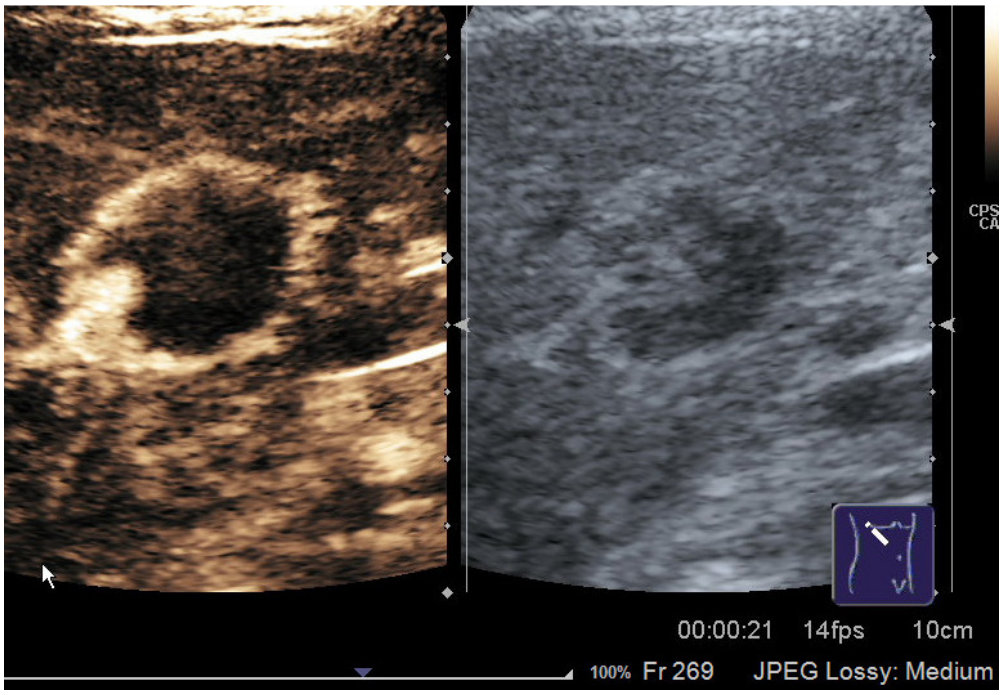
5.5 Enhancement patterns of focal lesions

A typical hemangioma shows peripheral discontinuous nodular enhancement with progressive centripetal filling. The speed and completeness of filling vary with lesion size and vascular structure. A characteristic pattern can establish the diagnosis, but an atypical lesion should not be forced into a benign category on the basis of echogenicity alone.

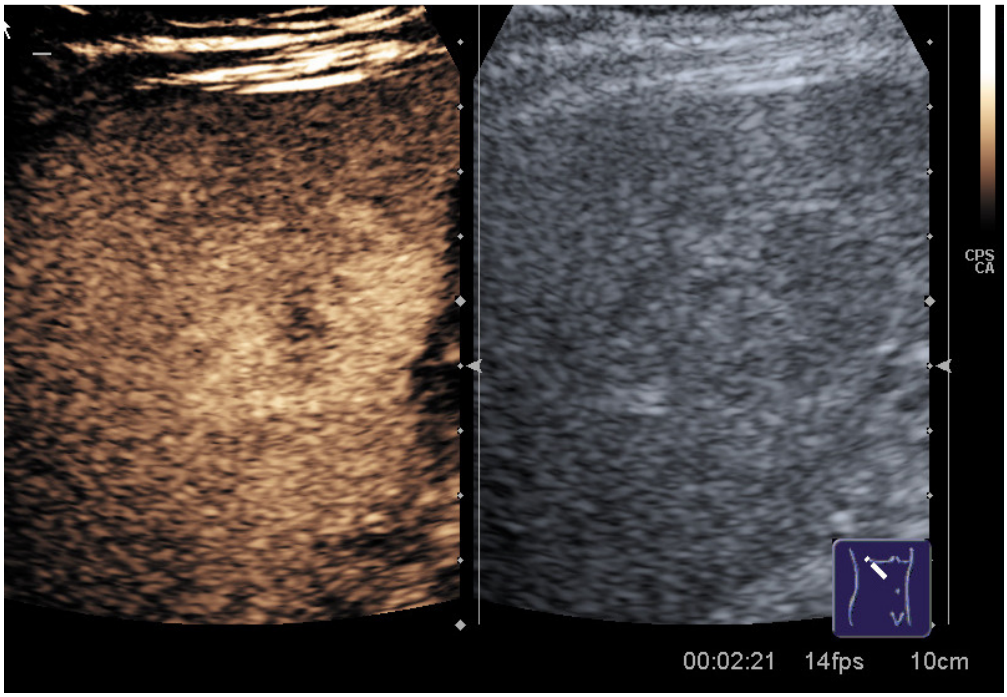
Focal nodular hyperplasia frequently shows rapid arterial enhancement, sometimes with a centrifugal or spoke-wheel vascular pattern, and sustained enhancement thereafter. Hepatocellular adenoma may show arterial enhancement but more variable later behavior. CEUS can assist the distinction, although MRI with hepatobiliary contrast often provides additional tissue-specific information.

Many malignant lesions demonstrate washout, meaning that the lesion becomes less enhanced than the surrounding liver over time. The timing and degree matter. In the appropriate high-risk setting, HCC may show non-rim arterial hyperenhancement and late, mild washout. Early or marked washout and rim enhancement raise concern for other malignancies or non-HCC malignant patterns. These features require a structured interpretation rather than a simple rule that all washout equals HCC.

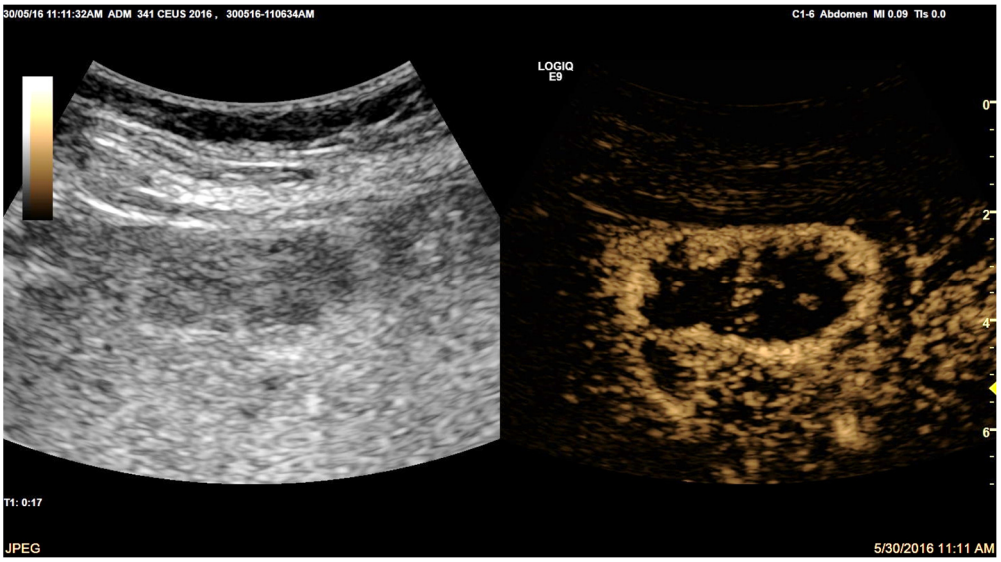
CEUS LI-RADS provides a standardized framework for eligible high-risk patients. It is distinct from CT/MRI LI-RADS because the contrast behavior and acquisition differ. A category should only be assigned when the patient and examination satisfy the framework's scope and technical requirements.



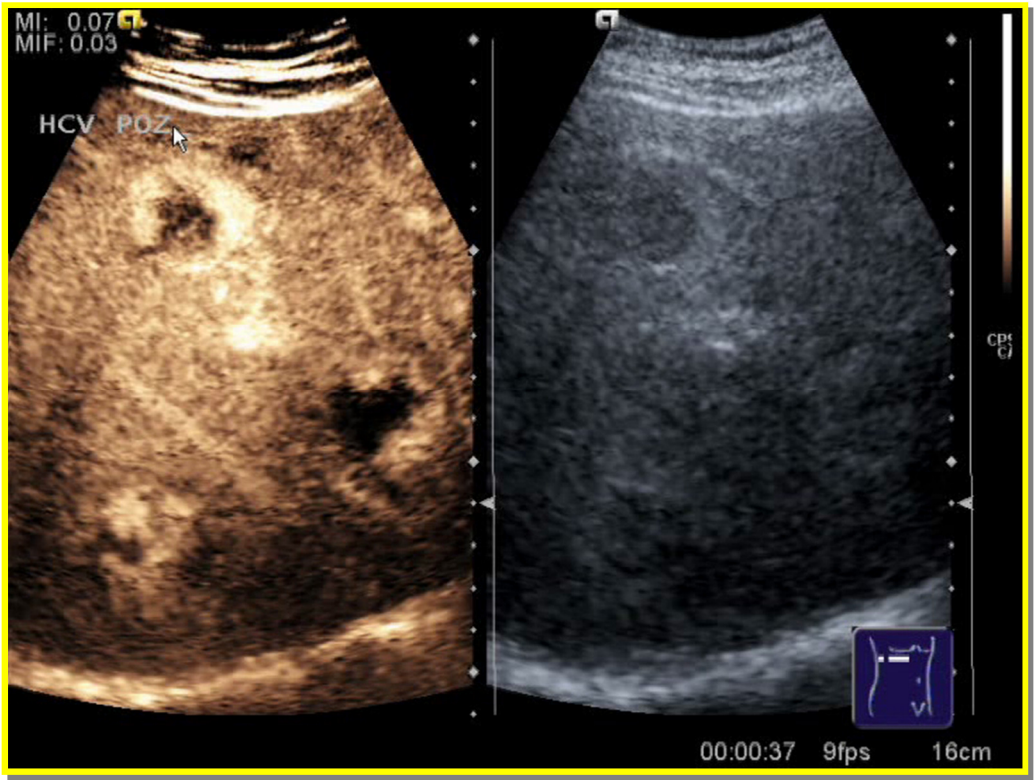
Hemangioma: peripheral nodular enhancement of the contrast (SonoVue) in arterial phase



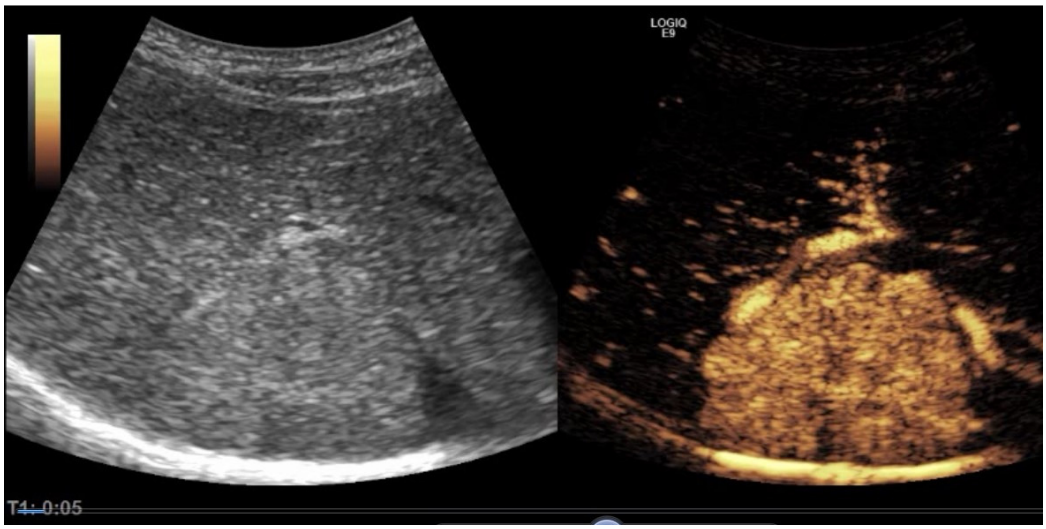
Hemangioma: in late phase keeping the contrast (no washout)



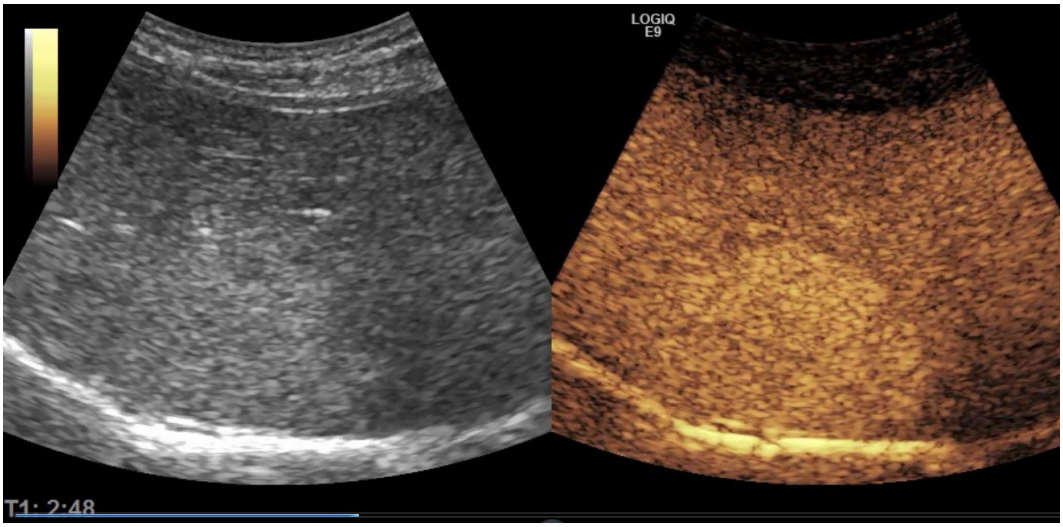
Hemangioma: peripheral nodular enhancement of the contrast (SonoVue) in arterial phase



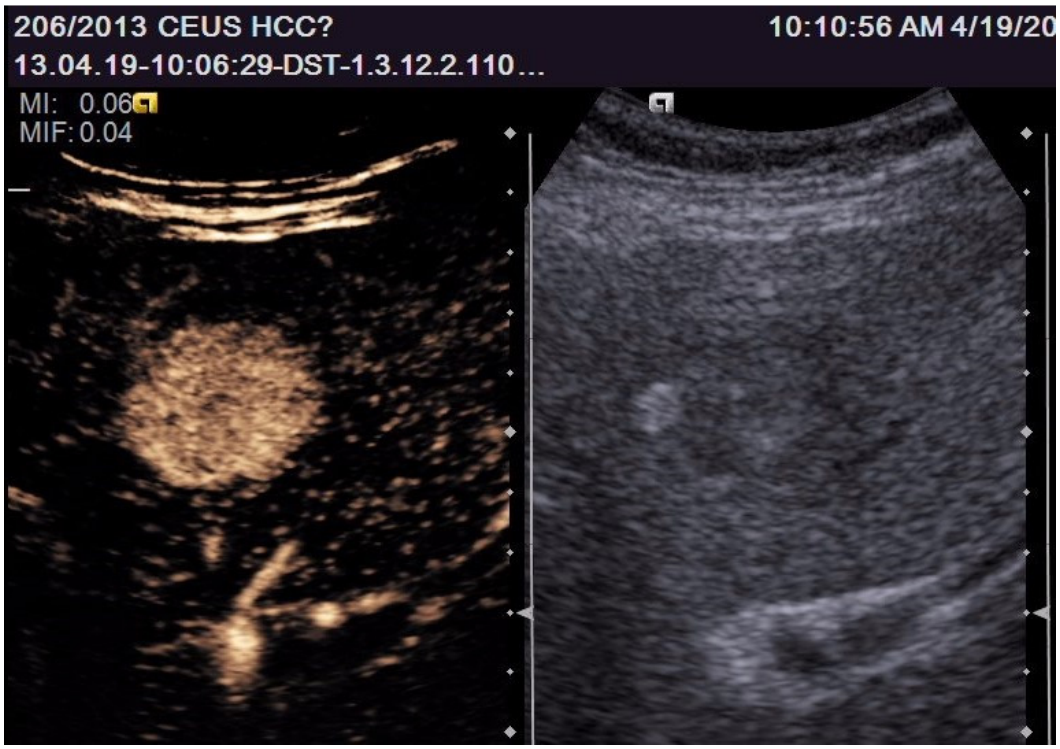
Liver hemangiomatosis (many lesions with peripheral, nodular enhancement)



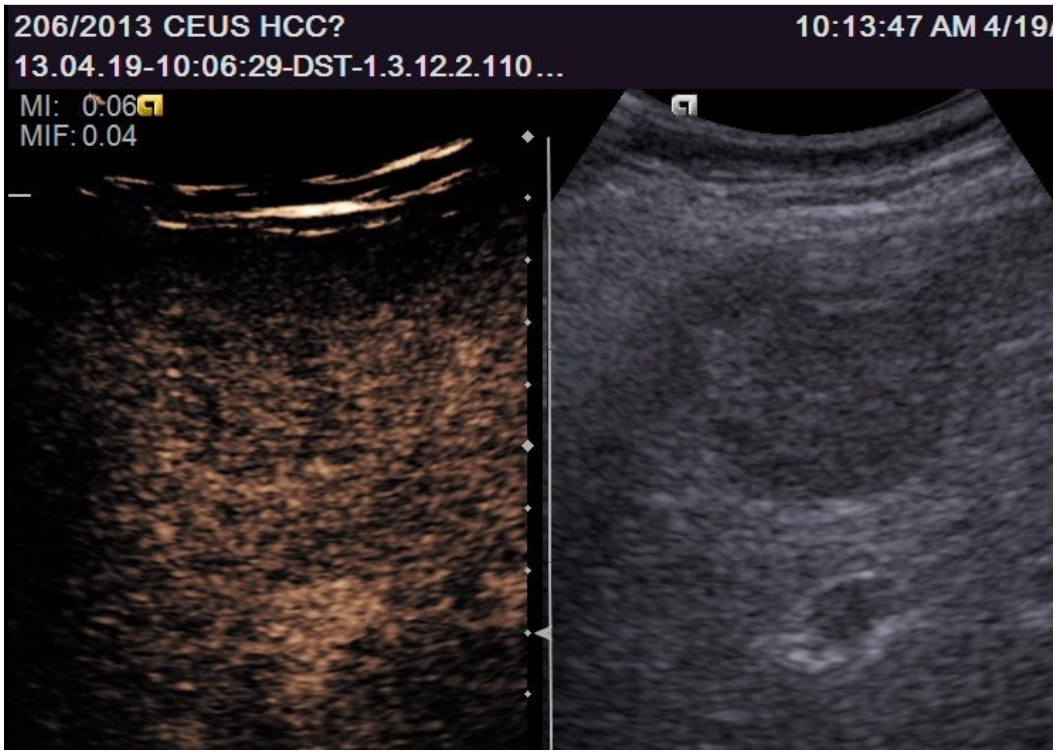
FNH with ultrasound contrast-SonoVue: very early wash-in in arterial phase (8 seconds after injection)



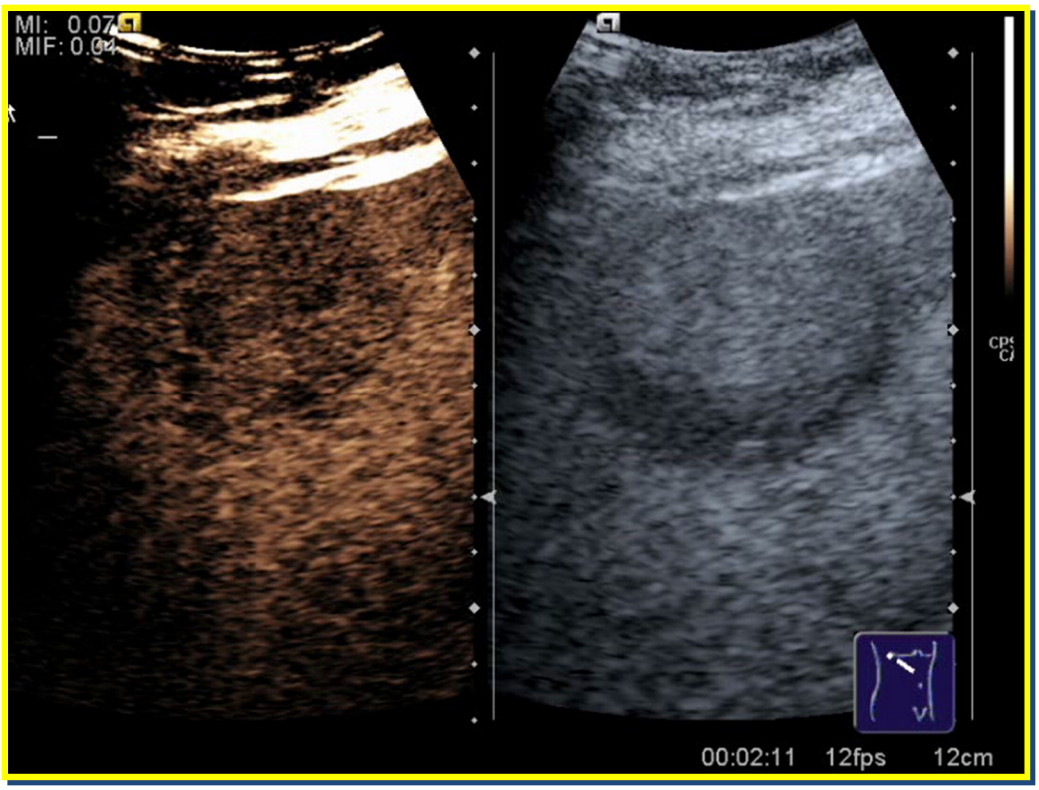
FNH with contrast in the lesion in the late phase (no wash-out)



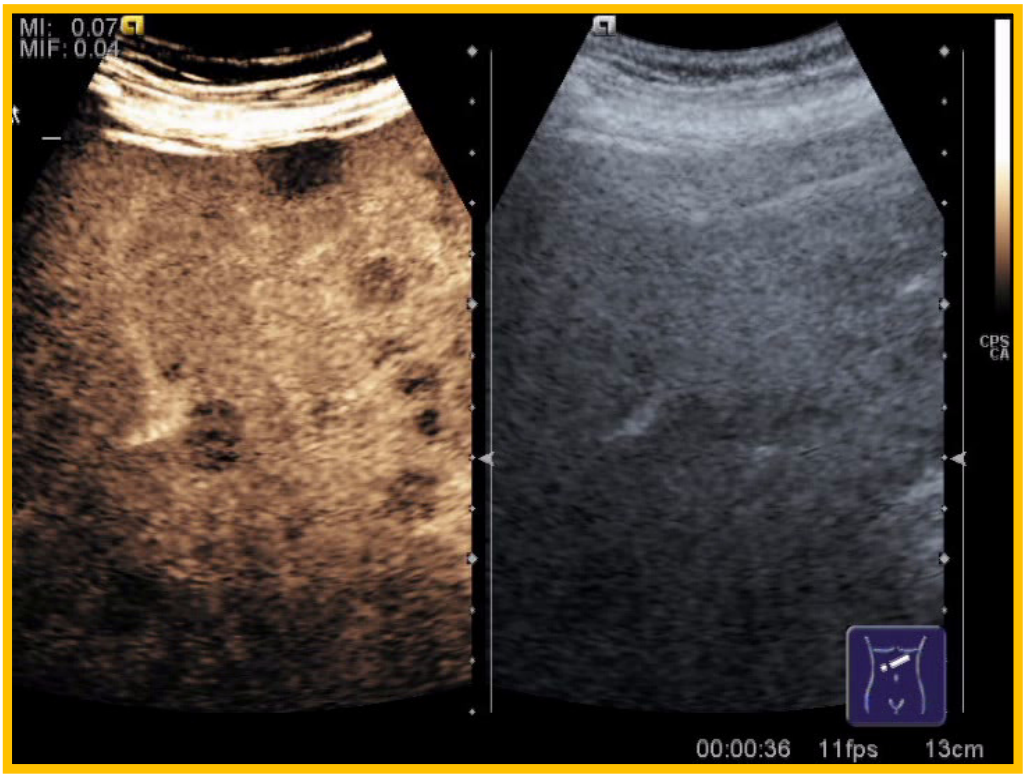
HCC: early wash-in of the contrast in arterial phase



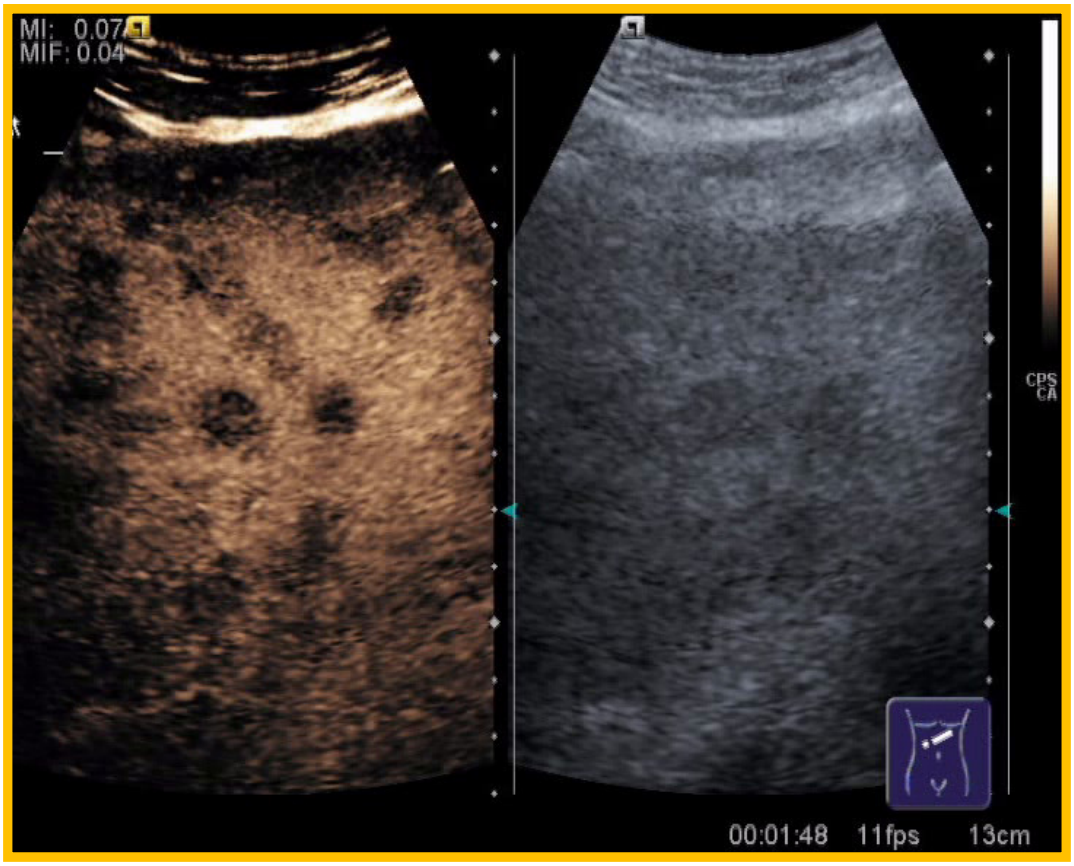
HCC: late and weak wash-out of the contrast in late phase (typically for HCC)



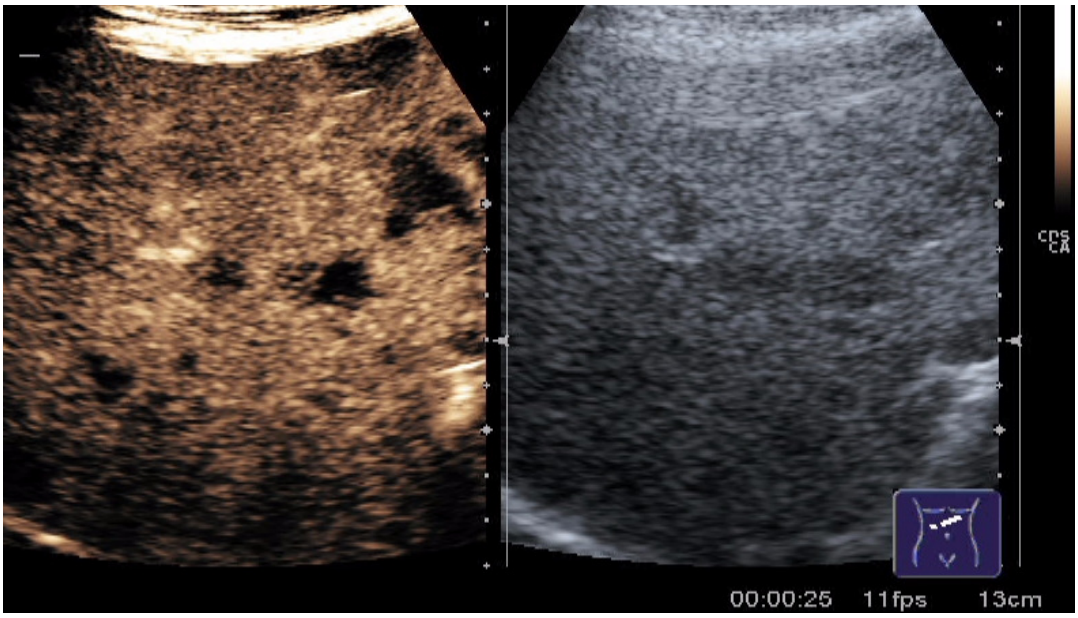
Other case of HCC: late and weak wash-out of the contrast (typical for HCC) in late phase



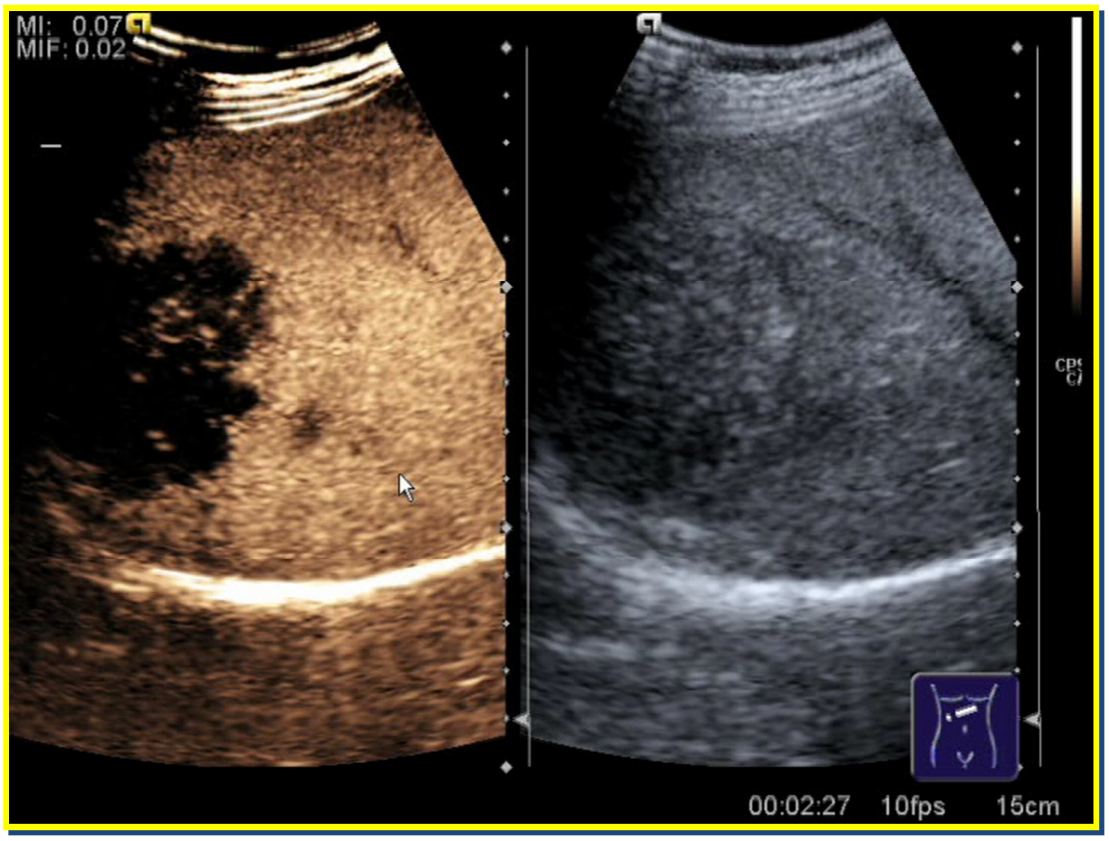
Liver metastasis: early wash-out in portal phasis



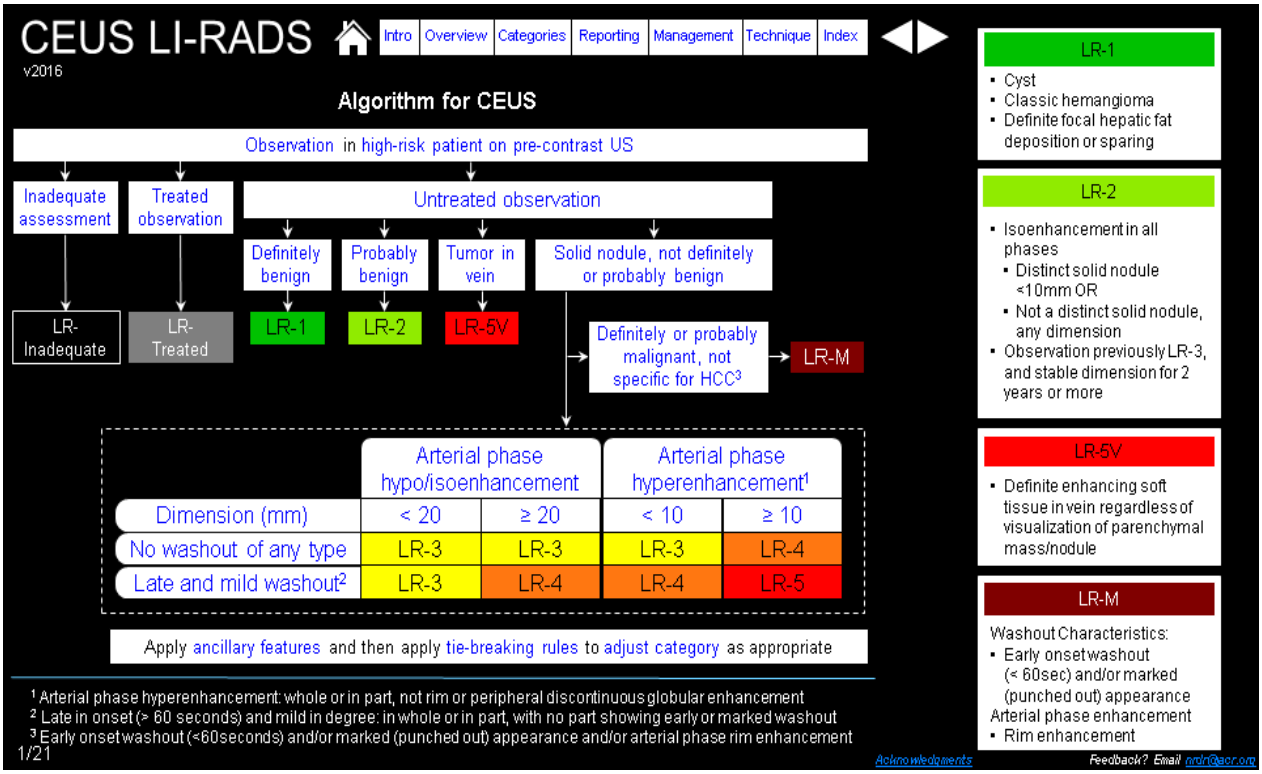
Liver metastasis: late portal phase (strong wash-out)



Usually wash-out means malignancy (multiple liver metastasis)



Liver metastasis: strong wash-out (sign of malignancy)



CEUS LI-RADS

5.6 Problem-solving and intervention

CEUS is useful for lesions that remain indeterminate on CT or MRI. It may clarify arterial vascularity, differentiate perfused tissue from debris, and support assessment of tumor thrombus. It can also help identify viable areas for biopsy and assess residual perfusion after ablation.

After local treatment, enhancing tissue may indicate residual or recurrent viable tumor, but interpretation depends on treatment type and timing. Reactive peri ablational changes can be misleading. A focused CEUS assessment may complement subsequent CT or MRI, which also evaluate the remainder of the liver and extrahepatic disease.

The principal limitations remain those of ultrasound: obesity, bowel gas, deep lesions, limited access to the dome, and operator dependence. Multiple lesions may require repeated injections or separate targeted assessments. The examination succeeds when it resolves a defined question and its limitations are incorporated into the next clinical decision.

Chapter 6. Computed Tomography

6.1 Physical principles and technological development

CT reconstructs cross-sectional images from X-ray attenuation measurements acquired around the patient. Each voxel is assigned an attenuation value expressed in Hounsfield units relative to water. Water is approximately 0 HU, air approximately −1,000 HU, and fat has negative values, commonly near −100 HU. Calcification and bone have substantially higher attenuation.

Thin-section volumetric acquisition permits multiplanar and three-dimensional reconstruction. Pixel size depends on the reconstruction field of view and matrix, while voxel dimensions also depend on section thickness. A small lesion can be obscured by partial-volume averaging even when the overall anatomical image appears sharp.

The development of continuous gantry rotation through slip-ring technology enabled helical scanning. Multidetector systems increased anatomical coverage per rotation and improved the acquisition of thin sections. Dual-source configurations and other technical refinements improved selected aspects of temporal resolution and sampling.

Conventional detectors generally convert X-ray energy into light and then an electrical signal. Reconstruction historically relied heavily on filtered back projection. Iterative and newer reconstruction methods can reduce noise and selected artifacts, supporting dose optimization, but image appearance and texture may vary with the algorithm.

6.2 Acquisition, reconstruction, and image quality

Diagnostic quality depends on spatial resolution, contrast resolution, noise, and artifacts. Lower radiation exposure generally increases noise if other factors remain constant. Patient size, section thickness, reconstruction method, and tube settings modify this relationship. A protocol should achieve sufficient quality for its specific task rather than aim for uniformly maximal detail.

Multiplanar reconstructions improve the assessment of lesion relationships, bile ducts, and vessels. Maximum-intensity projections emphasize enhanced vascular structures, while other projection and volume-rendering methods assist anatomical display. Such reconstructions supplement, rather than replace, review of the source images.

Motion, beam hardening, metallic material, and inadequate contrast timing may impair interpretation. Partial-volume effects can make small cysts appear denser than fluid and can obscure tiny lesions. Apparent tissue attenuation must therefore be related to the acquisition conditions rather than interpreted as an absolute pathological signature.

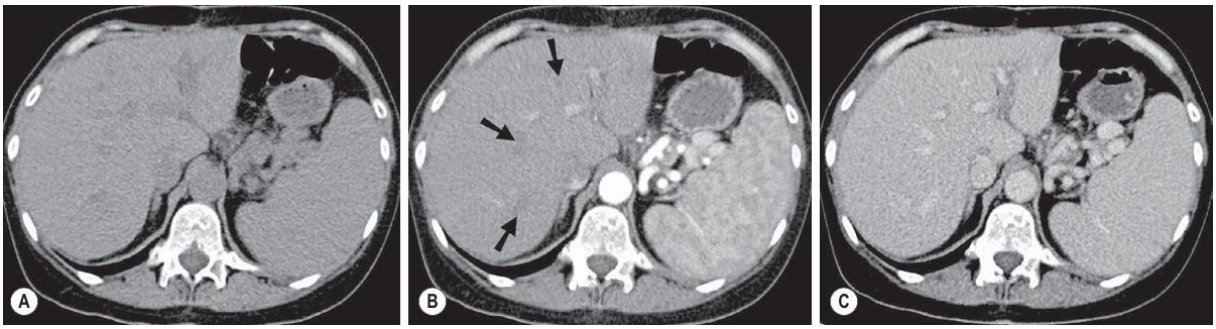
Dose optimization includes appropriate patient positioning, limited scan length, automatic exposure control, and selection of only the phases needed. The number of phases is especially important in liver imaging because each acquisition adds exposure. ALARA expresses the need for the lowest reasonably achievable dose that retains the required diagnostic information.

6.3 Multiphasic contrast-enhanced liver CT

Intravenous iodinated contrast permits assessment of perfusion and enhancement. A liver-specific examination may include unenhanced, late arterial, portal venous, and delayed acquisitions, but the combination should be tailored to the indication. A generic single-phase abdominal CT is not equivalent to a dedicated multiphasic liver study.

The unenhanced acquisition helps assess attenuation, fat, calcification, and hemorrhage in selected cases. The arterial phase is important for hyper-vascular lesions and arterial anatomy. The portal venous phase provides strong parenchymal enhancement and is central to the assessment of many metastases and abdominal organs. Delayed imaging may reveal persistent or progressive enhancement and contributes to characterization of selected lesions.

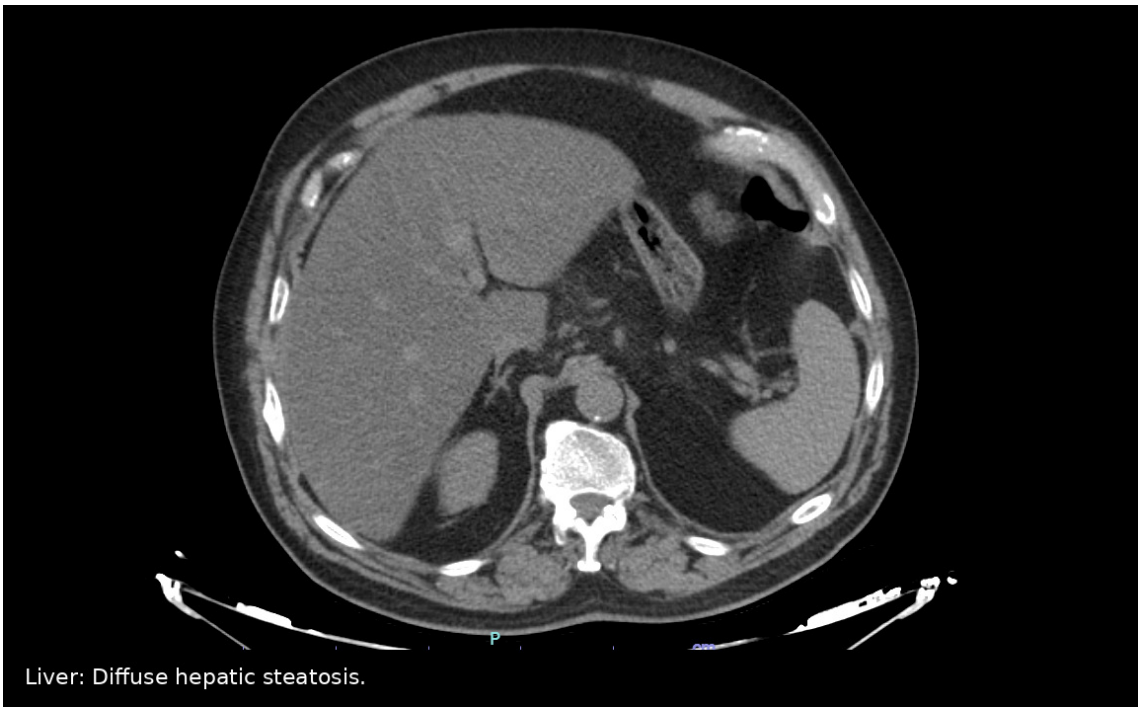
Bolus tracking or related timing methods coordinate scanning with the arrival of contrast. Injection rate, contrast concentration, body size, cardiac output, and scan speed all influence enhancement. A poorly timed arterial phase can reduce lesion conspicuity and cannot be compensated for solely by delayed images.



Multiphasic hepatic CT images illustrating changes in parenchymal and focal enhancement across acquisitions.

6.4 Diffuse hepatic disease

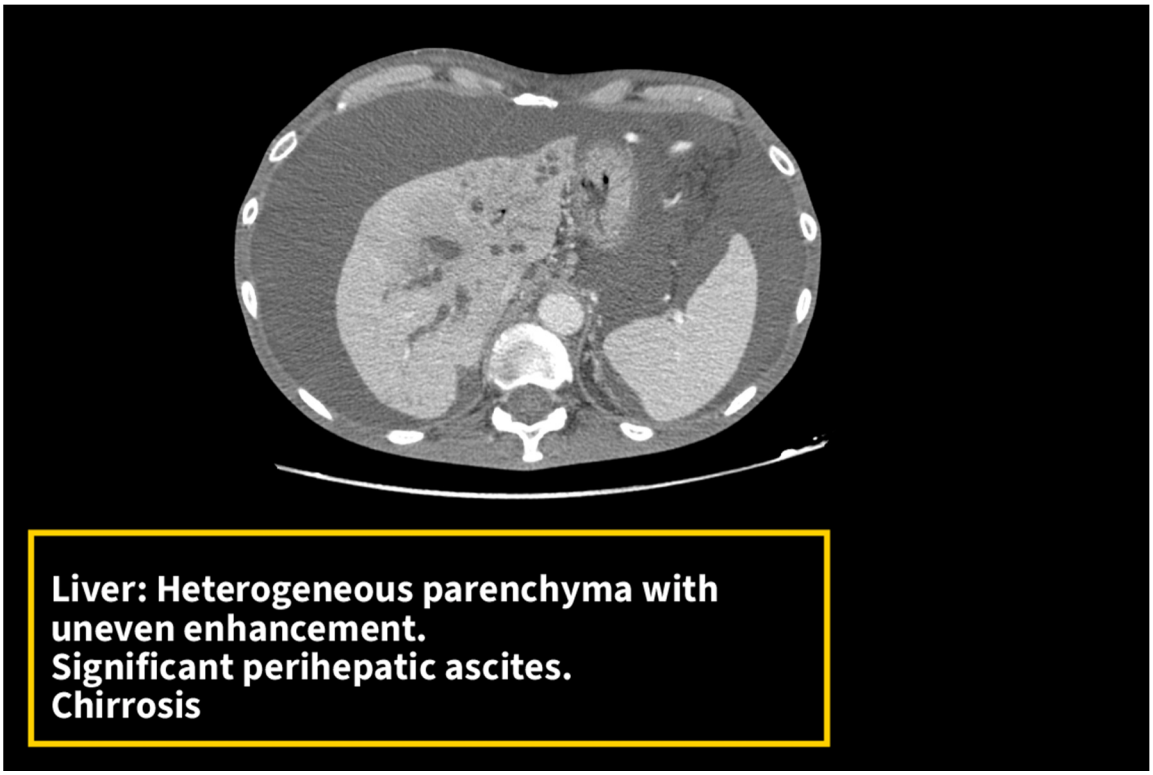
Unenhanced CT can suggest steatosis through reduced liver attenuation, often assessed relative to the spleen. Its accuracy is affected by disease severity, concomitant iron or fibrosis, scanner and acquisition factors, and other causes of altered attenuation. CT is not normally selected solely for precise serial measurement of hepatic fat when a more suitable quantitative technique is available.



Iron deposition may increase attenuation, but the finding is not specific and can be masked by coexisting fat. MRI provides a more appropriate quantitative assessment of iron concentration. In Wilson disease, CT

findings are usually non-specific and chiefly demonstrate structural consequences such as cirrhosis and portal hypertension.

Advanced cirrhosis may produce nodular contours, segmental atrophy and hypertrophy, heterogeneous enhancement, and signs of portal hypertension. CT depicts splenomegaly, ascites, and gastroesophageal, splenorenal, paraumbilical, and other collaterals. These findings are valuable for planning and complication assessment, but their absence does not exclude early fibrosis.



Acute hepatitis often has no specific CT signature. The examination is more useful when an alternative cause of jaundice, an acute complication, or an associated vascular or abdominal process must be excluded. It does not replace serological diagnosis or histological assessment of inflammatory activity.

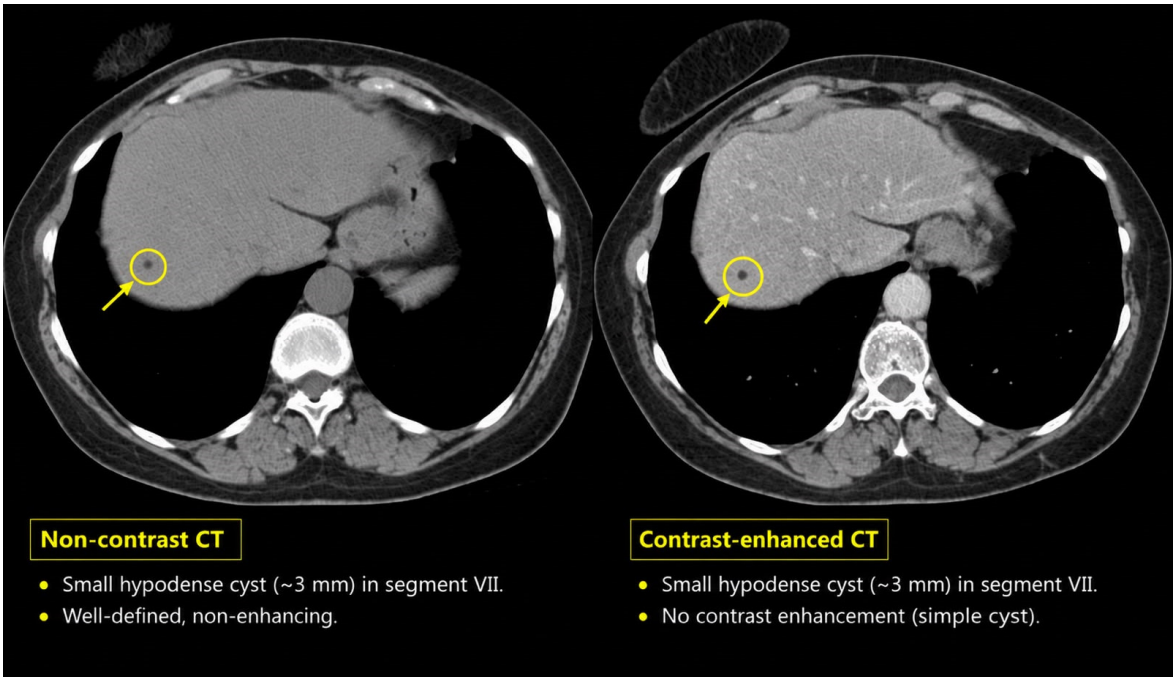
6.5 Benign, cystic, and infectious lesions

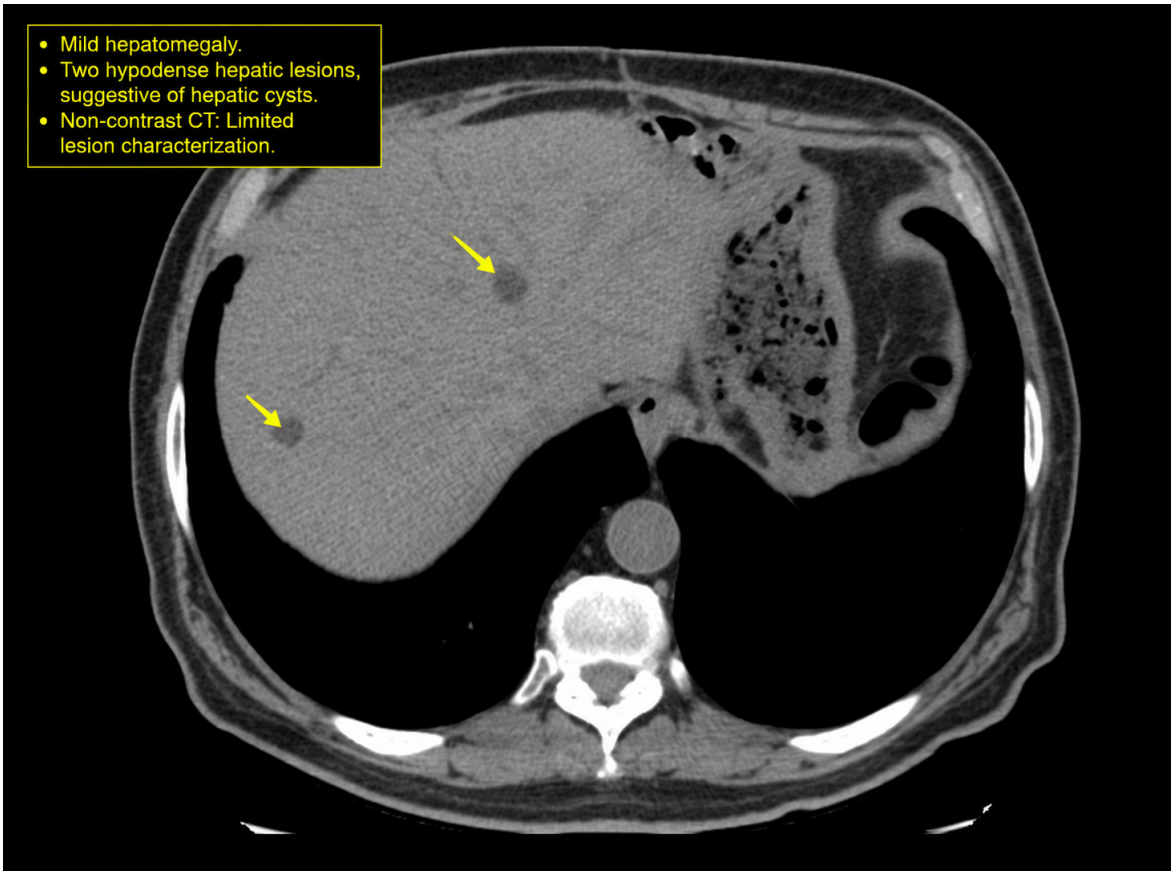
A simple cyst is usually homogeneous, low in attenuation, and non-enhancing, with a very thin or inapparent wall. Tiny cysts can be difficult to characterize because of partial-volume effects. A complex cystic lesion requires assessment of septa, wall thickness, mural nodules, internal density, and enhancement.

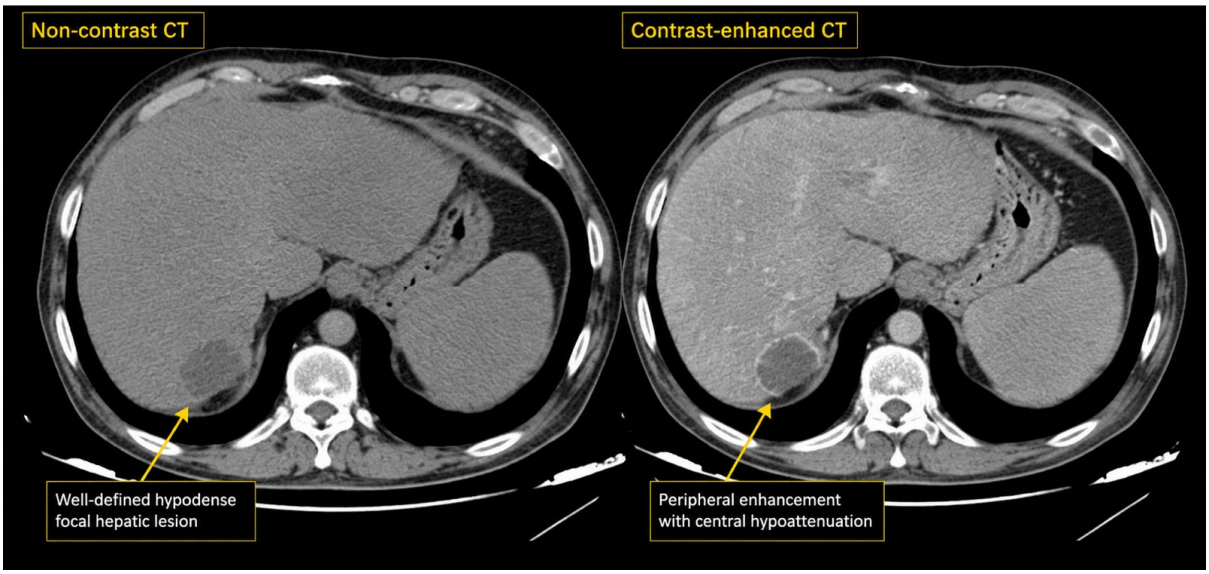
CT is particularly effective at showing calcification and gas. In hydatid disease, it may demonstrate wall calcification, daughter cysts, and complex internal structure, supplementing the ultrasound classification and clinical context. An abscess commonly appears as a low-attenuation collection with peripheral enhancement, sometimes with internal gas. Necrotic metastases can overlap in appearance, making clinical correlation and selected aspiration important.

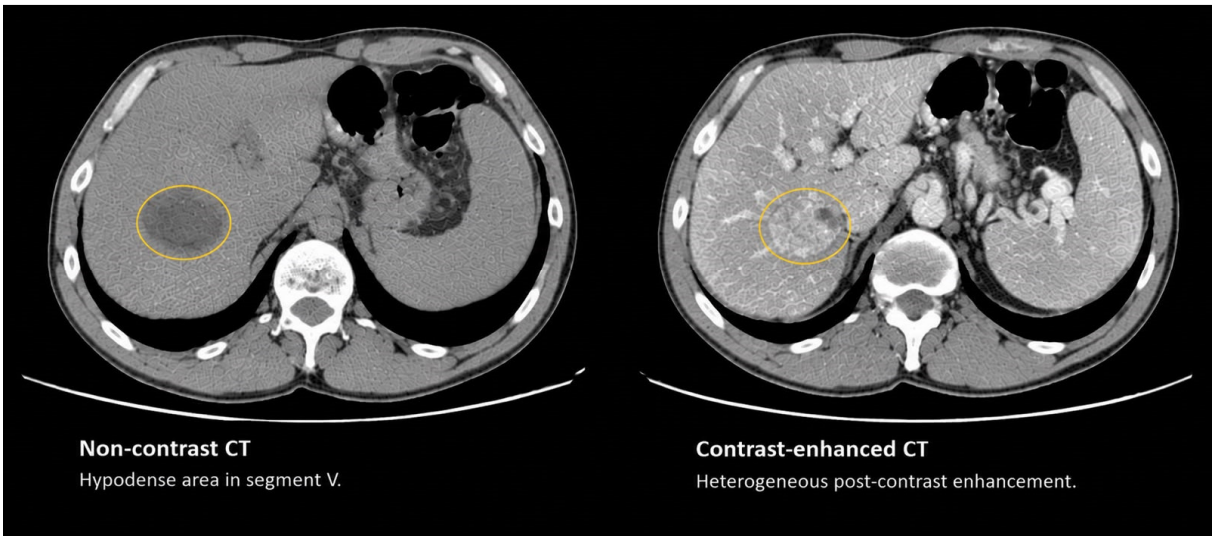
Pneumobilia tends to be centrally distributed, whereas portal venous gas often extends into peripheral branches. The significance of either finding depends on recent intervention, biliary-enteric communication, infection, bowel disease, and accompanying abnormalities. CT provides the anatomical context needed to interpret these signs.

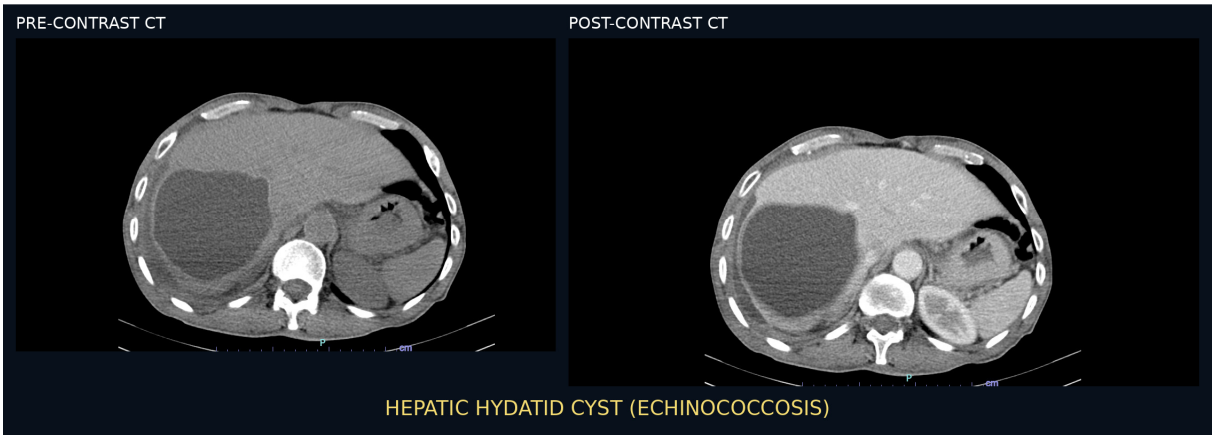
Hemangiomas classically demonstrate peripheral discontinuous nodular enhancement with progressive filling. FNH and adenoma can both enhance in the arterial phase, and CT may not confidently distinguish them. MRI is frequently the next examination when tissue characterization will influence management.

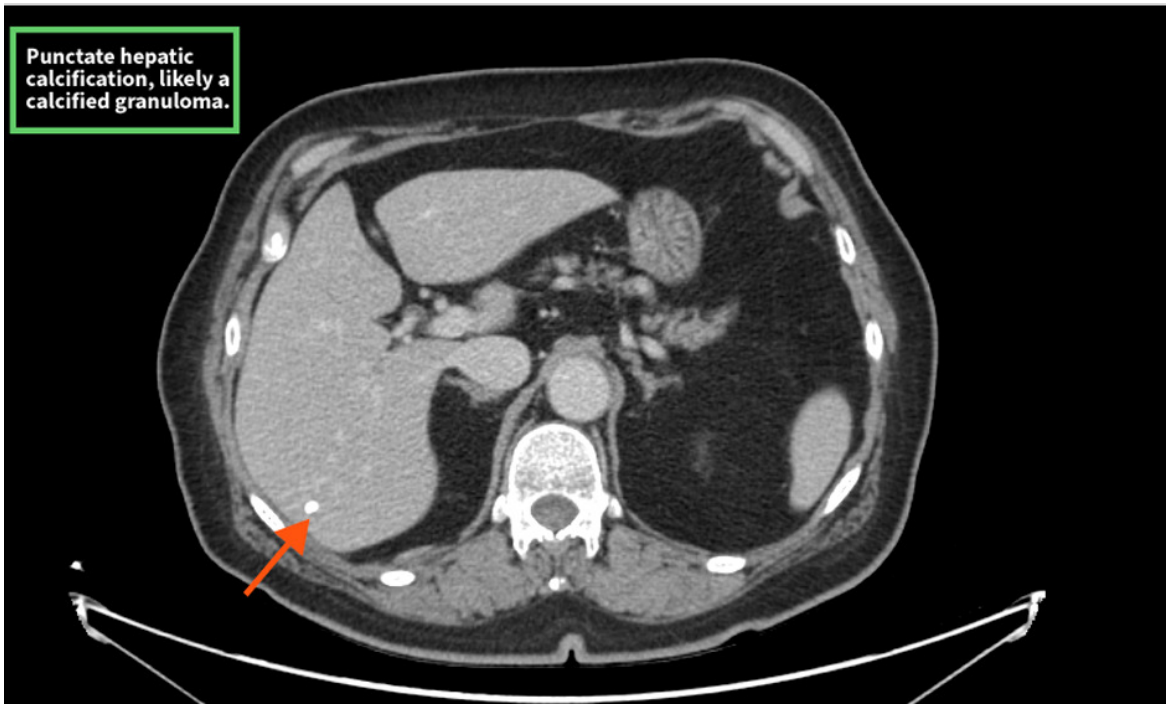


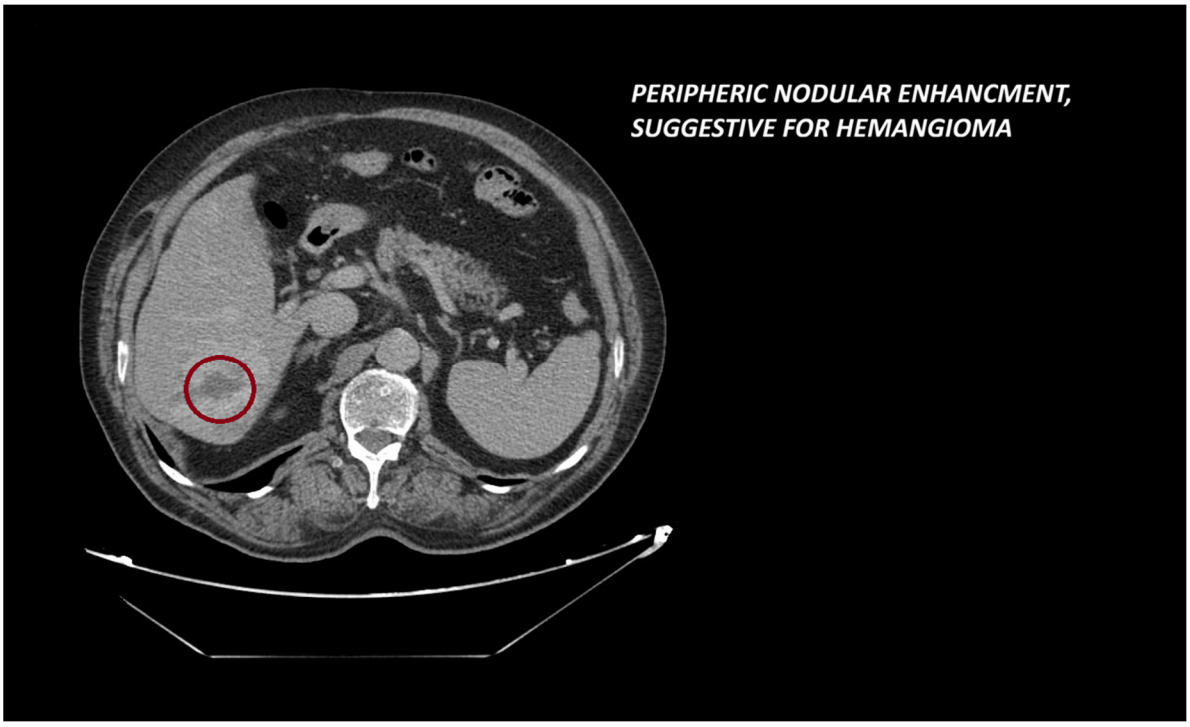



Non-contrast CT
Contrast-enhanced CT
Well-defined hypodense
focal hepatic lesion
Peripheral enhancement
with central hypoattenuation


Non-contrast CT
Hypodense area in segment V.
Contrast-enhanced CT
Heterogeneous post-contrast enhancement.


PRE-CONTRAST CT
POST-CONTRAST CT
HEPATIC HYDATID CYST (ECHINOCOCCOSIS)





6.6 Malignancy and oncological staging

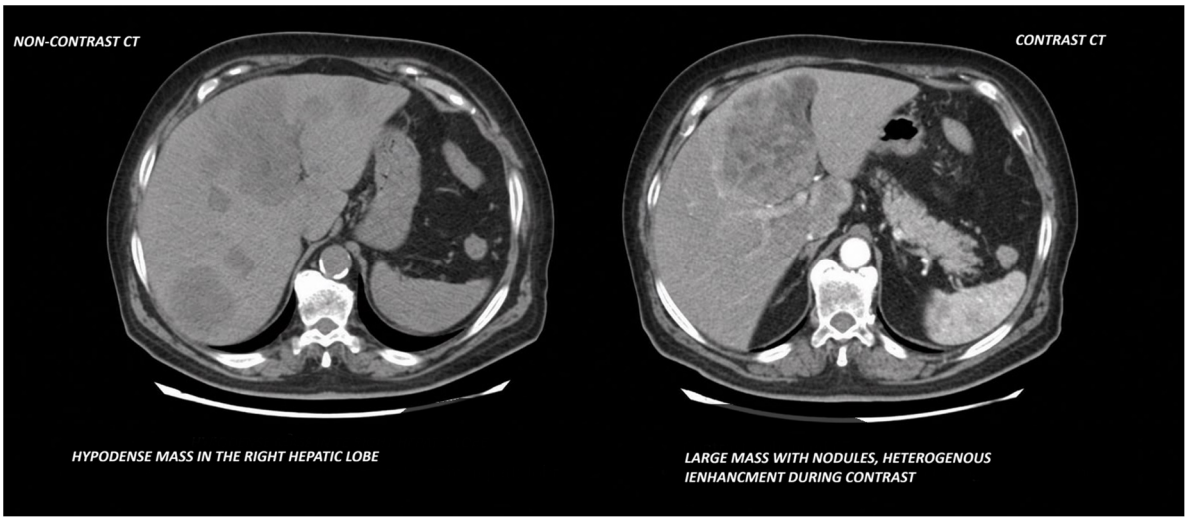
CT is widely used for staging because it rapidly evaluates the liver and extrahepatic organs. The assessment includes lesion number, size, distribution, vascular relationships, nodal disease, and distant spread. The clinically important endpoint may be resectability or treatment eligibility rather than detection of every microscopic abnormality.

Many metastases are hypo-vascular and become most conspicuous against enhancing liver in the portal venous phase. Hyper vascular metastases may require an arterial acquisition. Mucinous metastases and selected other lesions can contain calcification. The protocol should reflect the known or suspected primary tumor.

In high-risk patients, HCC can be diagnosed non-invasively when accepted imaging criteria are met. Non-rim arterial hyperenhancement, appropriate washout, capsule appearance, lesion size, and growth contribute within the relevant framework. Not all HCCs display the classic pattern, and arterial enhancement alone is not diagnostic.

Small lesions are particularly challenging in a heterogeneous cirrhotic liver. Perfusion abnormalities may mimic tumors, while early or atypically vascularized HCC may be difficult to identify. An indeterminate CT observation may require MRI, selected CEUS, interval imaging, or biopsy according to the category and clinical setting.

After therapy, assessment must match the treatment and response criteria. Tumor size remains important in many systemic oncology contexts, but viable enhancing tumor is central to selected HCC and locoregional treatment assessments. A decrease in enhancement can indicate treatment effect, yet technical differences between examinations must be excluded before concluding biological response.





6.7 Vascular disease and trauma

CT angiographic and venographic assessment maps hepatic vessels and the extent of thrombosis or obstruction. Portal thrombosis appears as a filling defect; chronic occlusion may produce cavernous transformation and collaterals. Internal enhancement, expansile tissue, and continuity with a tumor can support concern for tumor in the vein, although assessment requires the full examination.

Budd–Chiari syndrome can produce heterogeneous congestion, abnormal venous outflow, caudate enlargement, and collateral channels. Arterioportal shunts may cause early portal enhancement and transient regional parenchymal hyperenhancement. Recognizing a perfusion pattern across phases prevents a pseudolesion from being mistaken for a mass.

Contrast-enhanced CT is the principal comprehensive examination for significant hepatic trauma in patients sufficiently stable to undergo scanning. It identifies lacerations, intrahepatic and subcapsular hematomas, vascular injuries, hemoperitoneum, and active bleeding. FAST ultrasound can rapidly identify free fluid but cannot exclude or fully characterize parenchymal injury.

The injury grade, active bleeding, associated injuries, and physiological condition together guide management. CT also evaluates later complications such as collections, necrosis, or biliary-related abnormalities. A scan is one component of trauma decision-making and should not delay urgent resuscitative or operative care in an unstable patient.

6.8 Intervention, contrast safety, and limitations

CT guides biopsy, aspiration, drainage, and ablation when the target is inaccessible or poorly seen on ultrasound. It provides a clear map for needle planning. Repeated acquisitions and CT fluoroscopy can increase radiation exposure, and respiratory movement may complicate targeting.

Contrast administration requires consideration of previous reactions, current kidney function when clinically relevant, and other individual risk factors. Acute kidney injury temporally associated with contrast is not necessarily caused by contrast. The distinction between contrast-associated and contrast-induced injury avoids attributing every subsequent creatinine rise to the examination. In patients with acute kidney injury

or severe renal impairment, the benefit and risk require individualized assessment. Kidney disease should not be described as an automatic contraindication to every necessary contrast-enhanced CT. [3,70]

Other limitations include cumulative radiation, limited sensitivity for early microscopic fibrosis, reduced confidence in very small lesions, and overlapping enhancement patterns. CT offers excellent rapid anatomical coverage but comparatively less tissue-specific information than a comprehensive liver MRI. Its strongest role is determined by the clinical objective, particularly when speed, broad coverage, and vascular or traumatic assessment are priorities.

Chapter 7. Magnetic Resonance Imaging

7.1 Signal generation and tissue contrast

MRI uses a static magnetic field, radiofrequency excitation, and spatially varying gradients to generate images, mainly from hydrogen nuclei in water and fat. It does not use ionizing radiation. Its diagnostic flexibility arises from the ability to alter the acquisition so that different tissue properties contribute to contrast. [1,37–39]

Following excitation, longitudinal magnetization recovers according to T1 relaxation, while transverse coherence decays according to T2 relaxation. T2* additionally incorporates local magnetic field inhomogeneity. These processes vary between tissues and are affected by water, fat, blood products, iron, and the local molecular environment.

T1-weighted imaging is useful for anatomy, fat-related findings, selected hemorrhagic components, and post-contrast assessment. T2-weighted imaging emphasizes fluid and other tissues with prolonged T2. Gradient-echo techniques support rapid volumetric and dynamic imaging, while fast spin-echo and other sequences provide complementary tissue information.

Spatial encoding organizes data in k-space before image reconstruction. Two- and three-dimensional acquisitions allow imaging in multiple planes. Parallel imaging and accelerated techniques reduce acquisition time but can affect signal-to-noise ratio or introduce artifacts. The protocol should balance coverage, resolution, contrast, and motion robustness.

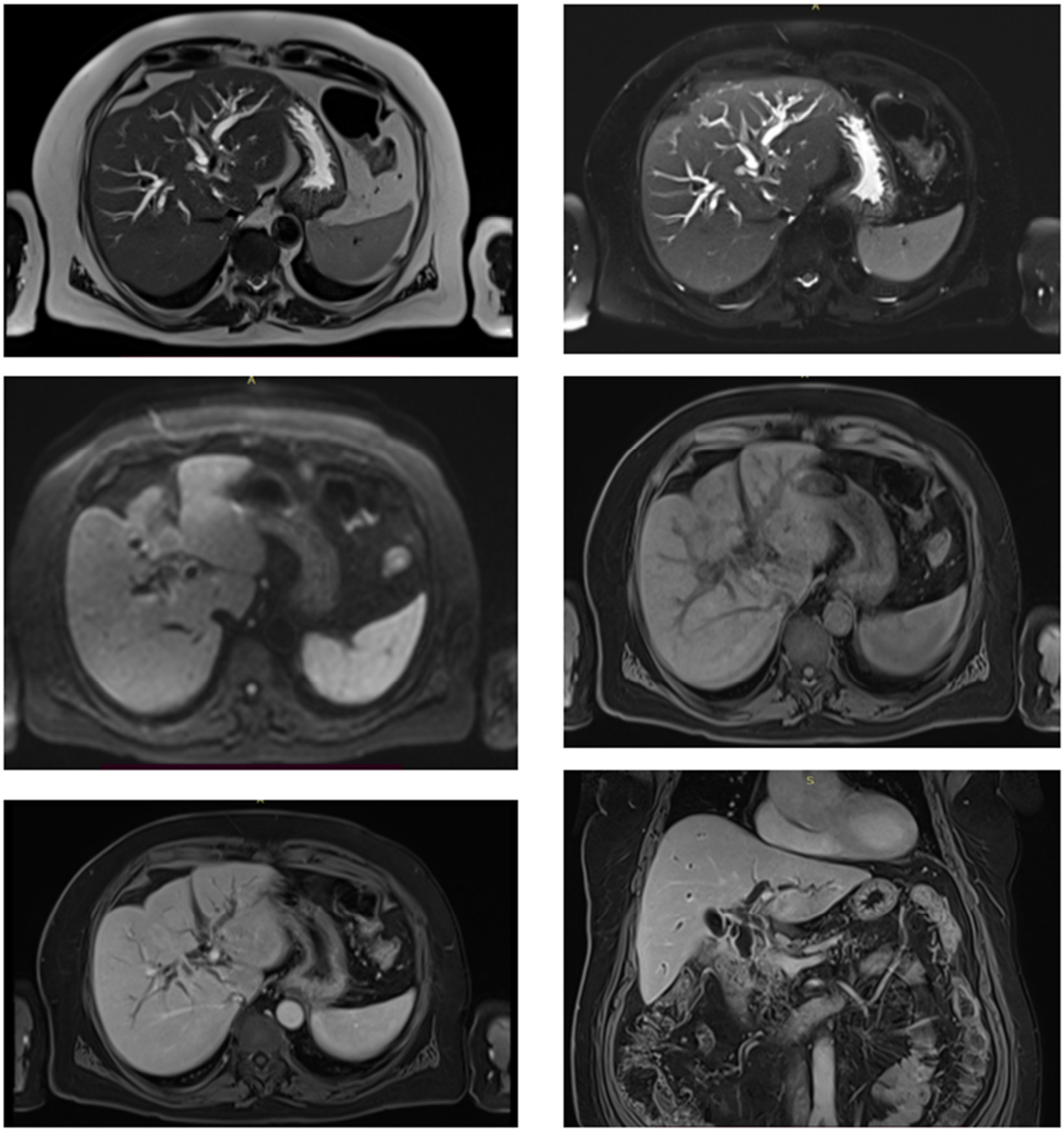
7.2 A liver-specific multiparametric examination

A typical comprehensive liver MRI combines T2-weighted sequences, T1-weighted chemical-shift or water–fat imaging, diffusion-weighted imaging with ADC maps, and dynamic contrast-enhanced T1-weighted acquisitions when indicated. Additional components include hepatobiliary-phase imaging, iron quantification, MRCP, and MRE according to the diagnostic question.

This combination is valuable because a lesion’s signal on one sequence is rarely sufficient. A mass may be identified on diffusion imaging, characterized by T2 signal and fat content, and classified further through vascular and hepatobiliary behavior. The confidence of interpretation depends on the coherence of these findings and the clinical context.

Respiratory motion is a central challenge. Breath-hold, respiratory-triggered, and motion-robust acquisitions can be selected according to patient ability. A technically degraded arterial phase may require an alternative strategy, particularly when arterial hyperenhancement is decisive for classification.

MRI signal intensity is not an absolute scale comparable to HU. It depends on sequence parameters, equipment, coil sensitivity, and other factors. Quantitative measurements require validated acquisition and analysis methods; a subjective signal description should not be mistaken for a standardized numerical biomarker.



Liver multiparametric examination sequences.

7.3 Chemical-shift imaging and fat quantification

Water and fat resonate at slightly different frequencies. In-phase and opposed-phase imaging exploit this difference to detect microscopic fat through signal cancellation. A reduction in signal on the opposed-phase acquisition supports coexistence of water and fat within the sampled tissue but does not independently establish a precise fat fraction.

Modern multi-echo water–fat separation methods permit estimation of proton density fat fraction. Appropriate techniques account for confounding influences such as T2* decay and the multiple spectral components of fat. MRI-PDFF provides a quantitative measure useful for evaluating steatosis and monitoring changes in hepatic fat.

Fat assessment also helps distinguish focal fatty deposition or sparing from a true mass. Preserved vessels, lack of displacement, and a compatible distribution support a pseudo-lesion, while MRI provides direct evidence of the relevant fat-related signal behavior.

MRI-PDFF does not measure every component of steatohepatitis. Inflammation, ballooning injury, and fibrosis require separate assessment. A change in PDFF may be a useful treatment endpoint, but it is not identical to a change in all histological or prognostic features of disease.

7.4 Iron-sensitive imaging

Iron shortens relaxation and causes signal loss, particularly on T2- and T2*-sensitive acquisitions. Quantitative R2 or R2* methods can estimate hepatic iron concentration using validated calibrations. These measurements support assessment and monitoring of hereditary or transfusional iron overload.

The distribution of signal loss in the liver and other organs may assist etiological interpretation, but quantitative assessment must follow an appropriate method. Coexisting fat and severe iron deposition can complicate acquisition or fitting. In marked overload, very rapid signal decay may require particular sequence choices.

CT may suggest iron through increased attenuation, but MRI provides a more suitable quantitative approach without repeated radiation exposure. The method and calibration should be documented when serial concentrations are compared.

7.5 Diffusion-weighted imaging

DWI is sensitive to the microscopic movement of water and is influenced by tissue cellularity, extracellular space, and other factors. High signal at higher diffusion weighting, accompanied by appropriately low ADC, supports restricted diffusion. High DWI signal without reduced ADC may reflect T2 shine-through.

DWI improves lesion detection, particularly when used alongside anatomical and contrast-enhanced sequences. It is not specific for malignancy: abscesses and other benign processes can restrict diffusion, while some tumors may show variable restriction. ADC values can overlap between entities and vary with the acquisition.

Diffusion findings should therefore strengthen a multiparametric interpretation rather than determine a diagnosis independently. Motion and susceptibility distortion, particularly near air–tissue interfaces, can produce misleading focal appearances.

7.6 Dynamic and hepatobiliary contrast imaging

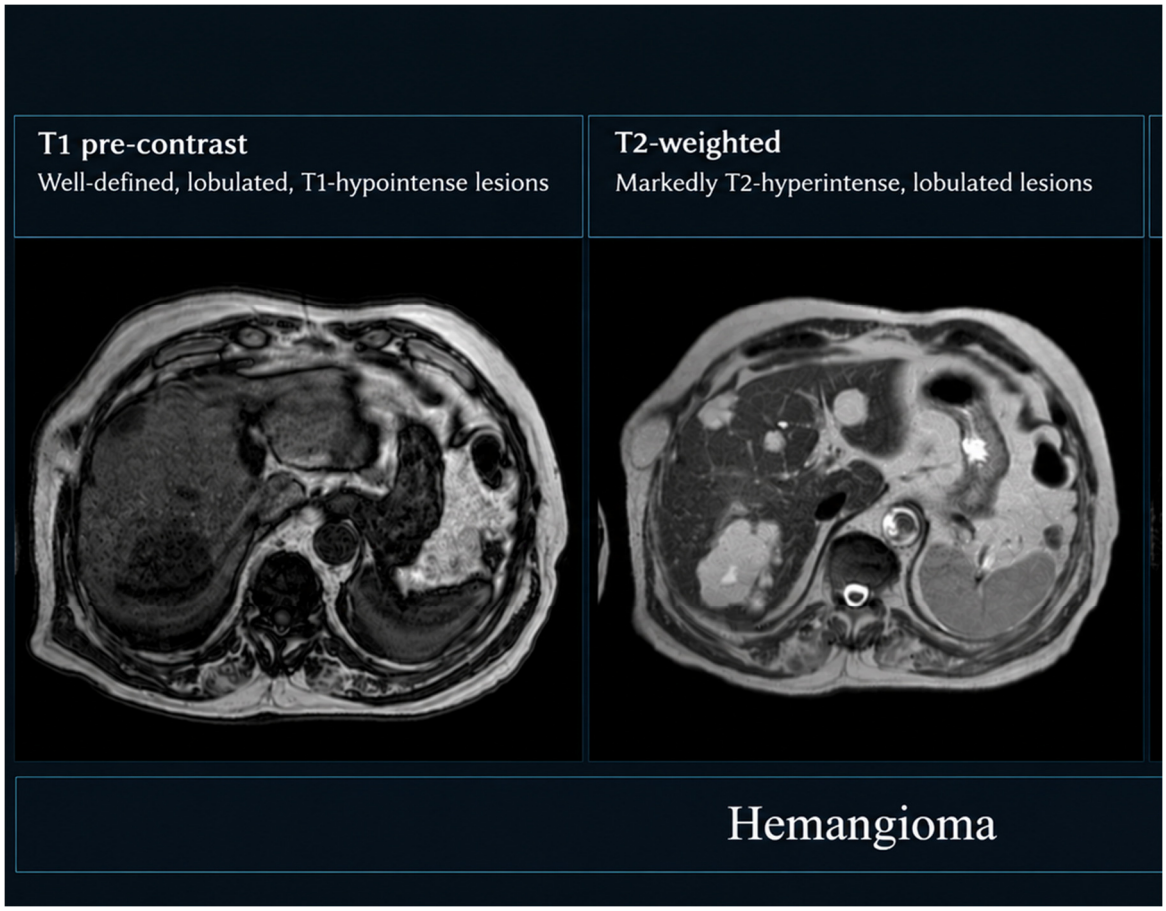
Gadolinium-based agents usually increase signal on T1-weighted images. Extracellular agents provide vascular and delayed extracellular information. Hepatobiliary agents add information about uptake by functioning hepatocytes and subsequent excretion, producing a later phase that can improve lesion detection and characterization.

The contrast agent changes the interpretation of phases. A transitional or hepatobiliary hypointense lesion is not automatically equivalent to true vascular washout. In particular, increased background hepatic uptake may make a lesion appear relatively darker without actual contrast loss. Criteria should follow the applicable CT/MRI LI-RADS framework and the agent used.

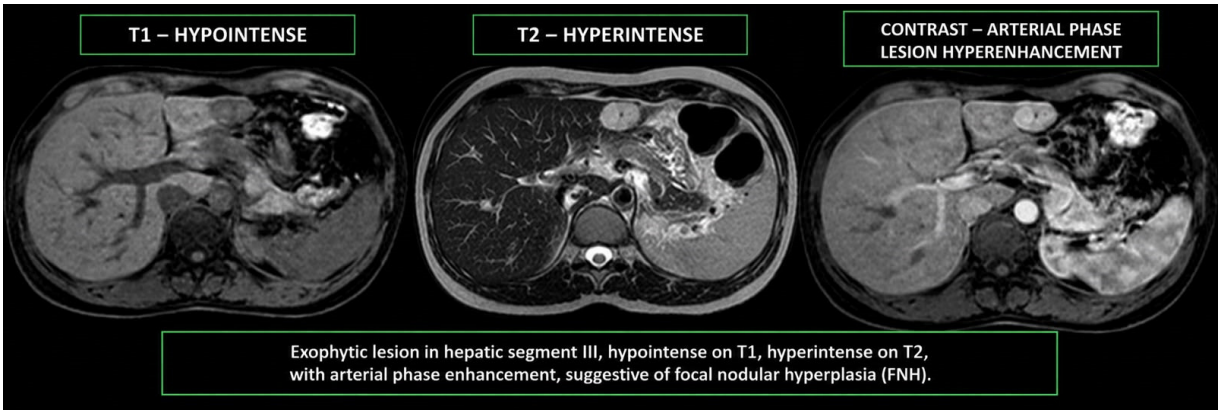
Hepatobiliary hypo-intensity is a valuable but non-specific observation. Metastases and many other lesions lack normal hepatocyte uptake. Some hepatocellular tumors may retain uptake, and reduced background function can impair the quality of the phase. Uptake must be interpreted as part of the lesion’s complete imaging profile.

7.7 Characterization of benign lesions

Hemangiomas typically have markedly high T2 signal and peripheral discontinuous nodular enhancement with progressive centripetal filling. The combination of morphology, T2 behavior, and vascular pattern can provide high diagnostic confidence. Atypical hemangiomas, including lesions affected by sclerosis or other change, may require a more cautious assessment.



FNH commonly shows relatively homogeneous arterial enhancement and usually retains hepatobiliary contrast. A central scar may be present, with characteristic T2 and delayed enhancement behavior, but small lesions may lack a visible scar. Its demonstration is therefore supportive rather than mandatory in every case.



Hepatocellular adenomas are heterogeneous in biology and appearance. Fat, hemorrhage, inflammation, and necrosis can affect their signal. Many are hypointense in the hepatobiliary phase, but some subtypes may retain contrast. The distinction from FNH cannot be reduced to a universal uptake/no-uptake rule, and imaging cannot reliably determine every adenoma subtype.

MRI is often the preferred problem-solving technique for these lesions because it combines tissue composition with enhancement and hepatobiliary behavior. The practical value lies in avoiding unnecessary intervention when characterization is confident and in identifying lesions whose uncertainty or risk requires further assessment.

7.8 HCC, metastases, and other malignancies

MRI contributes to the detection and characterization of HCC in eligible high-risk patients. Dynamic arterial enhancement, washout assessed in the appropriate phase, capsule appearance, size, and growth form part of established classification. Diffusion restriction, T2 signal, fat, and hepatobiliary features may provide additional information but do not replace major criteria indiscriminately.

Early HCC and dysplastic nodules may not show a classic vascular pattern. Hepatobiliary imaging can reveal observations before they acquire all conventional diagnostic features, yet a hypointense nodule alone is not definitive proof of HCC. The category and subsequent management should reflect the combination of findings and uncertainty.

MRI is particularly valuable when the presence or number of small metastases changes surgical planning. DWI and hepatobiliary-phase imaging improve conspicuity in many settings, including difficult background liver or indeterminate CT observations. Performance still depends on lesion size, contrast agent, tumor biology, and technical quality.

Intrahepatic cholangiocarcinoma may demonstrate rim enhancement, progressive enhancement of fibrous components, capsular retraction, or associated biliary changes. Such findings can overlap with other malignancies. A malignant pattern does not necessarily identify the exact histology, especially outside a defined non-invasive diagnostic framework.

7.9 MRCP and biliary assessment

MRCP uses heavily T2-weighted acquisitions to display fluid-filled biliary and pancreatic ducts. Standard MRCP can be performed without intravenous contrast and provides a non-invasive map of ductal anatomy. Three-dimensional datasets and source-image review are important because projection images can hide small defects or overlapping structures.

Applications include suspected obstruction, ductal stones, strictures, congenital variants, and primary sclerosing cholangitis. Multifocal narrowing with intervening dilatation can support PSC in the appropriate context. MRCP maps the distribution, while the accompanying MRI evaluates the parenchyma and potential masses.

Motion, small ducts, limited fluid content, and overlapping structures can reduce sensitivity. A negative examination does not exclude every small stone or early ductal abnormality. ERCP remains important when therapeutic intervention or selected tissue sampling is required; MRCP reduces the need for purely diagnostic invasive cholangiography in many settings.





7.10 Magnetic resonance elastography

MRE applies mechanical vibration and uses motion-sensitive sequences to evaluate wave propagation through the liver. The resulting stiffness maps sample a larger region than many ultrasound techniques and can be integrated into a multiparametric MRI examination.

It supports non-invasive fibrosis assessment when ultrasound elastography is unreliable, discordant with other evidence, or insufficient for the clinical question. As with ultrasound methods, stiffness is influenced by more than fibrosis, and appropriate quality assessment is necessary. Severe iron-related signal loss can impair some MRE sequences.

MRE does not provide a direct microscopic measurement of collagen or eliminate the need for every biopsy. Its value is greatest when the measurement changes risk assessment or management and is interpreted with the other imaging and clinical data.

7.11 Practical limitations and safety

MRI is generally longer and more technically complex than CT. Dyspnea, pain, large-volume ascites, inability to follow instructions, and claustrophobia may degrade quality or prevent completion. Availability, cost, and specialist expertise also influence its use, particularly in emergency settings.

Safety screening must address implants, devices, metallic foreign bodies, and the conditions under which a device may be scanned. The presence of a cardiac or other implant does not automatically establish an absolute contraindication; the specific device and required protocol determine suitability. Static-field hazards, radiofrequency heating, and acoustic exposure require established MRI safety procedures.

Gadolinium safety is agent-specific. The historical risk of nephrogenic systemic fibrosis is principally associated with particular agents and severe renal dysfunction; low-risk agents have a very low observed risk. A clinically necessary enhanced MRI should not be rejected solely by an undifferentiated statement that all gadolinium is prohibited in renal disease. Selection requires current agent guidance and individualized assessment. [3,71]

Gadolinium retention has been demonstrated, but its presence should not be equated with established clinical injury in every patient. Contrast should be used when it contributes necessary information, with the agent and dose appropriate to the task. Non-contrast MRI and MRCP can answer selected questions, but they are not equivalent to a full dynamic contrast examination for every focal lesion.

Finally, conventional MRI may be normal in early microscopic disease. Its high tissue contrast does not mean that it independently establishes all etiologies or grades all inflammation. A technically strong examination is most useful when it is integrated with the correct clinical question.

Chapter 8. Nuclear Medicine and Hybrid Imaging

8.1 Functional and molecular assessment

Nuclear medicine evaluates the distribution and kinetics of radiopharmaceuticals. The measured signal reflects a biological process, such as hepatocyte uptake and excretion, reticuloendothelial activity, or tumor metabolism. It therefore complements anatomical imaging rather than reproducing the information supplied by ultrasound, CT, or MRI. [1,7,22–26]

A gamma camera detects photons emitted by suitable radionuclides, while SPECT reconstructs their three-dimensional distribution. PET detects paired annihilation photons following positron emission. The tracer, physiological background activity, lesion size, and acquisition method determine what can be demonstrated.

The biological interpretation is tracer-specific. An abnormality on one study may not be visible on another because the two tracers measure different processes. Increased uptake is also not a universal marker of malignancy: inflammation and normal physiological activity can contribute to the signal.

8.2 Reticuloendothelial and hepatobiliary scintigraphy

Technetium-labeled colloid studies reflect reticuloendothelial uptake, particularly by Kupffer cells. Normal hepatic uptake is relatively homogeneous, while cirrhosis may produce heterogeneous uptake and redistribution toward the spleen and bone marrow. These patterns are functional observations and do not offer the detailed lesion characterization of contemporary CT or MRI.

A focal photopenic area is non-specific and may correspond to a lesion lacking normal reticuloendothelial tissue. Selected lesions containing Kupffer cells may show uptake, but this feature is not sufficient to characterize every focal mass. Many historical focal-lesion applications have been superseded by more anatomically detailed techniques.

Hepatobiliary scintigraphy uses tracers that undergo hepatocyte uptake and biliary excretion. Depending on the protocol, it can assess functional biliary questions and selected aspects of regional hepatic function. It is not interchangeable with a colloid scan, which measures a different biological pathway.

Before major resection, regional functional assessment can complement anatomical volumetry when the volume of the future liver remnant may not adequately represent its function. The clinical role depends on the tracer, analysis method, and local expertise. Anatomical size and functional capacity should not be assumed to be identical.

8.3 PET/CT in hepatic oncology

PET/CT combines tracer distribution with anatomical localization. Fluorine-18 fluorodeoxyglucose reflects glucose-related metabolism and can support staging or restaging of selected malignancies involving the liver. Its usefulness depends on the primary tumor's biology and the clinical decision being considered.

FDG uptake in HCC is variable, and well-differentiated tumors may be poorly conspicuous against normal hepatic background activity. Routine FDG PET is therefore not a substitute for liver-specific multiphasic CT or MRI and is not recommended by AASLD as routine HCC staging. Selected applications require a clearly defined question. [67]

For metastases, PET may provide valuable information about extrahepatic disease or metabolic response, but small hepatic lesions can remain below its effective resolution. Dedicated liver MRI may still be required when the number and distribution of small deposits determine surgical planning. A negative PET result does not exclude every metastasis.

Tracer choice matters in neuroendocrine disease and other tumors with biological targets different from glucose metabolism. Hybrid imaging should be selected according to tumor type and the intended therapeutic pathway rather than described as a uniform test for all hepatic malignancies.

8.4 SPECT/CT and PET/MRI

SPECT/CT adds anatomical localization to gamma-emitting tracer studies. It can improve confidence in assigning uptake to a specific structure and in interpreting regional function. The CT component may serve localization or a diagnostic purpose, with corresponding differences in protocol and radiation dose.

PET/MRI combines molecular information with MRI's tissue contrast, diffusion assessment, and other liver-specific techniques. It can provide a comprehensive examination in selected oncological settings and avoids the CT component of exposure. The radiopharmaceutical still produces ionizing radiation, so PET/MRI is not a radiation-free examination.

The added value of a hybrid system must be demonstrated for the actual indication. Longer examinations, cost, availability, motion-related misregistration, and technical complexity can offset potential advantages. More information does not automatically mean a better clinical decision unless the additional findings are reliable and relevant.

8.5 Limitations and selective use

Nuclear medicine has lower anatomical spatial resolution than dedicated cross-sectional imaging and may be affected by physiological background uptake, partial-volume effects, and inflammatory activity. Its results require correlation with anatomy and clinical history.

Radiation arises from the tracer and, in PET/CT or SPECT/CT, from the CT component. Exposure varies substantially with the protocol. Pregnancy, breastfeeding, repeated examinations, and the availability of alternatives require the appropriate tracer-specific procedures.

The strongest clinical rationale is a functional or molecular question that cannot be adequately answered by conventional anatomy alone. Research applications in diffuse inflammation or fibrosis are promising but should be distinguished from established routine assessment of steatosis and chronic fibrosis.

Chapter 9. Artificial Intelligence and Radiomics

9.1 Computational analysis in liver imaging

Artificial intelligence includes methods that learn associations from data and apply them to detection, classification, measurement, or prediction. In hepatic imaging, potential tasks include liver and lesion segmentation, steatosis detection, image reconstruction, enhancement analysis, and prediction of selected clinical outcomes. The source thesis discusses these developments through recent reviews and illustrative reports. [27–36]

Machine-learning systems may use explicitly selected variables, while deep-learning systems can learn image representations directly. Neither approach is inherently reliable simply because it processes a large dataset or produces a high numerical accuracy. The quality of the labels, study design, validation, and intended clinical use are decisive.

Automated segmentation can reduce the time needed to calculate liver volume, identify anatomical regions, and support treatment planning. Detection tools may help draw attention to subtle lesions, while quantitative analysis may reduce some forms of observer variation. These benefits remain task-specific and must be evaluated within the workflow in which the system will be used.

9.2 Steatosis, fibrosis, and lesion assessment

Computational methods have been investigated for identifying steatosis from ultrasound, CT, and MRI. Their potential includes analysis of subtle texture differences and consistent quantification. The clinical meaning depends on the reference standard: agreement with another imaging label is different from agreement with biopsy, quantitative MRI, or an outcome-based definition.

Fibrosis prediction presents further challenges because imaging features can also reflect inflammation, congestion, and acquisition differences. A model trained on a narrow disease group may perform poorly in another etiology. Its apparent ability to predict stage must be tested against clinically relevant alternatives and an appropriate reference standard.

For focal lesions, models may analyze arterial enhancement, washout, morphology, diffusion, or hepatobiliary features. CEUS applications can incorporate temporal information, while MRI models may combine multiple sequences. However, small, highly selected datasets can exaggerate apparent performance, especially when the controls do not reflect the difficult cases encountered in routine practice.

Claims that a model exceeds expert performance should be interpreted according to the study's reader comparison, case selection, and independent test set. The source's recent AI references contain incomplete records, so specific claims of superiority are not treated here as established clinical evidence.

9.3 Radiomic features and workflow

Radiomics converts medical images into quantitative features that describe intensity, shape, texture, and related properties. A conventional workflow includes image acquisition, region segmentation, preprocessing, feature extraction, feature selection, model development, and validation.

The resulting measurements may capture heterogeneity that is difficult to summarize visually. In liver research, investigators have explored relationships with fibrosis, inflammatory activity, tumor grade, vascular invasion, and recurrence after treatment. These associations are hypotheses or validated results within particular datasets; a feature does not become a clinical biomarker simply because it correlates with pathology in a single study.

Tumoral and peritumoral features can be combined with clinical and laboratory variables. This approach may improve prediction when each component contributes independent information. It can also introduce overfitting if the number of variables is large relative to the number of patients or events.

Ultrasound radiomics offers an attractive route because the underlying modality is accessible. Its reproducibility is influenced by transducer, gain, depth, compression, viewing plane, and other settings. CT and MRI features are likewise affected by acquisition and reconstruction choices, so standardization is a prerequisite for transportable results.

9.4 Validation, reproducibility, and clinical utility

A model should be tested on data separated from development at the patient level. Repeated examinations or multiple lesions from one patient can create leakage if they appear in both training and test sets. External multicenter evaluation assesses whether performance persists across equipment, protocols, populations, and disease prevalence.

Discrimination measures such as area under the receiver-operating-characteristic curve are insufficient alone. Calibration, clinically relevant sensitivity and specificity, failure modes, and comparison with current practice are also important. A model that discriminates well may still overestimate risk or cause unnecessary downstream examinations.

Reproducibility requires attention to segmentation variation and feature stability. Transparent reporting of acquisition, preprocessing, model development, and validation supports assessment and replication. An explanation map may show image regions associated with a prediction, but it does not independently establish biological causality or freedom from bias.

The final test is clinical utility: whether the system improves decisions, workflow, or patient outcomes without unacceptable new errors. Computational tools should support accountable clinical interpretation. The radiologist retains responsibility for recognizing inappropriate inputs, technical limitations, and discrepancies with the clinical context.

9.5 Future direction

The most plausible direction is integration of quantitative imaging with clinical, laboratory, and molecular information. Such models may support risk assessment and longitudinal monitoring, while reconstruction and segmentation tools may improve operational efficiency.

The transition from research performance to routine use requires prospective evaluation, quality monitoring, and evidence that the tool remains reliable after implementation. The promise of AI and radiomics should therefore be expressed in terms of specific validated tasks and unresolved questions rather than broad claims that they already replace established diagnostic pathways.

Chapter 10. Comparative Assessment and Diagnostic Pathways

10.1 Principles of comparison

A comparison of imaging modalities is meaningful only when the clinical task is defined. Detection of steatosis, diagnosis of a focal mass, staging of cancer, measurement of fibrosis risk, and demonstration of active bleeding are different tasks with different standards of success.

Sensitivity describes the proportion of affected cases correctly identified, while specificity describes the proportion of unaffected cases correctly classified. Predictive values also depend on prevalence and the patient's pre-test probability. Reported performance varies with lesion size, etiology, disease stage, protocol, reader expertise, and the reference standard.

A table assigning a single sensitivity and specificity to each modality across all liver diseases is therefore misleading. The broad percentages in the original thesis are not carried forward as universal estimates. The comparison below emphasizes indication-specific strengths and limitations, with a clearly identified numerical example for HCC.

10.2 Diagnostic performance in context

Conventional ultrasound is valuable for initial assessment and detects pronounced steatosis more reliably than mild disease. It also demonstrates advanced morphological changes and biliary dilatation. Its lesion-detection performance declines with poor acoustic access, small lesion size, and a heterogeneous background.

CEUS improves targeted lesion characterization by displaying perfusion continuously. Elastography answers a separate question about mechanical stiffness. Neither should be considered merely a more accurate version of every aspect of conventional ultrasound: each adds a defined type of information.

Multiphasic CT combines rapid anatomical coverage with enhancement assessment. MRI adds multiple tissue mechanisms and may improve detection of small lesions or characterize abnormalities that remain indeterminate on CT. However, a high-quality CT can be more useful than a motion-degraded MRI, and broad theoretical advantages do not ensure better performance in every patient.

As an indication-specific example, the 2023 AASLD HCC guidance cites pooled diagnostic sensitivity of approximately 82% for MRI and 66% for CT, with similar specificity of approximately 92% and 91%, respectively. These are literature-derived estimates for HCC diagnosis, not guaranteed values for individual centers or all lesion sizes; both modalities are recommended options. [67]

Nuclear medicine performance depends particularly on tracer biology. FDG PET has important limitations in well-differentiated HCC and small lesions. Its value often lies in systemic staging or a defined functional question rather than in primary detailed characterization of the liver.

Table 1. Principal roles and practical limitations of hepatic imaging techniques.

Technique	Principal information	Major strengths	Main limitations	Common clinical role
B-mode ultrasound	Morphology and echotexture	Accessible, real-time, no ionizing radiation	Operator and acoustic-window dependence; limited tissue specificity	Initial assessment, duct dilatation, ascites, surveillance
Doppler ultrasound	Direction and pattern of vascular flow	Hemodynamic assessment at the bedside	Angle and settings affect results; slow flow may be missed	Portal and hepatic venous disease; vascular follow-up
Ultrasound elastography	Tissue stiffness	Rapid and repeatable fibrosis risk assessment	Confounded by inflammation, cholestasis, congestion, and technique	Non-invasive staging and longitudinal evaluation
CEUS	Targeted dynamic perfusion	Continuous arterial observation; no ionizing radiation; useful in renal impairment	Limited field and acoustic access; not whole-body staging	Focal-lesion problem-solving and selected treatment assessment
Multiphasic CT	Anatomy and enhancement	Fast, broad coverage, strong vascular and trauma assessment	Radiation; contrast considerations; limited early fibrosis assessment	Oncology staging, acute complications, trauma, intervention
Multiparametric MRI	Tissue composition, diffusion, and enhancement	Detailed characterization, fat and iron measurement, no ionizing radiation	Motion, examination time, cost, access, device screening	Small or indeterminate lesions; quantitative and biliary assessment
MRE	Regional stiffness mapping	Larger sampling volume; integration with MRI	Specialized equipment; stiffness confounders; sequence limitations	Fibrosis assessment when additional evaluation is needed
Scintigraphy/SPECT	Tracer-specific function	Functional or regional assessment	Limited anatomical resolution and tracer-specific interpretation	Selected hepatobiliary or preoperative functional questions
PET/CT or PET/MRI	Molecular information plus anatomy	Systemic oncological evaluation in selected tumor types	Tracer dependence, small-lesion limits, expense, radiation	Selected staging, restaging, or treatment-response questions

10.3 Safety, access, and resource use

Safety must be considered together with the expected diagnostic benefit. Ultrasound and non-contrast MRI avoid ionizing radiation, but an inadequate examination can delay diagnosis. CT may be the most appropriate test when rapid assessment of bleeding or acute abdominal disease is required.

Repeated CT phases contribute to cumulative exposure, making protocol justification important in long-term follow-up. MRI avoids this exposure but introduces other considerations, including device safety, motion, and the use of gadolinium when necessary. Microbubble, iodinated, and gadolinium agents have different risk profiles and should not be grouped under a single undifferentiated category of contrast risk.

Renal impairment modifies decision-making but does not uniformly prohibit enhanced imaging. The required diagnostic information, renal status, agent, alternatives, and urgency should guide the choice. A non-contrast study is preferable only when it can adequately answer the question or offers the best balance in the specific circumstances.

Availability and cost influence the pathway. A sequential approach often uses an accessible initial examination followed by a targeted higher-information study. Repeating several inconclusive examinations can be less efficient than selecting a definitive problem-solving technique early when the initial limitation is already evident.

10.4 Diffuse steatosis and fibrosis

The first question is whether the aim is to detect fat, quantify fat, assess fibrosis risk, or identify complications. Conventional ultrasound often establishes an initial impression of steatosis. MRI-PDFF is more appropriate when an accurate quantitative measurement will change assessment or monitoring.

Fibrosis evaluation uses clinical information, blood-based scores, and elastography. MRI-PDFF should not be used as a surrogate for fibrosis stage. CT may show advanced morphology but is not a preferred standalone test for early fibrosis or repeated fat quantification.

If non-invasive findings are discordant, technical quality and possible confounders should be reviewed. A second validated technique, MRE, specialist assessment, or selected biopsy may then be appropriate. This approach preserves the distinction between risk stratification and a definitive explanation of inflammatory histology.

10.5 Jaundice, cholestasis, and ductal disease

Ultrasound generally begins the anatomical investigation of jaundice when obstruction is suspected. It assesses the gallbladder, visible ducts, and liver and may identify stones or a mass. The absence of visible dilatation does not resolve every case, particularly early or intermittent obstruction.

MRCP is useful for detailed ductal mapping, suspected strictures, and unresolved ductal stones. CT is often selected when a pancreatic, hilar, or other malignant cause, acute complication, or broad abdominal assessment is the priority. The next study should follow the question left unanswered by the initial examination.

When drainage, stone extraction, or tissue sampling is required, an endoscopic or interventional procedure may follow. Diagnostic imaging should support the appropriate intervention rather than lead to repeated non-therapeutic examinations without resolving the obstruction.

10.6 Incidental focal lesions

A focal observation is interpreted differently in a low-risk person, a patient with chronic liver disease, and a patient with known malignancy. A clearly simple cyst may need no further characterization, while a solid or complex lesion requires a context-specific approach.

MRI provides detailed tissue characterization, especially for small or indeterminate lesions and suspected FNH or adenoma. Multiphasic CT is a practical alternative and may be particularly useful when staging is also needed. CEUS can resolve a targeted question when the lesion is accessible or when dynamic arterial information is missing.

The report should explain the level of confidence and the reason for further assessment. Describing a lesion as indeterminate is useful only if its significance and an appropriate next step are made clear in relation to the patient's risk.

10.7 HCC surveillance and diagnostic recall

Surveillance applies to eligible at-risk populations and commonly uses ultrasound at six-month intervals, with AFP according to the adopted guideline. A technically limited surveillance examination should be recognized explicitly. Selected patients may require an alternative surveillance strategy based on their visualization quality and risk. [67]

A suspicious observation leads to a diagnostic pathway that depends on its size and other clinical information. Dedicated multiphasic CT or contrast-enhanced MRI characterizes lesions that meet the criteria

for further assessment. Very small observations may require short-interval reassessment rather than being treated as definitively diagnosable tumors.

LI-RADS standardizes eligible high-risk patient examinations; it is not a universal classification for every liver lesion in every patient. Major and ancillary features, contrast agent, technical adequacy, and population eligibility all affect interpretation. Indeterminate or non-HCC malignant categories require a different response from a definitive imaging diagnosis.

Surveillance, diagnostic characterization, staging, and treatment-response assessment are separate steps. A modality chosen for one step should not automatically be assumed to be optimal for the next.

10.8 Metastases and surgical planning

CT is commonly used for systemic staging and provides a broad assessment of extrahepatic disease. Dedicated liver MRI is valuable when small lesions remain uncertain or when the precise intrahepatic burden determines eligibility for resection or ablation.

The imaging report should provide the distribution of lesions, relevant segmental locations, vascular or biliary involvement, and findings affecting the future liver remnant. A change in lesion count can be more clinically important than a small change in the diameter of a dominant mass.

PET contributes when the tumor type and intended decision support its use, particularly for selected extrahepatic staging questions. It does not replace MRI or appropriately timed CT for every small hepatic deposit. Multidisciplinary review connects the findings to the treatment strategy.

10.9 Infection, vascular disease, and trauma

Ultrasound may identify an abscess and provide guidance for accessible drainage. CT is valuable for mapping complex infection, gas, associated abdominal disease, and intervention routes. MRI is reserved for selected unresolved characterization questions or other specific indications.

Doppler ultrasound usually provides the initial hemodynamic assessment of portal and hepatic venous disease. CT or MRI follows when the extent, cause, or full vascular anatomy must be established or the acoustic examination is inadequate.

In significant trauma, CT provides comprehensive assessment in sufficiently stable patients. FAST serves a rapid bedside role but cannot exclude hepatic injury. MRI is generally not part of the immediate acute assessment. Physiological stability and urgent treatment needs take priority over completing an ideal imaging sequence.

Table 2. Indication-based selection of imaging examinations.

Clinical question	Common initial approach	Targeted next examination	Essential interpretive point
Suspected steatosis	Clinical assessment and ultrasound	MRI-PDFF when quantification is needed	Fat quantity is distinct from inflammatory activity and fibrosis
Risk of advanced fibrosis	Blood-based score with elastography as appropriate	MRE or selected further assessment for discordance	Stiffness is affected by non-fibrotic processes
Suspected biliary obstruction	Ultrasound	MRCP for ducts; CT for masses or acute complications	A non-dilated ductal system does not exclude every obstruction
Indeterminate focal lesion	Review initial imaging and clinical risk	MRI, multiphasic CT, or targeted CEUS	Background liver and cancer history change the differential
HCC surveillance finding	Guideline-based recall assessment	Dedicated CT or MRI when indicated	Small observations and technical limitations affect the pathway
Metastatic disease planning	Systemic contrast-enhanced CT	Liver MRI for precise intrahepatic mapping	Lesion number and distribution can determine resectability
Portal or hepatic venous disease	Doppler ultrasound	CT or MR vascular assessment	Confirm absence of flow with adequate technique
Complex hepatic infection	Ultrasound or CT according to urgency	CT mapping and image-guided intervention	Abscess and necrotic tumor can overlap in appearance
Significant hepatic trauma	Resuscitation and rapid bedside assessment	Contrast-enhanced CT when sufficiently stable	Imaging must fit the physiological condition
Regional functional question	Define the functional endpoint	Tracer-specific scintigraphy/SPECT when indicated	Volume alone does not establish functional reserve

10.10 Integrated interpretation and reporting

Useful reporting begins with the indication and technical adequacy. It distinguishes observations from diagnoses and states relevant limitations. For a lesion, location, size, composition, enhancement, diffusion behavior, and relation to vessels or ducts should be integrated into a coherent conclusion.

Quantitative results require the technique and appropriate context. A stiffness value, PDFF measurement, or iron estimate should not appear as an isolated number without explaining the property measured and the reliability of the acquisition. Serial comparison should identify meaningful differences in method or quality.

The best comparative strategy is not the accumulation of modalities. It is the selection of complementary examinations that progressively reduce uncertainty. Clinical history, laboratory tests, pathology when indicated, and multidisciplinary discussion remain essential to converting imaging information into management.

Chapter 11. Conclusions

The liver's complex vascular, biliary, and metabolic organization explains both the diversity of its diseases and the need for multiple imaging approaches. Each technique samples a different aspect of this organization. Conventional ultrasound depicts morphology in real time; Doppler evaluates flow; elastography measures stiffness; CEUS demonstrates targeted perfusion; CT provides rapid anatomical and vascular coverage; MRI combines tissue characterization with quantitative and biliary techniques; and nuclear medicine evaluates tracer-specific function or molecular activity.

No modality is universally optimal. Ultrasound remains an accessible initial and surveillance tool, but limited visualization and non-specific appearances must be acknowledged. Elastography has broadened non-invasive fibrosis assessment, provided that stiffness is interpreted with its confounders and method-specific criteria. CEUS is a valuable targeted problem-solving technique, particularly when continuous arterial observation or an alternative contrast approach is useful.

CT is especially effective for acute complications, significant trauma, systemic oncological staging, and comprehensive vascular mapping. Its speed and coverage must be balanced against radiation exposure and the requirements of iodinated contrast. MRI offers detailed characterization of focal lesions and quantitative assessment of fat, iron, and stiffness, together with non-invasive biliary mapping. Its usefulness depends on an appropriate protocol and adequate examination quality.

Nuclear medicine and hybrid imaging have selective roles defined by tracer biology and the clinical endpoint. They complement, rather than replace, dedicated liver CT and MRI. AI and radiomics may improve measurement, workflow, and prediction, but their claims require careful validation and evidence of clinical utility.

The central conclusion is an indication-based and integrated approach. The modality should be chosen according to whether the clinician needs detection, characterization, staging, functional assessment, intervention planning, or follow-up. The final diagnosis and management decision arise from the synthesis of imaging with the clinical, biochemical, and, when necessary, pathological evidence.

Abbreviations

Abbreviation	Meaning
ADC	Apparent diffusion coefficient
AFP	Alpha-fetoprotein
AI	Artificial intelligence
ALT	Alanine aminotransferase
AST	Aspartate aminotransferase
CEUS	Contrast-enhanced ultrasound
CT	Computed tomography
DECT	Dual-energy computed tomography
DILI	Drug-induced liver injury
DWI	Diffusion-weighted imaging
ERCP	Endoscopic retrograde cholangiopancreatography
FAST	Focused assessment with sonography in trauma
FDG	Fluorodeoxyglucose
FIB-4	Fibrosis-4 index
FNH	Focal nodular hyperplasia
GBCA	Gadolinium-based contrast agent
HCC	Hepatocellular carcinoma
HU	Hounsfield unit
LI-RADS	Liver Imaging Reporting and Data System
MASH	Metabolic dysfunction-associated steatohepatitis
MASLD	Metabolic dysfunction-associated steatotic liver disease
MRCP	Magnetic resonance cholangiopancreatography
MRE	Magnetic resonance elastography
MRI	Magnetic resonance imaging
NAFLD	Non-alcoholic fatty liver disease
NASH	Non-alcoholic steatohepatitis
PDFF	Proton density fat fraction
PET	Positron emission tomography
PSC	Primary sclerosing cholangitis
SPECT	Single-photon emission computed tomography
SWE	Shear-wave elastography
US	Ultrasound
VCTE	Vibration-controlled transient elastography

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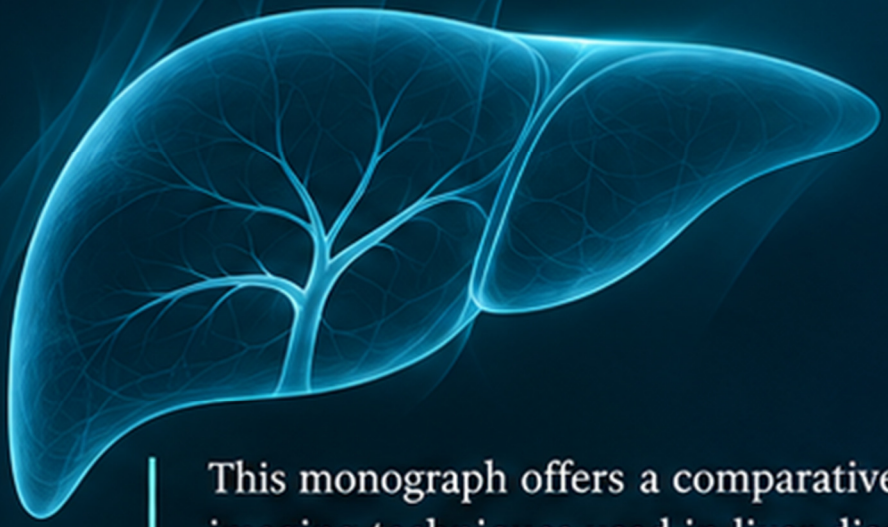
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This monograph offers a comparative overview of the principal imaging techniques used in liver diseases, linking anatomical foundations with diagnostic methods and clinical applications. It examines ultrasound, computed tomography, magnetic resonance imaging, and nuclear medicine, while also addressing elastography, contrast-enhanced ultrasound, artificial intelligence, radiomics, and hybrid imaging. Designed as an academic reference, the book supports clinicians, imagers, and trainees involved in hepatology and abdominal imaging.

