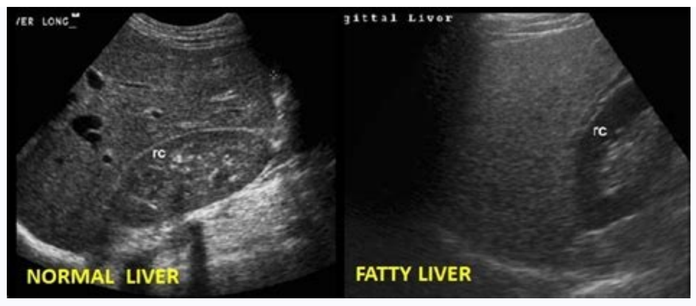
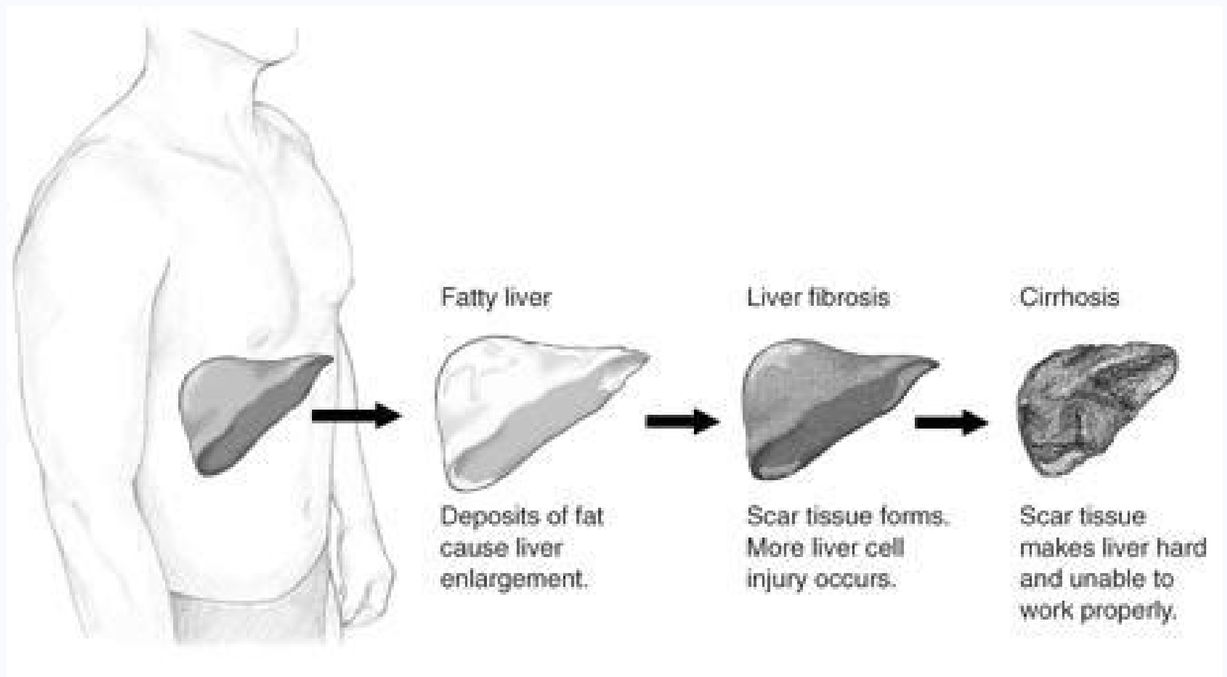
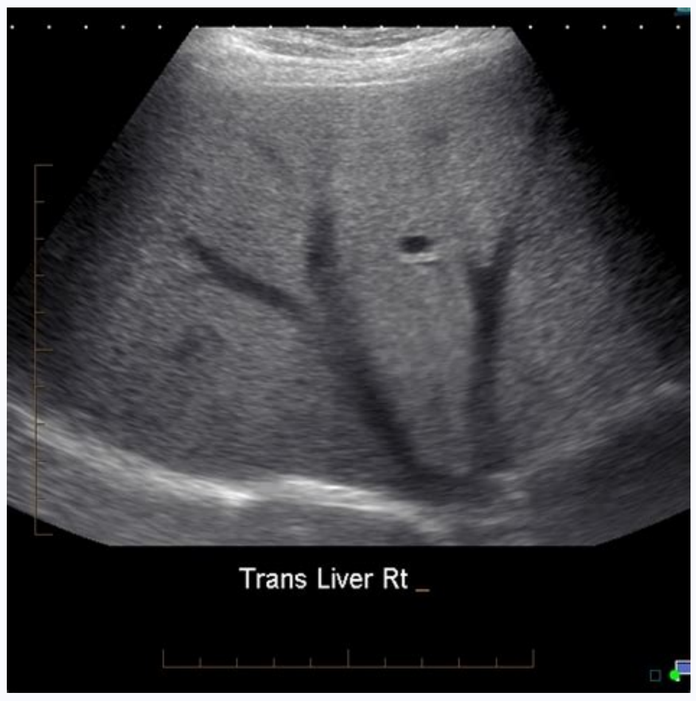
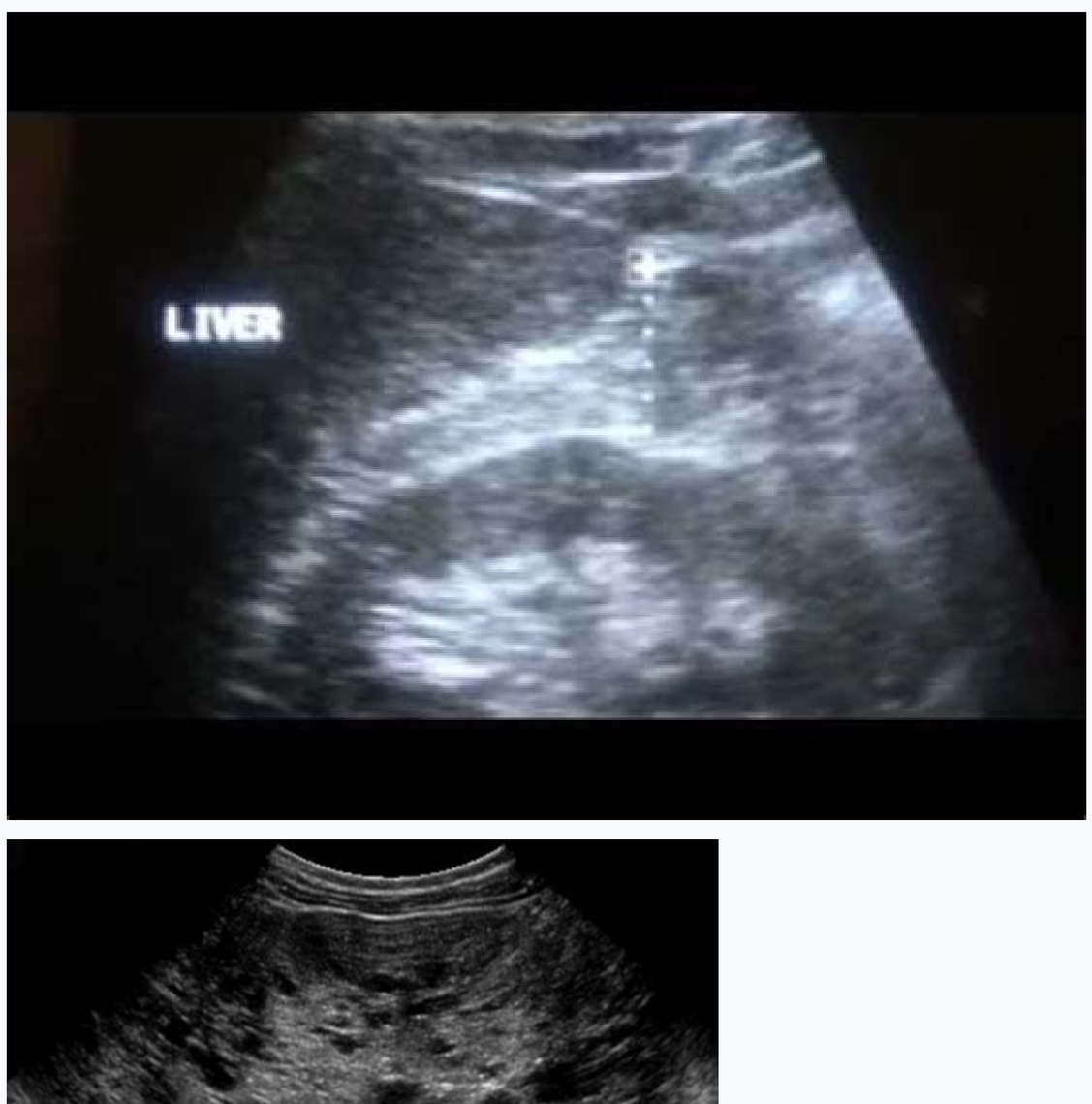
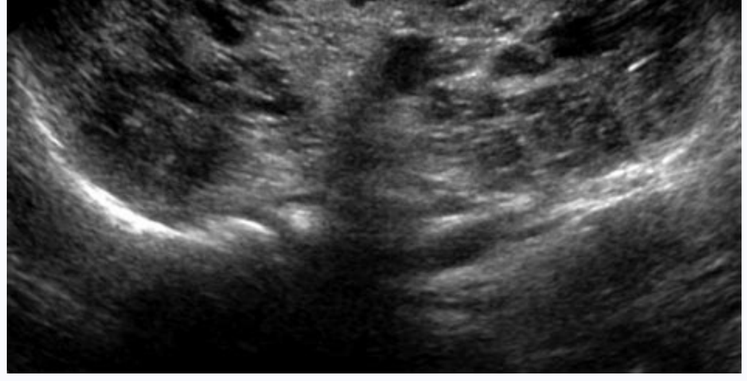
Liver cirrhosis ultrasound report











Can liver problems be detected by ultrasound. What does liver cirrhosis look like on ultrasound. Is liver cirrhosis seen on ultrasound. Does ultrasound show liver cirrhosis.

The concept of ‘cirrhosis’ is evolving and it is now clear that compensated and decompensated cirrhosis are completely different in terms of prognosis. Furthermore, the term ‘advanced chronic liver disease (ACLD)’ better reflects the continuum of histological changes occurring in the liver, which continue to progress even after cirrhosis has developed, and might regress after removing the etiological factor causing the liver disease. In compensated ACLD, portal hypertension marks the progression to a stage with higher risk of clinical complication and requires an appropriate evaluation and treatment. Invasive tests to diagnose cirrhosis (liver biopsy) and portal hypertension (hepatic venous pressure gradient measurement and endoscopy) remain of crucial importance in several difficult clinical scenarios, but their need can be reduced by using different non-invasive tests in standard cases. Among non-invasive tests, the accepted use, major limitations and major benefits of serum markers of fibrosis, elastography and imaging methods are summarized in the present review. Classically, cirrhosis is defined by its histological hallmark findings on liver biopsy (regenerative nodules surrounded by fibrotic tissue) and is considered as the final evolution stage of any progressive liver disease, irrespective of its etiology. The advances in diagnostic methods allow now early diagnosis, even before the development of complications, which are mostly related to development of portal hypertension [1]. Recently, with the development of new and very effective treatments, especially in the viral-related cirrhosis scenario, there is increasing evidence that cirrhosis can regress and that histological improvement is associated with better prognosis [2]. However, in the particular case of direct acting antiviral (DAA) treatment of hepatitis C virus (HCV), some data suggest that the complications of portal hypertension can occur even after sustained virological response (SVR) and the risk of hepatocellular carcinoma (HCC) is not abolished [3,4]. Therefore, the international expert consensus currently suggests continuing screening and surveillance of these patients according to the standard guidelines used for portal hypertension and HCC, and it is still unknown whether these patients should be managed and followed according to different schemes.The natural history of cirrhosis is marked by the transition from the compensated stage (with good prognosis) to the occurrence of decompensation events, such as ascites, variceal bleeding, jaundice and hepatic encephalopathy. If the diagnosis of cirrhosis is relatively straightforward during the decompensated stage when the treatment may be problematic, on the contrary, diagnosing cirrhosis while it is still in the compensated stage is more challenging. The progression of fibrosis parallels the increase in portal pressure and, frequently, patients with severe fibrosis in the pre-cirrhotic stage have a hepatic venous pressure gradient (HVPG) >5 mmHg [5]. Since chronic liver disease is a continuum, and due to the inhomogeneity of fibrosis within the liver [6], the border between severe fibrosis and compensated cirrhosis is often unclear and, recently, the Baveno VI consensus recommended that this clinical scenario including severe fibrosis and initial cirrhosis should be named compensated advanced chronic liver disease (cACLD) [7].Moreover, the concept of diagnosis of cirrhosis is changing from the documentation of histological F4 fibrosis to the identification of patients truly at risk of developing complications. It has been clearly demonstrated that the onset of clinically significant portal hypertension (defined as HVPG ≥10 mmHg) marks the progression to a stage at risk of clinical complications. In this scenario, non-invasive methods able to mirror the haemodynamic threshold play an important role. For instance, according to the recommendations of the last Baveno consensus conference, liver stiffness (measured by transient elastography) over 21 kPa is accurate enough to identify patients with clinically significant portal hypertension, so allowing a simple and readily available risk stratification when more sophisticated and exact methods are not available [7]. The natural history of chronic liver disease eventually leading to cACLD and complications of cirrhosis is represented in Figure 1, together with the main tests used for its diagnosis, staging and risk stratification. Open in new tabDownload slide Natural history of diagnostic on-invasive diagnostic tests in compensated advanced chronic liver disease. The most appropriate timeframe for using different techniques in order to maximize the information for clinical use is given. Combination of different non-invasive unrelated tests can further improve the amount of information retrieved and reduce the risk of false-positive and false-negative results.In this manuscript, we review the diagnostic performance of gold-standard invasive methods and surrogate non-invasive methods used in this setting in light of the new concept of cACLD. Gold-standard diagnostic methods for cirrhosis and portal hypertension Liver biopsy is still considered the gold-standard diagnostic method to identify the typical features of cirrhosis. Alternative diagnostic methods have been validated in comparison to liver biopsy and have a good diagnostic accuracy for the diagnosis of cirrhosis. As a consequence, the use of liver biopsy has dramatically decreased in the last 10 years; nonetheless, it remains a crucial diagnostic tool when concomitant potential etiological factors for liver disease coexist and when the identification of features other than fibrosis leads to changes in the clinical management of patients, such as in the case of acute or chronic liver injury. Currently, the most important and frequent scenario that requires a mandatory liver biopsy is the differentiation between severe alcoholic hepatitis and decompensated alcoholic cirrhosis, because by now there are no specific clinical signs or non-invasive methods to differentiate between the two conditions [8]. Liver biopsy is largely used in patients with suspected liver cirrhosis of unknown aetiology in order to confirm the diagnosis and expand on its possible cause (e.g. indicating the distribution of fibrosis in the liver). It remains key also in the case of suspected non-alcoholic steatohepatitis (NASH)-related ACLD and in cholestatic and autoimmune chronic liver disease for which data regarding the diagnostic accuracy of non-invasive methods are scarce.Liver biopsy can be carried out from a percutaneous or a transjugular route. Percutaneous liver biopsy is done through a right intercostal space after or under ultrasound control, on local anaesthesia, using Menghini core-aspiration or Tru-cut automatic 16-gauge needles. Before the procedure, coagulation parameters should be checked (including platelet count and prothrombin time/international normalized ratio). The 50/50 rule (prothrombin time over 50% and platelet count over 50 × 109/L) is frequently used to consider the coagulation and platelet status acceptable. The contraindications for percutaneous liver biopsy include severe coagulopathy, biliary ducts dilatation, sepsis, ascites, suspicion of vascular lesions, hydatid disease or uncooperative patient [9]. Some of the contraindications (especially coagulopathy and ascites) are overcome by using a transjugular approach that carries lower haemorrhagic risk. The most frequent indications for liver biopsy are presented in Table 1 [9,10]. Table 1.The main indications for performing liver biopsy Percutaneous liver biopsy

. Transjugular liver biopsy . Diffuse liver diseases with multiple aetiologies Need for parallel measurement of hepatic venous pressure gradient (HVPG) Abnormal liver test from unknown origin Contraindications to percutaneous access (note that dilatation of the biliary tree is a contraindication for any liver biopsy) Non-alcoholic fatty liver disease Suspicion of severe alcoholic hepatitis Autoimmune hepatitis Acute liver failure of unknown aetiology Focal lesions Suspicion of non-cirrhotic portal hypertension Abnormal liver test in haematological patients Regarding the diagnostic performance of both approaches, although previously transjugular liver biopsy was considered inferior because of the use of thinner needles (18G), there is strong evidence suggesting that the two techniques are similar in terms of sample length and the number of complete portal spaces [11]. The greatest advantage of the transjugular route is that it allows concomitant HVPG measurement and multiple passes without increasing the risk of complications.Liver fibrosis and its patterns remain of paramount importance in risk stratification of patients, even in those who have fully established liver cirrhosis. Four main patterns of fibrosis development according to different aetiologies are described: (i) portal-to-central fibrosis distribution (characteristic to viral and autoimmune hepatitis); (ii) portal-to-portal distribution (specific for biliary diseases); (iii) perisinusoidal and pericellular distribution (metabolic diseases, alcoholic and non-alcoholic liver diseases) and (iv) central-to-central fibrosis distribution (for venous outflow obstruction such as Budd-Chiari syndrome) [12]. According to the different types of fibrosis distribution, the portal hypertension occurs earlier, as in the case of viral, autoimmune or Budd-Chiari syndrome, or later in the course of the disease, as in the case of metabolic diseases. Interestingly, even if sinusoidal portal hypertension develops later in the case of biliary diseases, due to the portal-to-portal distribution of fibrosis and development of porto-portal septa, there is an increased presinusoidal resistance that will increase portal pressure, so that the HVPG underestimates the value of the portal pressure gradient in patients with cholestatic liver disease.As the disease progress, the amount of fibrosis increases and in parallel the portal pressure rises [13] corresponding to worsening in the prognosis [14,15]. The Laennec sub-classification of cirrhosis [16] in three subclasses of stage 4: 4A—mild cirrhosis with thin septa and large nodules; 4B—at least two broad septa, but no very broad septa and less than half of biopsy length composed of minute nodules; 4C—very broad septum or more than half of biopsy length composed of minute nodules (micronodular cirrhosis) offers additional prognosis relevance [17,18]. Moreover, histological markers of fibrosis regression under therapy, named ‘hepatic repair complex’, can be observed, and consist of delicate and perforated fibrous septa; isolated and thick collagen fibres; delicate periportal fibrous spikes; portal tract remnants; hepatic vein remnants with prolapsed hepatocytes; hepatocytes within portal tracts or splitting septa; minute regenerative nodules; and aberrant parenchymal veins [19,20].Because the majority of complications are conditioned by portal hypertension occurrence, the measurement of HVPG has probably an important prognostic relevance that might exceed that of histological modifications. HVPG is through internal jugular vein, femoral vein or cubital vein access under local anaesthesia [21,22]. One of the hepatic veins is catheterized with a balloon catheter under fluoroscopic control. By the inflation of the balloon, the hepatic venous outflow is blocked and, at the end of 1–2 minutes, the pressure at the tip of the catheter equals that of the hepatic sinusoidal pressure and portal pressure, respectively. That represents the wedge hepatic venous pressure (WHVP). Free hepatic venous pressure (FHVP) is measured by deflating the balloon at 2–3 cm from the hepatic vein ostium, and usually its value is very close to the inferior vena cava. HVPG is the difference between WHVP and FHVP, and represents the pressure gradient that the portal blood flow has to exceed to pass through the liver. While some authors consider that FHVP should be substituted by the pressure in the inferior vena cava or right atrium pressure [23], HVPG loses its prognostic value if it is calculated to any other vessel except the liver vein, so that FHVP should be mandatorily used [24,25].Due to the relative invasiveness and the lack of wide diffusion of the method, HVPG is considered by many as only a research method to assess portal pressure but, in the authors’ opinion, it should be considered a crucial and readily available, mature method to achieve clinically important information. In clinical practice is a useful technique to make the differential diagnosis in case of clinical signs of portal hypertension, especially if cirrhosis is not obvious on non-invasive techniques such as ultrasound or transient elastography. HVPG is a safe technique that has no absolute contraindications [26]. In patients requiring transjugular liver biopsy, the measurement of HVPG adds only a few minutes to the procedure but provides very relevant haemodynamic information. In our practice, whenever liver biopsy is indicated in patients with cACLD, we prefer the transjugular approach to obtain both histological findings and HVPG measurement.HVPG is probably the most validated tool for assessing prognosis in cACLD. In Table 2 are presented the most relevant clinical end-points with which HVPG was associated [1,5,24,27–33]. All this body of evidence indicates HVPG to be the best tool for assessing the prognostics of patients with cACLD. Table 2.Different thresholds of hepatic venous pressure gradient (HVPG) correlated with clinical end-points in compensated advanced chronic liver disease (cACLD) HVPG . Clinical end-points . 6 mmHg Progression of chronic viral hepatitis [5] High risk of recurrence after liver transplantation [27] >10 mmHg Clinically significant portal hypertension >10 mmHg Oesophageal varices development [28,29] Ascites [1] Decompensation [1] Hepatocellular occurrence [30] Decompensation after hepatic resection [31] >12 mmHg Oesofageal varices bleeding >16 mmHg High mortality [24] >20 mmHg Failure to control bleeding [32] >22 mmHg High mortality in severe alcoholic hepatitis [33] An ideal diagnostic and prognostic method would reflect also the changes under therapy. A decrease in HVPG

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