Review Article

An Overview on Non-invasive Assessment of Cirrhosis

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Abstract

Chronic liver disease is a long-term degradation of liver processes, such as the production of clotting factors and the detoxification of toxic metabolic products. It is a constant cycle of liver parenchyma inflammation, destruction, and regeneration that leads to cirrhosis and fibrosis. the last phase of it being cirrhosis, characterized by distortion of hepatic architecture, the development of extensive nodules. Additionally, it is mostly in a cirrhotic liver that there is a development of hepatocellular carcinoma (HCC). Cirrhosis and HCC are responsible for 3.5% of all fatalities globally. Nevertheless, cirrhosis may be prevented since the diseases that most often cause it to develop slowly and can be prevented and treated. Therefore, early detection and adequate evaluation of the disease are important for preventing the progress or even reversing some of the effects of the disease. To provide an adequate review discussing the evaluation of chronic liver disease cases and cirrhosis in particular. The following keys were used in the mesh of the PubMed database for the choosing of articles. Characteristic findings on clinical examination, laboratory testing, and auxiliary studies are used to diagnose the disease. If firmly established through imaging investigations and medical symptoms that cirrhosis has been diagnosed, a liver biopsy is unnecessary, if not even contraindicated.

Keywords: Chronic liver disease, Non-invasive assessment, Cirrhosis, Management

Introduction

Chronic liver disease is a long-term degradation of liver processes, such as protein production like clotting factors, the secretion of bile, and the cleansing of toxic metabolic products. Chronic liver disease is a constant cycle of liver parenchyma inflammation, destruction, and regeneration that leads to fibrosis and cirrhosis. Chronic liver disease has a wide range of etiologies, including toxins, long-term alcohol abuse, infection, autoimmune illnesses, hereditary, and metabolic problems.

Cirrhosis is the last stage of chronic liver disease, characterized by distortion of hepatic architecture, vascular rearrangement, extensive nodules development, neo-angiogenesis, and extracellular matrix deposition. The recruitment of stellate cells and fibroblasts, which results in fibrosis, is the fundamental process of fibrosis and cirrhosis at a cellular level, while hepatic stem cells are required for parenchymal regeneration.

Chronic liver disease is a major cause of mortality, particularly in developing countries. the prevalence of the chronic liver disease has been on the rise of late. Atherosclerosis, chronic viral hepatitis, including hepatitis B and C, non-alcoholic fatty liver disease (NAFLD), and hemochromatosis are the most common chronic liver diseases in the industrialized world. About 4.5 million individuals in the United States have chronic liver disease and cirrhosis, accounting for 1.8% of the adult population. Chronic liver disease and cirrhosis caused 41,473 fatalities (12.8 deaths per 100,000 population).

Additionally, it is mostly in a cirrhotic liver that there is a development of HCC cases. HCC and cirrhosis are responsible for 3.5% of all fatalities globally. Nevertheless, cirrhosis may be prevented since the diseases that most often cause it to develop slowly and can be prevented and treated [1-3]. Therefore, early detection and adequate evaluation of the disease are important for preventing the progress or even reversing some of the effects of the disease. So, we aim in this study to provide an adequate review discussing the evaluation of chronic liver disease cases in general and cirrhosis in particular.

Materials and Methods

The following keys were used in the mesh of the PubMed database for the identification of articles ((“cirrhosis"[Mesh])

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AND (“evaluation”[Mesh]) OR (“chronic liver disease"[Mesh])).

Concerning the inclusion criteria, the articles were chosen because they covered the following topics: cirrhosis, non-invasive evaluation.

All other articles not having one of these topics as their chief endpoint were excluded.

Results and Discussion

Cirrhosis Pathogenesis

Encapsulation or replacement of damaged tissue by a collagenous scar is referred to as fibrosis. The normal wound healing response is sustained in liver fibrosis, resulting in a pathological perpetuation of fibrogenesis (production and deposition of connective tissues). The advancement of Fibrosis is relative with a dependence on environmental and host variables as well as the etiology of the liver disease [2-4].

Cirrhosis is a more aggressive form of liver fibrosis that is accompanied by hepatic vascular disruption. The portal and arterial blood supplies are shunted straight into the central veins (hepatic outflow), impairing the interchange amid hepatic sinusoids and the surrounding liver parenchyma, i.e., hepatocytes. Fenestrated endothelia line the hepatic sinusoids, which are supported by a layer of the Disse space which is porous connective tissue containing hepatic stellate cells and some mononuclear cells. Hepatocytes line the other side of the Disse space, performing the majority of known liver activities. Through the process of capillarization, there is a combination of Disse space with scar tissue in the cirrhotic liver, whereby there is a loss of endothelial fenestrations [2].

Cirrhosis is defined histologically by vascularized fibrotic septa that connect portal tracts and to central veins, making up a hepatocyte island that is surrounded by fibrotic septa while lacking a central vein. Cirrhosis has three primary physiological effects: diminished hepatocyte function, increased intrahepatic resistance (portal hypertension), and HCC development. Cirrhosis-related circulatory abnormalities (splanchnic vasodilation, vasoconstriction and hypoperfusion of the kidneys, salt and water retention, and raised cardiac output) are closely connected to hepatic vascular changes and the consequent portal hypertension. vascular deformation and its cause, cirrhosis had long been thought to be permanent, however new evidence suggests that cirrhosis can be regressed or even reversed [5, 6].

The most common causes of chronic liver disease in the Western world are alcohol addiction and to a lesser degree, viral hepatitis [7]. When compared to these primary etiological variables, other causes such as chronic cholestasis, venous outflow obstruction, metabolic disorders such as hemochromatosis or Wilson's disease, drug-induced abnormalities, or autoimmune illnesses are rare. Portal hypertension, ascites, variceal hemorrhage, hepatic encephalopathy, disrupted glucose metabolism, and vitamin and trace element deficiencies are all common consequences of chronic liver diseases, regardless of their etiology. Hepatocellular damage, followed by hepatocyte degeneration or necrosis, is central to the pathophysiology of cirrhosis or fibrosis. In most cases, harm is the result of a complicated process rather than a single toxic principle. This process involves both parenchymal and nonparenchymal cells in the liver. Hepatocyte necrosis causes Kupffer cells to activate and macrophages to migrate. Chemotaxis attracts neutrophils, which causes hepatocyte injury. Cytokines like interleukin-I also recruit and activate T lymphocytes (fL-I). ITO or fat storage cells, which are converted into myofibroblasts, also attract and partially activate fibroblasts. These cells are in charge of increasing collagen and other extracellular matrix component production [2].

Furthermore, in the mammalian body, ITO cells are the primary physiological location for retinol storage. Only 1 to 2% of the connective tissue is in a normal liver. Cirrhosis patients have a total quantity of extracellular matrix proteins that reaches a maximum of around 50% of total proteins. In addition, the kinds of proteins are altered. Collagen types IV and VI, in particular, are considerably elevated [7].

Cirrhosis Burden

The 11th and 16th most prevalent reasons for death are Cirrhosis and liver cancer killing 1.16 million people 788,000 people worldwide each year, respectively [8]. They are responsible for 3.5% of all fatalities globally. Liver-related fatalities with cirrhosis and liver cancer ranked 13th and 20th respectively accounted for 3% of all deaths, among the major causes of death. This is considered an increase from 2000. When acute hepatitis (145,000 fatalities) and alcohol-use disorders are included, the burden is likely to be greater (129,000). According to these figures, liver disease is responsible for roughly 2 million deaths globally. Latin America and the Caribbean, as well as the Middle East and North Africa, had the highest proportion of regional fatalities due to liver disease, whereas South Asia and East Asia, and the Pacific had the highest absolute number of deaths. Cirrhosis mortality rates in Egypt, Moldova, and Mongolia are among the highest worldwide.

India contributes to one-fifth of all cirrhosis-related mortalities worldwide with its populace load, while China accounts for 11%. The death rate is rising in Central Asian nations and the Russian Federation. Mortality is rising in the United Kingdom, but falling in France and Italy. Cirrhosis affects more males than females worldwide, although the ratio is about equal in Moldova and Russia [9].

Cirrhosis has a variety of reasons: Alcohol and non-alcoholic fatty liver disease have surpassed viral hepatitis in the industrialized and western nations as the leading reasons, but hepatitis B remains a major cause in China and other Asian countries [10]. Cirrhosis is caused by viral hepatitis B and C in 99% in Mongolia, and 20% of patients have hepatitis B and C coinfection [11]. Chronic liver disease and cirrhosis are the 12th and 4th major causes of mortality in the United States, respectively, among individuals aged 45–64 years [8].

Morbidity

Patients with chronic liver disease have a significant economic burden and bad quality of life indicators, in addition to an increased chance of mortality [12]. Cirrhosis is regularly among the top 20 disability-adjusted life years and years of life lost in global and regional estimates of causes of chronic liver disease-related issues. South-East Asia took the largest burden of the load [8]. Extrahepatic morbidities are common in people with liver disorders, and they contribute considerably to death and poor living standards. In the United States, individuals with chronic liver disease have a higher joblessness ratio (55% vs. 30%), with their disability-related unemployment rates being higher (30.5% vs. 6.6%), and spend more money on health care ($19,390 vs. $5,567) than those without the condition [12].

Patients with chronic liver illness have a higher utilization for inpatient healthcare growing in the previous twenty years; the chronic liver disease had the greatest inpatient mortality among gastrointestinal-related hospitalizations [13]. These figures are frightening, but they also point to the significant potential to enhance public health, as the majority of liver illnesses are avoidable. The sections that follow break down the worldwide burden by type of liver disease [8].

The final phase of a variety of chronic liver disorders that have slow progressed is cirrhosis. Cirrhosis, on the other hand, maybe prevented since the diseases that most often because it develops slowly and can be prevented and treated [14]. Furthermore, cirrhosis prevention is also HCC prevention this is because most cases of HCC develop in a cirrhotic liver. The possibility of HCC is determined by the preexisting illness: it is low when it is autoimmune hepatitis (2.9% in ten years), however, it is high (19.8% in 13 years) when the viral load of hepatitis B is the underlying illness is more than 107 copies/mL [15, 16].

Apart from chronic viral hepatitis, fatty liver disease caused by other prevalent preexisting illnesses (diabetes, obesity, or alcohol misuse) frequently develops into cirrhosis, necessitating expert medical therapy as well as constant monitoring by the primary care physician [14].

General Liver Cirrhosis Evaluation

Fibrous septa between the portal fields define cirrhosis, which appears in micro-and macronodular forms.

Characteristic findings on clinical examination, laboratory testing, and auxiliary studies are used to diagnose the disease. Cirrhosis is characterized by a hard liver on palpation, cutaneous indications of liver disease, and specific risk factors, such as metabolic syndrome, excessive liquor intake, hepatotoxic chemical introduction, and/or hepatotoxic medicine usage [2, 17].

Irregularity of the hepatic surface, inhomogeneity of the hepatic tissue, or expansion of the caudate lobe is all early indications of cirrhosis on Ultrasonography. Splenomegaly is caused by portal hypertension. Thrombocytopenia is seen in advanced liver disease approaching the stage of cirrhosis, as well as impaired hepatic biosynthesis (as evidenced by low concentrations of albumin and cholinesterase and an increase in the international normalized ratio (INR)). In addition, a handicap of the liver's cleansing purpose is seen too by elevated bilirubin concentration. Transaminase levels are usually within normal limits or very slightly increased. However, there is no well-defined laboratory test threshold value that can be used to decide whether cirrhosis screening should be done.

Upper abdomen ultrasonography and gastroscopy are examples of ancillary investigations. When cirrhosis is first identified or suspected, an esophagogastroduodenoscopy should be conducted to see if there are any esophageal varices and to determine the risk of them bleeding [2, 17].

If cirrhosis diagnosis has been firmly settled through imaging investigations and medical symptoms, a liver biopsy is unnecessary, if not even contraindicated (e.g., an indication of decompensation, with ascites and damaged hepatic biosynthesis). If the cause of liver illness is unknown or the stage of the disease cannot be established from the results of the tests listed above, a liver biopsy is recommended. Transcutaneous liver biopsy is recommended in patients with suspected cirrhosis if the clinical symptoms raise uncertainty about the diagnosis or if the biopsy is likely to reveal information regarding the etiology of cirrhosis that may influence treatment options. It is important to remember that after the liver illness has progressed to the point of cirrhosis, determining the original underlying etiology histologically might be difficult or impossible [18, 19].

For the noninvasive techniques, multiple techniques based on ultrasound and laboratory methods have been newly developed. When the sole issue to be answered is the stage of fibrosis, these noninvasive techniques can frequently eliminate the necessity for a liver biopsy; nevertheless, the information they give must always be weighed against the clinical results [19, 20]. Techniques based on laboratory assessment of the degree of hepatic fibrosis may be split into the ones that rely on specific laboratory values linked to fibrosis such as hyaluronic acid content, or ones relying on standard functionality tests of the liver [21].

The AST-to-platelet ratio index (APRI) is a simple screening measure for advanced fibrosis and cirrhosis that is computed as the quotient of the AST (GOT) and the number of platelets. Ultrasonography is used to diagnose cirrhosis because there is a direct relationship between the level of fibrosis and the degree of liver stiffness assessed by ultrasonography. The acoustic radiation force impulse (ARFI) technique and transient elastography are now well-established techniques for fibrosis staging in a variety of liver disorders. These two procedures can also be combined or done on two separate appointments [19, 20].

Longitudinal monitoring of the development and reversal of fibrosis in individuals with chronic liver disease is possible thanks to techniques for measuring liver stiffness and laboratory markers of hepatic fibrosis. Although in more than 90% of cases ultrasonography rules cirrhosis' presence, it is not 100% accurate because of occasional erroneous readings and false-positive results. Values that do not pass the required limitations for ruling advanced fibrosis or the presence of cirrhosis may be difficult to understand; in such cases, the time-based history of the variable in issue is its medically important aspect. It is worth noting that diagnostic thresholds differ based on the underlying cause of liver illness [14, 22].

Imaging

Ultrasonography, computed tomography (CT), and magnetic resonance imaging (MRI) are not always sensitive enough for cirrhosis identification. Histology is still used to make the definitive diagnosis. However, when an evident source is apparent and an indication is captured that an inhomogeneous hepatic texture or surface, enlarged caudate lobe, rarefied hepatic central vein, splenomegaly, or collateral veins, their specificity is high [23]. Other etiologies must be ruled out, such as portal vein thrombosis, parasite infections, or hematological malignancies, and normal radiographic results do not rule out compensated cirrhosis. The primary function of radiography is to identify and quantify cirrhosis consequences such as ascites, HCC, and hepatic or portal vein thrombosis [2].

Their presence following antecubital injection is inversely proportional to the degree of fibrosis. Ultrasonography is the initial imaging modality used to rule out HCC, however its sensitivity and specificity in detecting HCC are lower than MRI or CT, therefore verification of the nodular lesions ought to be done using helical CT and/or MRI. Even in the absence of ultrasonographic lesions, these more stringent methods are required when there is a heightened degree of doubt, such as in patients with alpha-fetoprotein levels above 200 g/L or during a pretransplant assessment. Identification HCC is done by allowing for sensitive visualization of abnormal vessels through Harmonic imaging, Contrast ultrasonography, and power Doppler, although they are not widely accessible yet [24].

When HCC or vascular lesions are suspected, regular CT and MRI are not effective in determining the severity of cirrhosis, but helical CT and MRI with contrast are the modalities of choice. In a study, MRI outperformed helical CT in detecting tiny HCCs with a diameter of 1-2 cm [25]. In hemochromatosis and liver steatosis, MRI has also been proven to help detect hepatic iron and fat levels [26].

Elasticity measurement (Fibroscan) is a potential technology that uses an intercostally positioned transmitter to detect the velocity of an elastic wave. Pulse ultrasonography detects shear wave velocity, which is related to liver stiffness, or fibrosis. Morbid obesity, ascites, and narrow intercostal gaps restrict the investigation. Histological cirrhosis was distinguished from milder phases of fibrosis in a study of 327 individuals with a receiver operating characteristics curve of 97% with hepatitis C, thought of as an optimal diagnostic method. Elasticity scans with a sampling scale of 1/500 of the liver, are a valuable non-intrusive diagnostic for detecting or ruling out cirrhosis [2, 27].

An important role in the diagnosis and management of chronic liver disorders is played by ultrasound, since it may provide diagnostic and prognostic information as well as detect complications such as HCC and portal hypertension. While conventional ultrasonography is useful for assessing the liver parenchyma and detecting liver abnormalities, a variety of additional Ultrasound methods have been developed to expand its use. The performance capabilities and availability of non-invasive techniques of measurements in chronic liver disease are rapidly evolving [28].

Laboratory testing and imaging examinations are examples of this. Elastography has attracted a lot of attention recently because of its capacity to offer noninvasive data on the liver fibrosis state. The reviews' goal being summarize the present state of Ultrasound methods and to offer some insight into their present and possible later utility, with a particular focus on one of the most widely used techniques, elastography.

Hepatic fibrosis is a complication of chronic liver damage leading to cirrhosis and final phase liver disease. While hepatitis B and C virus infection and alcohol remain the primary causes of liver disease globally, the rising occurrence of metabolic syndrome and obesity has upshot an increase in the occurrence of cirrhosis due to NAFLD [29]. NAFLD prevalence is greater than previously thought [30]. If obesity and diabetes rates continue to grow at their present rates, NAFLD prevalence in the United States is predicted to approach 50% in 2030, reaching epidemic proportions [31].

Non-alcoholic steatohepatitis is a progressive and extreme type of NAFLD that is now recognized as a significant source of cirrhosis. With new therapies for hepatic fibrosis and NAFLD on the horizon, there is a rising need for precise diagnosis, prognosis, and disease nursing. The gold standard

in fibrosis evaluation has always been liver biopsy [32-34]. There are some drawbacks to liver biopsy. It has a complication rate of about 1% as being an invasive test [35].

In individuals with widespread parenchymal liver disorders, liver biopsy has been demonstrated to have a significant risk of sampling mistakes. Although a normal core biopsy specimen comprises just 1/50,000 of the liver's volume, fibrosis is spread unevenly across the organ. Samples collected from the right and left hepatic lobes, for example, varied in histological staging and grading in a group of 124 hepatitis c virus patients. Cirrhosis was underdiagnosed in 14.5% of patients as a result of sampling error.

The texture of the liver parenchyma is a subjective feature with a limited sensitivity for detecting cirrhosis. Routine Ultrasound is not a reliable predictor of early or severe fibrosis in chronic viral hepatitis, according to new retrospective research on the exactness of normal Ultrasound in the phase detection of fibrosis.

However, a study of 103 individuals with chronic liver illness found a statistically significant association between liver parenchymal texture (classified as fine echotexture, slightly coarse, coarse, and very coarse) and the fibrosis degree [36]. When additional characteristics (liver surface nodularity and liver edge) were included, an association to the increase in the degree of fibrosis. When compared to echotexture, liver surface nodularity has a higher sensitivity and specificity for detecting cirrhosis, with a sensitivity and specificity of 88% [37].

Ascites must be present in order to establish a fluid-tissue contact for optimum assessment. Cirrhosis is typically more advanced and less of a diagnostic problem once ascites are evident. When ascites are absent, an alternative strategy is to employ the hepatic vein lumen as an internal fluid-tissue interface. The morphology of the hepatic veins would be changed if internal nodularity in cirrhosis caused architectural deformation. The following characteristics were examined in potential initial research involving 50 patients without liver disease and 38 patients with cirrhosis: straightness of the hepatic vein, uniformity hepatic vein echogenicity uniformity, and a 1-cm segment of hepatic vein visibility.

Using real-time compound imaging (RTCI) with a 5–2 MHz transducer, the straightness of the hepatic vein was divided into three groups (somewhat wavy, straight, and extremely wavy) and respectively produced the greatest precision and specificity of 97% and 91%. The second beneficial characteristic was uniformity of hepatic vein wall echogenicity, which respectively had a precision and specificity of 88% and 86%. similar to previous research on superficial surface nodularity. When all three characteristics were combined, RTCI had a specificity of 98% for cirrhosis, but a sensitivity of 65%. The analysis of the morphology of the hepatic vein was shown to be a good predictor of cirrhosis in this pilot research, with little inter-and intra-observer error. The three hepatic vein morphological features may be easily assessed in clinical practice, however more thorough clinicopathological association and greater patient numbers are necessary for confirmation [38].

It is evident that absolute portal vein diameter varies much too much to be utilized in the diagnosis of portal venous hypertension. Furthermore, we have found that ultrasonography access and adherence to breathing instructions are frequently insufficient to achieve accurate and repeatable measures of portal vein diameter change [28].

Hepatic fibrosis, nodular distortion of hepatic architecture, and perfusion anomalies are all common pathologic characteristics of cirrhosis [39]. Bridging bands or focal confluent fibrosis are fibrotic alterations that show as bridging bands or focal confluent fibrosis. Bridging bands often vary in thickness and, due to delayed contrast enhancement, may resemble a tumor capsule. On unenhanced and venous phase CT, focal confluent fibrosis is characterized as a hypoattenuating region that is wedge-shaped on the periphery. Enhancement of the lesion may occur during the delayed phase [40].

The presence of overlying capsular retraction and loss of volume in regions of localized confluent fibrosis is a key characteristic that distinguishes this entity from malignant diseases [41, 42]. The stage of cirrhosis affects the morphology of the liver. Hepatomegaly affects more than 60% of individuals with early cirrhosis. broadening of the porta hepatis, amplification of the interlobar fissure, and extension of the pericholecystic space are further early observable morphologic alterations of the liver [43].

In severe stages of cirrhosis, the liver shrinks, especially in cases of alcohol-induced cirrhosis. The left lobe's medial segment (IV) decreases, while the lateral segments (II, III) hypertrophy, giving it a "tongue-like" appearance. Cirrhosis is traditionally linked with a nodular contour and heterogeneity of the liver as a result of these alterations [39].

Hepatic steatosis is a nonspecific, reversible hepatocyte reaction to chronic damage that is often found in cirrhosis caused by alcohol. The most frequent pattern is a diffuse homogeneous fatty infiltration encompassing the whole liver. On unenhanced CT, the splenic parenchyma is at least 10 Hounsfield Units (HU) higher than the normal liver attenuation when hepatosteatosis occurs. To distinguish this anomaly from hepatic malignancies, normal course vascular structures in regions of fatty infiltration must be identified [39].

Cirrhosis is also characterized by developing hepatic nodular lesions. Names and definitions have been brought forward by an international working group for nodular lesions in cirrhotic individuals in an attempt to standardize nomenclature.

Regenerative nodules, dysplastic nodules, and HCC are the three types.

As a result of necrosis and altered circulation, a region of the liver parenchyma may become inflamed in a well-defined region to form a regenerative nodule. The nodular regeneration can be categorized as micronodular (less than 3 mm in diameter) or macronodular (greater than 3 mm in diameter) based on gross morphologic characteristics. A non-contrast CT seldom shows a regenerative nodule unless it includes iron. On a non-contrast CT, if there is iron deposition (siderotic nodule), the nodule looks hyperdense in comparison to the surrounding liver. Even though micronodular alterations are present in all cirrhotic livers, micronodular changes are seldom seen on CT. Regenerative nodules are indistinguishable from the hepatic backdrop because they are isodense to the residual parenchyma in the venous phase and inert in the major phase. The accuracy of a non-contrast CT scan in identifying a regenerative nodule is about 25%. The most typical morphologic appearance found in cirrhotic individuals is a mix of micro-and macronodular regeneration [44].

Because of its capacity to consistently display HCC, MRI is commonly employed in the detection of tumor development. Ultrasound, computed tomography, and conventional MRI are currently accessible imaging techniques that are neither sensitive nor specific in detecting early parenchymal alterations [45]. Classic morphological and signal intensity alterations can be used to detect several indications of mild and severe cirrhosis. Fat and iron accumulation, regenerative nodules, necroinflammatory infiltration, fibrosis, varices, perfusion anomalies, and hepatocyte functioning may all be seen using new functional MRI sequences [46, 47]. Imaging modalities should be used to assess chronic hepatitis and cirrhosis at the earliest stages of the illness. Any technique for analysis of chronic hepatitis and cirrhosis must properly detect regeneration, necrosis, iron, swelling, fibrosis, fat, and neoplasia in order to be clinically helpful. The most significant criteria for antiviral therapy indication and follow-up include fibrosis, necroinflammatory activity, fat, and iron deposits. The radiologist's report and measurement of these characteristics are critical in order to influence patient care [45].

Conclusion

Characteristic findings on clinical examination, laboratory testing, and auxiliary studies are used to diagnose the disease. If firmly established through imaging investigations and medical symptoms that cirrhosis has been diagnosed, a liver biopsy is unnecessary, if not even contraindicated. Therefore, non-invasive techniques such as imaging are important to be used properly for the evaluation. For the noninvasive techniques, multiple ultrasound-based and laboratory methods have been newly developed. In the diagnosis and management of chronic liver disorders, ultrasound has a significant position since it may give indicative and analytical information whilst detecting impediments like portal hypertension and HCC.

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