SMGroup

Viral Hepatitis

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Viral hepatitis is a group of systemic infectious diseases caused by various hepatitis viruses, mainly characterized by hepatic inflammation and necrosis. It is transmitted primarily via the fecal-oral route, blood or body fluids. The defined pathogenies so far include 5 hepatitis viruses including hepatitis A, B, C, D and E viruses. The hepatitis A and E viruses are transmitted via the fecal-oral route with seasonal variance, which may develop into acute hepatitis and cause epidemic outbreak. The other hepatitis viruses mainly spread along with blood and their infection may develop into acute hepatitis, chronic hepatitis or asymptomatic infection. And they commonly develop into chronic hepatitis, with sporadic occurrence. Some patients may develop hepatocirrhosis and hepatic cellular carcinoma.

IMAGING DEMONSTRATIONS OF VIRAL HEPATITIS

Acute Viral Hepatitis

Ultrasound

The ultrasound demonstrations include slightly enlarged liver volume, generally increased liver echo, density and roughness, with even distribution Based on the detailed ultrasound demonstrations, viral hepatitis can be divided into 3 types: mild, moderate and severe hepatitis.

Acute viral hepatitis can be accompanied by thickened gallbladder wall that is mostly bilateral, poor filling the gallbladder, shrinkage of the gallbladder as well as abnormal stasis of echo spots in the gallbladder lumen that filled in the gallbladder lumen with consolidation manifestations. The specific demonstrations of the gallbladder can be found in the early stage of viral hepatitis. Along with the improved condition of hepatitis, the gallbladder wall commonly recovers, with decreased thickness of the gallbladder wall, absent lamination, normal volume and absent echo within the bile.

The spleen is subject to slight enlargement, which returns to normal along with the improved conditions. Enlarged lymph nodes may be found in the portal area.

CT scanning

Diffusive hepatomegaly and normal proportion of each hepatic lobes are demonstrated by CT scanning.

Plain CT scanning demonstrates diffusive decrease of liver parenchyma density, which is lower than 42 Hu or the density of spleen, with even or uneven density. In the cases of acute severe hepatitis, the hepatic density is obviously uneven, with multiple flakes of low density shadows with poorly defined boundaries and unfixed location. The shadows and normal hepatic parenchyma intersect to form map liked changes.

CT scanning also demonstrates enlarged and more abdominal lymph nodes, which mainly distribute in the portacaval space, liver hilum and around duodenal ligaments and around the abdominal aorta.

Pleural effusion is also demonstrated by CT scanning, in different quantities and mainly distributes in the perihepatic and perisplenic areas as well as in the omental bursa and bilateral paracolic sulci.

The other CT scanning demonstrations include moderate enlargement of the spleen, slightly dilated intrahepatic bile duct, possible enlargement of the gallbladder with a long diameter of above 5 cm, edema of the gallbladder wall. There are poorly defined boundaries between gallbladder wall and gallbladder fossa with surrounding liver tissue or presence of low density ring.

By contrast CT scanning, no obviously abnormal enhancement of the liver parenchyma can be found in the arterial phase; obvious enhancement of liver periphery in the venous phase; and weak medial enhancement. Otherwise, uneven enhancement with lobar distribution can be found, with slow enhancement, even enhancement in the equilibrium phase, but weak enhancement of the whole liver.

MR imaging

MR imaging demonstrates enlarged liver volume, diffuse slightly long T1 slightly long T2 signals of the liver parenchyma due to inflammatory edema, with poorly defined boundaries and even signals. In the cases of acute severe hepatitis, MR imaging demonstrates obviously uneven signals and multiple scattering patches of long T1 long T2 signal shadows, indicating necrosis of liver parenchyma.

Chronic Viral Hepatitis

Ultrasound

Mild chronic viral hepatitis can be demonstrated with normal size of liver and spleen or slightly enlarged liver, more intrahepatic echoes and clearly defined running course of the hepatic vein.

Moderate chronic viral hepatitis can be demonstrated with slightly enlarged liver and spleen, more intrahepatic echoes that unevenly distribute, clearly defined running course of the hepatic vein and no widened lumen diameter of the hepatic and splenic veins. CDFI demonstrates decreased velocity of portal blood flow.

Severe chronic viral hepatitis can be demonstrated with unsmooth liver surface with dull margins, obviously coarse intrahepatic echoes with uneven distribution, poorly defined running course of the hepatic vein or slight stenosis and twists of the hepatic vein. There are also widened lumen diameter of the portal and splenic veins, with the lumen diameter of the portal vein being above 1.2 cm and the splenic vein being above 0.8 cm. In addition, enlarged spleen with a thickness of above 4.5 cm can be found, with bilateral sign of the gallbladder. CDFI demonstrates decreased velocity of the portal blood flow.

CT scanning

By CT scanning, the liver can be normal or slightly enlarged in size. Along with progress of the conditions, the volume of the right liver lobe can gradually shrink with appropriate proportion of all liver lobes or slightly increased proportion of the left liver lobe. The liver surface is not smooth, with slightly dull liver margin. The liver density unevenly decreases, being close to the spleen, which can be complicated by fatty liver. By contrast CT scanning, there are obviously uneven enhancement of the liver parenchyma and diffuse spots of low density shadows. In the delayed phase, the enhancement and the spots are more obvious. The spleen is moderately enlarged, which is progressive. The portal vein is commonly poorly defined, with rare findings of dilated portal vein and its branches. Contrast CT scanning demonstrates low density shadows around

the portal vein, namely halo sign around intrahepatic vessels. There are commonly enlarged gallbladder, thickened gallbladder wall and cholecystolithiasis. Enlarged and more abdominal and retroperitoneal lymph nodes can also be found. Sometimes, secondary changes occur including pleural effusion, pericardial effusion and pleural thickening. In the advanced stage, liver cirrhosis and portal hypertension occur.

CT perfusion scanning of the liver demonstrates obviously increased blood flow in the hepatic artery along with progress of the conditions, significantly increased average passing time of the blood flow through the liver parenchyma, and decreased hepatic blood volume and hepatic blood flow. These findings may be related to obstructed blood flow in the portal vein due to swollen hepatocytes, compression of hepatic sinusoid and increased interstitial fibers.

MR imaging

Routine MR imaging: The demonstrations include shrinkage of the liver volume, improper proportion of hepatic lobes, irregular liver margins and a small amount of subcapsular effusion. The signal from the liver parenchyma is significantly uneven, especially in delayed phase by contrast imaging, with diffuse spots of low signal shadows. Circular edema in T1WI low signal and T2WI high signal can be found around the portal vein, which is more favorably demonstrated by MRCP. The gallbladder wall is thickened with edema that form bilayer phenomenon. Edema of loose connective tissue in the outer membrane layer can be found, with a high T2WI signal. The thickening of mucosa and muscle layer is not obvious, with shrinkage or disappearance of gallbladder lumen.

Enlarged portal lymph nodes are sometimes the only MRI demonstration in the cases of acute or chronic viral hepatitis. By fat suppression FSE T2WI, the signal intensity of extrahepatic lymph nodes increases along with increased activity of chronic viral hepatitis. The enlarged lymph nodes are commonly distributed along with lymphatic drainage area in the liver and bile duct, namely from the hilum to the first segment level of the duodenum.

Dynamic contrast MR imaging demonstrates intrahepatic patches of enhancement in the early stage, indicating present or recent hepatocytic lesions. The intrahepatic linear enhancement in the late stage indicates hepatic fibrosis. In the delayed phase, the enhancement intensity of the liver parenchyma gradually increases along with the decreased hepatic functions, with delayed enhancement peak of the liver parenchyma. This is possibly related to decreased velocity of the blood flow in the portal vein.

DWI: Studies have demonstrated that ADC value can be applied to assess early hepatic fibrosis and activity of chronic viral hepatitis. The coefficients of the ADC value with the activity of chronic viral hepatitis and the severity of liver fibrosis are 0.470 and 0.659, respectively, which are higher than the results of clinical and laboratory tests. The ADC value can also be applied to assess the grading of chronic viral hepatitis and the therapeutic responses. Along with the increased grading of liver fibrosis, the average ADC value decreases. Different average ADC values can be

demonstrated in different pathological types as well as before and after treatment. The average ADC value is closely related to the grading of fibrosis. The ADC value can be used for the diagnosis of hepatitic cirrhosis and the assessment of the therapeutic efficacy.

MRS: MRS is a noninvasive examination that can accurately demonstrates the biochemical information of living tissues. The roles of MRS in early detection of liver fibrosis and in the staging of chronic viral hepatitis have attracted increasing scholarly attention. Normal and fibrotic livers have different demonstrations by 1H-MRS. The relative average values of glutamate compounds as well as the metabolites and the lipid ratio of phosphomonoesterase complexes in normal livers are 0.14±0.04, 0.03±0.01 and 0.21±0.04, respectively. The most significant change of chronic viral hepatitis demonstrated by MRS is that the lipid peak is obviously lower than the lipid level of normal liver, which is increasingly lower along with progress of chronic viral hepatitis, namely severity of hepatic fibrosis. Apart from stage 0, the average values of different stages of chronic viral hepatitis are obviously different in aspects of glutamate compounds as well as metabolites and the lipid ratio of phosphomonoesterase complexes and glycoconjugate. And such changes are corresponding with histopathological manifestations of chronic hepatitis. Therefore, some scholars proposed that in vivo 1H-MRS replace liver biopsy for the diagnosis and grading of chronic viral hepatitis.

Cholestatic Hepatitis

Cholestatic hepatitis is mainly characterized by different degrees of dilation of intrahepatic bile duct, thickened gallbladder wall and inflammatory change of intima. Due to the susceptibility of secondary gallbladder infection in patients with cholestatic hepatitis, intrahepatic bile duct and gallbladder stones are commonly found. And the pathological changes of the gallbladder are related to the degree of bilirubin deposits.

VIRAL HEPATITIS RELATED COMPLICATIONS

Liver Cirrhosis and Primary Liver Cancer

Ultrasound

The ultrasound demonstrations include shrinkage of the liver volume; obviously unsmooth liver surface in serrated apprearance; rough and increased echo of the liver parenchyma that is nodular and uneven; thinner, twisted and stiff hepatic vein with uneven diameter; splenomegaly; increased lumen diameters of the portal vein and splenic vein; and abdominal effusion. Bilateral sign can be found in the gallbladder. CDFI demonstrates a decreased blood flow velocity in the portal vessels.

CT scanning

Size of the liver: In the early stage of cirrhosis, the liver volume can be normal or slightly increased, with no specific lesions by CT scanning. In the middle and advanced stages of cirrhosis, enlarged and atrophic liver lobes can be found.

Morphology of the liver: Due to nodular regeneration and contraction from fibrosis, the hepatic margin is unsmooth. In some cases, the normal morphology of the liver is absent.

Density of the liver: Plain CT scanning demonstrates alternative high density shadow and low density shadow in the liver parenchyma that diffusively distribute. Fibrosis is demonstrated as regenerative nodules surrounded by mottled, bridge like and reticular low density shadows. Contrast CT scanning demonstrates more serious uneven density of the liver than plain scanning, which may also be demonstrated with a tendency of more even density.

Regenerative nodule: The details are described in the part of MR imaging demonstrations (cirrhotic nodules).

Widened hepatic fissures and enlarged gallbladder fossa

Secondary changes: Secondary changes include portal hypertension, splenomegaly and ascites.

MR imaging

MR imaging has the same demonstrations of liver size, liver morphology, splenomegaly and portal hypertension as CT scanning. In addition, MR imaging can demonstrate the following abnormalities:

Signal abnormalities of the liver parenchyma: Fibrosis can cause diffuse or local signal abnormalities of the liver parenchyma. Diffuse patients have patchy, thin-banded, thick bridge around regenerative nodules and perivascular raglan-sleeve shape of high signals with unclear margins on T2WI. T1WI signal is low and unobvious with slight enhancement on enhanced scan. About 15% patients with liver cirrhosis in late period appear fused fibrosis, wedge shape or strips, usually locating in liver segments IV, V and VIII while some involving the hepatic lobes or liver segments, accompanied with atrophy of the liver lobes or segments and capsular retraction. The features of fused fibrosis are low signal intensity on T1WI and high signal intensity on T2WI. Gd contrast scanning typically demonstrates early low signal; delayed scanning, high signal; with early enhancement. Contrast scanning demonstrates the lesions of SPIO as wedge-shaped high signals and low signals at the residual liver parenchyma.

Liver stiffness is positively correlated with hepatic fibrosis. MRE may aid in the grading of hepatic fibrosis.

Cirrhotic nodules

CT scanning demonstrations of RN: RN occurs in nearly all cirrhotic livers except early stage cirrhotic liver. By plain CT scanning, most RN are in equal density; RN containing iron and/or glycogen may be in slightly high density; with strips or spots of low density shadow of fibrous septum around RN. RN is mainly supplied by the portal vein, with no enhancement in the arterial phase after dynamic contrast and no finding in the venous phage due to the equal density. In the

cases with obvious fibrous septum around RN, RN can be contrasted with slightly low density in the portal vein phase due to the enhancement of the fiber tissue. In some rare cases of RN, the blood supply from the hepatic artery can be compensatorily increased due to decreased blood supply from the portal vein of unknown causes, which can be dynamically demonstrated as high density nodules in the arterial phase as well as high, equal or low density in the portal vein phase. RN is demonstrated by CTHA as low density nodules surrounded by enhanced fibrous septum but by CTAP as slightly high density nodules surrounded by low density fibrous septum in the liver, indicating benign nodules mainly supplied by the portal vein. In some cases, RN is demonstrated as slightly high density by CTHA and slightly low density by CTAP, indicating a mild increase of blood supply from arteries.

MR imaging demonstrations of RN: By T1WI and T2WI, RN is commonly demonstrated as equal signal, with occasional high signal by T1WI and low signal by T2WI, which may be related to hemosiderin deposition or fibrous septum around the nodules. Hemosiderin deposition causes a decrease of T2WI signal. The increased water content in fibrous septum due to inflammatory responses or dilated blood vessels causes the demonstrations of small ring shape or reticular high signal shadow to contrast low signal RN (Figs 14-7-14~16). RN containing iron deposits is demonstrated as low signals by T1WI and T2WI. RN containing copper or fatty deposits are demonstrated as high signal by T1WI and possibly decreased signal in contrary phase. The blood supply of RN is mainly from the portal veins, with a small quantity from the hepatic artery, which cannot be demonstrated by dynamic contrast imaging. However, the thick fibrous septum in the cases of liver cirrhosis is commonly enhanced in contrast portal vein phase, with relatively high signal. Therefore, the not enhanced RN is contrasted as relatively low signal. RN is commonly accompanied by normal functioning of the hepatocytes, which can uptake and secrete contrast agent of the liver and gall. By contrast delayed imaging, the hepatocytes are in equal signal. However, some rare nodules can only uptake but cannot secrete hepatocytic contrast agent, which are demonstrated as high signal by delayed imaging. Most RN can uptake SPIO and is demonstrated in low signals by SPIO contrast T1WI and T2WI.

Radiological demonstrations of DN: By plain CT scanning, DN is in equal density. DN and its peripheral hepatic tissues are demonstrated as equal density in any phase of dynamic contrast CT scanning. Otherwise, they are demonstrated as equal density in the arterial phase but slightly low density in the portal vein phase and/or delayed phase. Sometimes, the fibrous septum around DN is thick, which can be enhanced in the arterial phase to contrast the relatively low density DN. In some enhanced nodules, there are smaller enhanced nodules in the large low density nodules, which is known as nodules in nodules and may be a demonstration of cancer lesions in HGDN. By CTHA, most of DN have the same or decreased blood supply as or than their peripheral hepatic tissues; some rare DN, especially HGDN, has increased arterial blood supply. By CTAP, most DN have the same or decreased blood supply as or than their peripheral hepatic tissues, and sometimes may even have slightly increased blood supply.

MR imaging of DN is based on histological changes, with various demonstrations. The signal intensity and regenerative nodules have an overlap with well-differentiated HCC. Generally, MR imaging demonstrates high T1WI signal and equal or low T2WI signal. T1WI can also demonstrate equal or low signal except for necrosis in the nodules. By T2WI, DN is not demonstrated as high signal, which is believed to be a relative specific demonstration. However, it is sometimes still difficult for its differentiation from HCC nodules. By T2WI, different grade of DN has different signal, with low signal for low grade DN and high signal for high grade DN. Some DN contains lipids, which are in high or equal signal by both T1WI and T2WI. After applying fat suppression sequence, DN in high signal can be demonstrated as equal or low signal; and DN in equal signal can be demonstrated as low signal. Despite of the variance in blood supplies of DN, most DN is supplied by the portal vein and some rare DN, especially HGDN, have more arterial blood supply. Therefore, dynamic contrast T1WI generally fails to demonstrate enhancement of DN in the arterial phase. However, the thick and obviously enhanced fibrous septum is demonstrated as high signal, while DN is demonstrated as relatively low signal. In the portal vein phase, the enhanced DN is demonstrated as high signal and some as equal signal. In the equilibrium or delayed phase, some DN are demonstrated as equal signal and some as low signal. By contrast SPIO, LGDN is demonstrated like enhanced RN, therefore, presenting difficulties for their differentiation; HGDN is demonstrated like well differentiated HCC, therefore, presenting difficulties for their differentiation.

CT scanning demonstrations of small hepatocellular carcinoma (SHCC): Plain CT scanning demonstrates lesions in equal, low or high density. Non-dynamic contrast CT scanning fails to distinguish the lesions.

MR imaging demonstrations of SHCC: MR imaging demonstrates SHCC as slightly low, equal or slightly high signal and T2WI demonstrates high signal. HCDN and SHCC sometimes can be demonstrated as nodules in nodules, namely singular or multiple high signal lesions in the low signal nodules by T2WI. By T1WI, the large nodules are in low signal and the internal nodules are in equal signal.

During the development of the lesions from RN, DN to SHCC, MR signal of the nodules develops from high to low by T1WI and from low to high by T2WI. Despite partial overlapping, the demonstration of high signal in the lesions by T2WI should be highly suspected as HCC.

Fatty Liver after Hepatitis

Ultrasound

The demonstrations include enlarged liver volume, bright liver sign of the liver parenchyma, unclearly defined and blunt round liver contour, almost the same echo levels from the intrahepatic blood vessels and the liver parenchyma with absent discrepancy of the echoes. Therefore, the intrahepatic structures are poorly defined.

CT scanning

CT scanning has a high sensitivity to density discrepancies and thus has been a valuable examination for the diagnosis of fatty liver. Plain scanning demonstrates decreased liver density. Diffuse fatty infiltration is demonstrated as decreased density of the whole liver. And focal fatty infiltration is demonstrated as decreased density of local liver lobe or segment. By plain CT scanning, the normal Generally, the diagnostic criteria for fat deposition in the liver by CT scanning include a CT value of the liver being lower than 40 Hu, or the CT value discrepancy between the liver and the spleen being less than -10 Hu, or CT values ratio of the liver to the spleen being lower than 0.85. That is to say, by CT scanning the liver density is lower than spleen density.

By contrast CT scanning, the enhancement of fatty liver is consistent with that of the normal liver parenchyma, but with a low density that is still lower than the spleen density. Intrahepatic vascular shadows are well defined. Sometimes, the blood vessels become thinner due to compression, but the blood vessels are not invaded or surrounded.

MR imaging

Most of the cases are demonstrated normal by MR imaging. In rare cases, there are demonstrations of slightly high signal by T1WI and T2WI and absent slightly high signal by STIR sequence. At the contrary phase of the chemical shift, the signal intensity in the area of fatty degeneration obviously decreases.

Hepatic Encephalopathy

CT scanning

CT scanning demonstrates cerebral cortex atrophy and brain edema. SPECT demonstrates regional cerebral blood flow abnormalities, namely, decreased regional blood flow volume in the frontal lobe, temporal lobe and basal ganglia region. The degree of the decrease in regional blood flow volume is related to the severity of cognitive impairment.

MR imaging

MRI T1WI demonstrates abnormal high signal in the bilateral globus pallidus, the periphery of the red nucleus in the midbrain and the anterior lobe of pituitary gland, whose pathological basis is related to manganese deposition in the basal ganglia. By T2WI FLAIR sequence, there are bilaterally symmetric high signals in the white matter of the cerebral hemisphere. The lesions are symmetrically distributed.