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Review article

'Nonportal' splanchnic venous supply to the liver: abnormal findings on CT, US and MRI

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Introduction

With the advent of imaging modalities such as ultrasound (US), computed tomography (CT) and magnetic resonance imaging (MRI), unknown diseases and scarcely recognized states have been detected and clarified in the liver: focal fat deposits and focal sparing of fatty liver are typical examples. Moreover, the introduction and frequent application of dynamic CT/MRI as well as angiography-assisted CT have also revealed not only tiny lesions but also pseudolesions such as nontumorous focal enhanced areas and nonpathological perfusion defects [1–7].

The liver is an unusual organ from the point of view of its blood supply: (1) it has a dual blood supply from the portal vein and the hepatic artery, and (2) many vessels supply the liver from other than the portal trunk and the proper hepatic artery [1]. As normal or variant, there are two kinds of veins that supply venous blood to the liver: veins originating from a digestive organ, such as the cystic vein and parabiliary venous system [3, 6, 8], and systemic veins, such as the inferior and superior vein of Sappey [9, 10].

The portal venous blood is vital to the maintenance of liver function, and one of the most important roles of the portal venous system is transportation of dietary elements absorbed in the small intestine ('portal' venous blood) to the liver. However, parts of the liver parenchyma receive venous flow instead of the 'portal' venous flow, especially around the gallbladder and the hepatic hilum [11–13], as well as in some areas along the outer hepatic surface such as the anteromedial portion of S_4 [14–16].

The presence of a venous blood supply other than the 'portal' blood is interesting not only from the physiological point of view but especially since it causes problems in liver imaging; these areas are depicted as a perfusion defect on CT during arterial portography (CTAP) obtained by injection of the superior mesenteric artery beyond the inferior pancreatic arcade. They also appear as focal sparing in the case of (nutritional) fatty liver [12], and sometimes appear as a focal fat deposit [17] or a hyperplastic area [13] on unenhanced CT/MRI and US, or as focal highly enhanced area on intravenous dynamic CT/MRI and CT hepatic arteriography (CTHA) [13, 18].

To correctly recognize such imaging findings we have recently suggested the term 'nonportal' (gastrointestinal) splanchnic veins for veins draining from digestive organs and flowing into the liver without passing the portal trunk [19]. 'Nonportal' splanchnic veins whose blood lacks any dietary elements absorbed in the jejunum may directly perfuse the hepatic parenchyma instead of the 'portal' vein. Thus specific parts of the liver may be perfused with 'portal' blood, 'nonportal' splanchnic blood and/or blood from systemic veins, as well as with blood from the hepatic and/or extrahepatic artery [1, 20].

In this pictorial essay we will illustrate the spectrum and discuss the etiologies of abnormal imaging findings due to 'nonportal' splanchnic venous supply to the liver and how these can be distinguished from true mass lesions.

'Nonportal' splanchnic vein

Most draining veins from digestive organs finally flow into the portal trunk via either the superior mesenteric vein or the splenic vein. However, some veins flow directly into the liver parenchyma and supply venous blood therein instead of 'portal' venous blood via the portal trunk (Fig. 1). The cystic vein from the gallbladder [3], the parabiliary venous system from the pancreatic head, duodenum and distal stomach [8] and the aberrant gastric venous drainage from the gastric antrum and pancreatic head [6] are important constituents of this entity (Fig. 2).

These veins may flow directly into the liver parenchyma, but often they are also part of a network that

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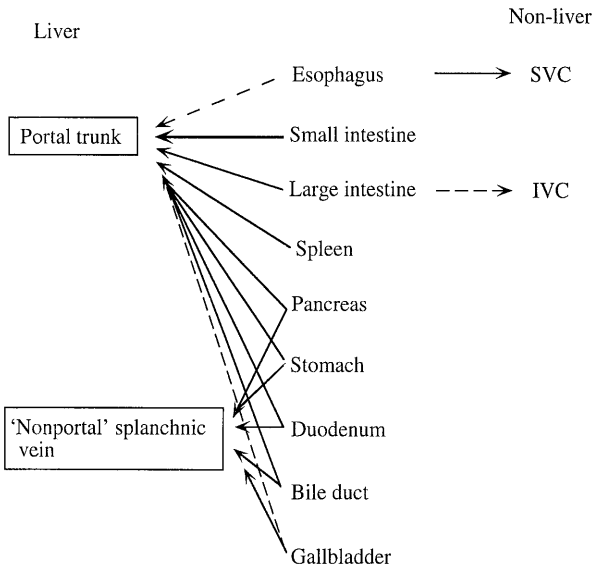


Fig. 1. Gastrointestinal splanchnic veins. *IVC,* inferior vena cava; *SVC,* superior vena cava

communicates with (and actually flows into) branches of the portal trunk outside the liver without perfusing the liver parenchyma independently [8, 21].

More recently, using color/power Doppler US [22, 23] and reformatted enhanced CT [24], it has become possible to depict such veins (Fig. 3). When veins flow into the liver directly, they perfuse the parenchyma irrespective of the presence or absence of communications with the intrahepatic portal vein. When there are communications of the 'nonportal' splanchnic vein (NPSV) with the intrahepatic portal vein, blood flow may reverse (i.e., from the periphery to the center) in that specific intrahepatic portal vein [19].

The area of the liver perfused by these veins can be evaluated on enhanced CT/MRI: (1) CTAP [1], (2) CTHA or intravenous dynamic CT/MRI [5, 18] and (3) CT during injection of the individual artery such as the cystic artery and the right gastric artery (Fig. 4) [21, 22, 25]. According to the imaging modality used the areas depicted may differ somewhat.

Focal sparing of fatty liver

In diffuse fatty liver some areas frequently show a relatively high attenuation almost like that of the spleen [11]. These areas are usually limited to specific sites: around the gallbladder (Fig. 5) [26, 27] and the hepatic hilum (Fig. 6) [12, 22]. Their shapes are roundish to semicircular or zonal in contact with the hepatic capsule over a long distance. Their contour is generally rather irregular, especially at the opposite side of the hepatic capsule.

The close relationship of focal sparing with the NPSV supply to the liver has frequently been demonstrated: (1) both have a similar location (Fig. 7); (2) at CTAP perfusion defects are generally associated with focal sparing in diffuse fatty liver [12]; (3) an area of focal sparing often has a normal-appearing vessel widening toward the hepatic capsule (Fig. 8) as is the case in a NPSV-supplying hepatic area [28]; (4) color/power Doppler US often demonstrates a hepatopetal-flowing vein entering the focal sparing area from the hepatic hilum or the gallbladder wall (Fig. 3) [22, 23]; and (5) patients after cholecystectomy never show focal sparing around the gallbladder [27] whereas patients with distal gastrectomy can gave focal sparing around the hepatic hilum.

Theoretically, focal sparing in nutritional fatty liver occurs in the hepatic parenchyma lacking supply of 'portal' blood flow rich in dietary elements absorbed in the small intestine. Additional evidence will be obtained by comparing the incidence and site of focal sparing in nutritional fatty liver with those in fatty liver induced by hyperalimentation or drug adverse effect.

Focal enhanced area

In intravenous dynamic CT/MRI as well as CTHA there may be many highly enhanced areas that do not correspond to true mass lesions [3, 5, 7, 13, 18]. These areas frequently correspond to the typical sites of NPSV supply to the liver (Figs. 7–9) [13]. The enhancement in NPSV-supplying areas can be explained by the earlier venous return of contrast agent through the NPSV com-

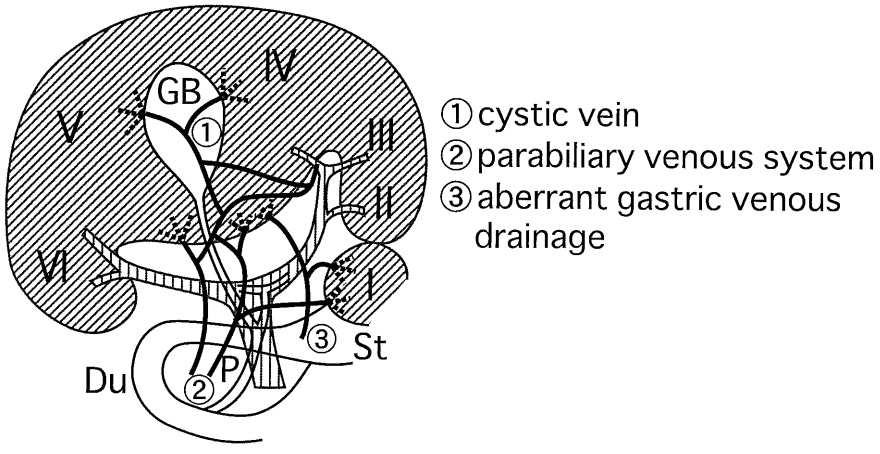
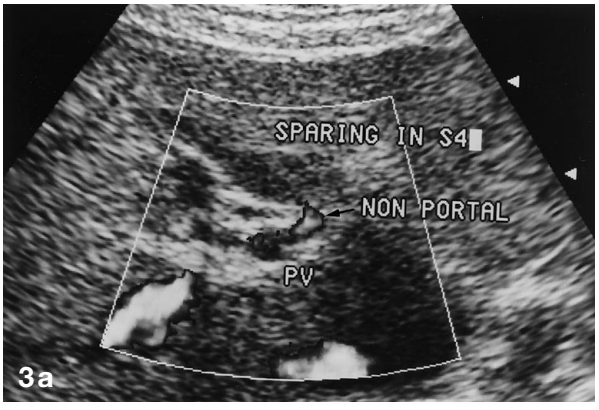


Fig. 2. 'Nonportal' splanchnic venous supply to the liver. *I–VI,* segments of the liver; *GB,* gallbladder; *Du,* duodenum; *P,* pancreas; *St,* stomach



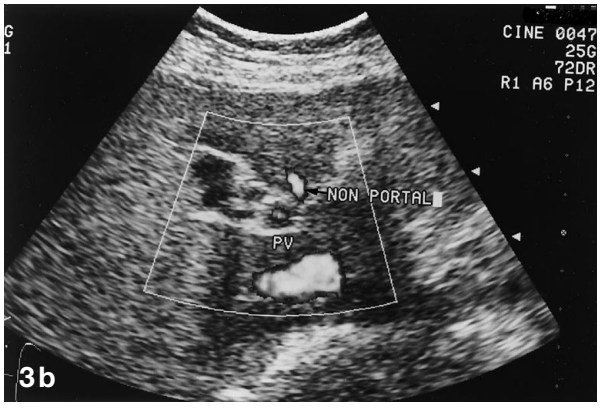


Fig. 3 a, b. 'Nonportal' splanchnic vein depicted by power Doppler ultrasound. **a** A patient with fatty liver has focal sparing in the posteromedial part of S_4. **b** Power Doppler ultrasound depicts a vein (nonportal: *arrow*) entering the focal spared area. *PV,* portal trunk

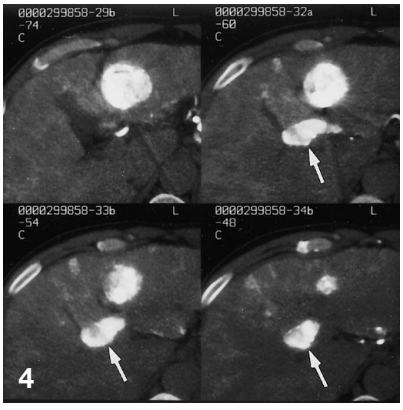
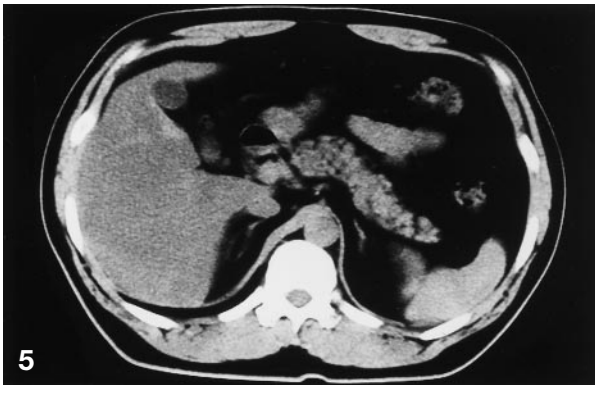
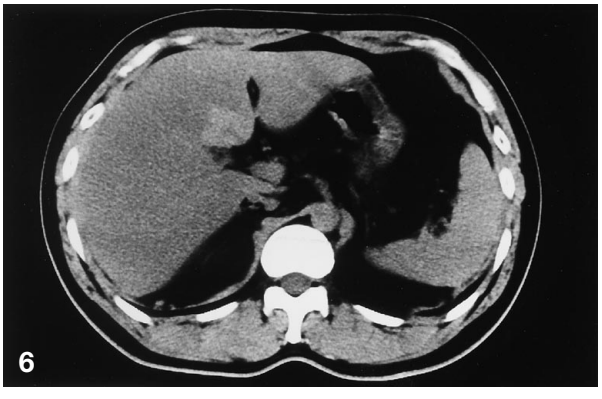
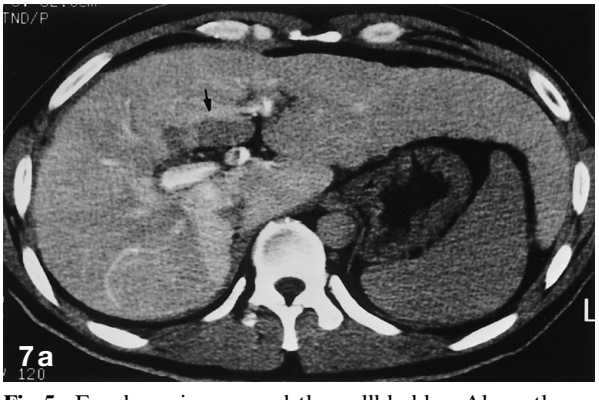


Fig. 4. CT arteriography through the right gastric artery. Posteromedial part of S_4 shows strong enhancement *(arrows).* A round hyperattenuated area in S_3 is lipiodol deposit in a hepatocellular carcinoma previously treated with chemoembolization. (Courtesy of K. Matsueda, Aichi Cancer Center Hospital)







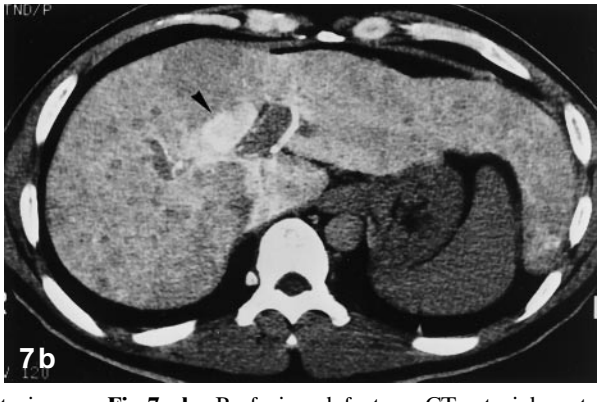


Fig. 5. Focal sparing around the gallbladder. Along the anterior surface of S_5 focal sparing is noted

Fig. 6. Focal sparing around the hepatic hilum. There is a rectangular-shaped hyperattenuated area in a patient with fatty liver. Note that the hyperattenuated area is in contact with the hepatic hilum over a long distance

Fig. 7 a, b. Perfusion defect on CT arterial portography (CTAP) and a highly enhanced area on CT hepatic arteriography (CTHA). In the posteromedial part of S_4 a perfusion defect *(arrow)* on CTAP (**a**) and highly enhanced area *(arrowhead)* on CTHA (**b**) are noted. Because of their location and relationship with the hepatic capsule these areas are believed to be induced by NPSV supply to the liver

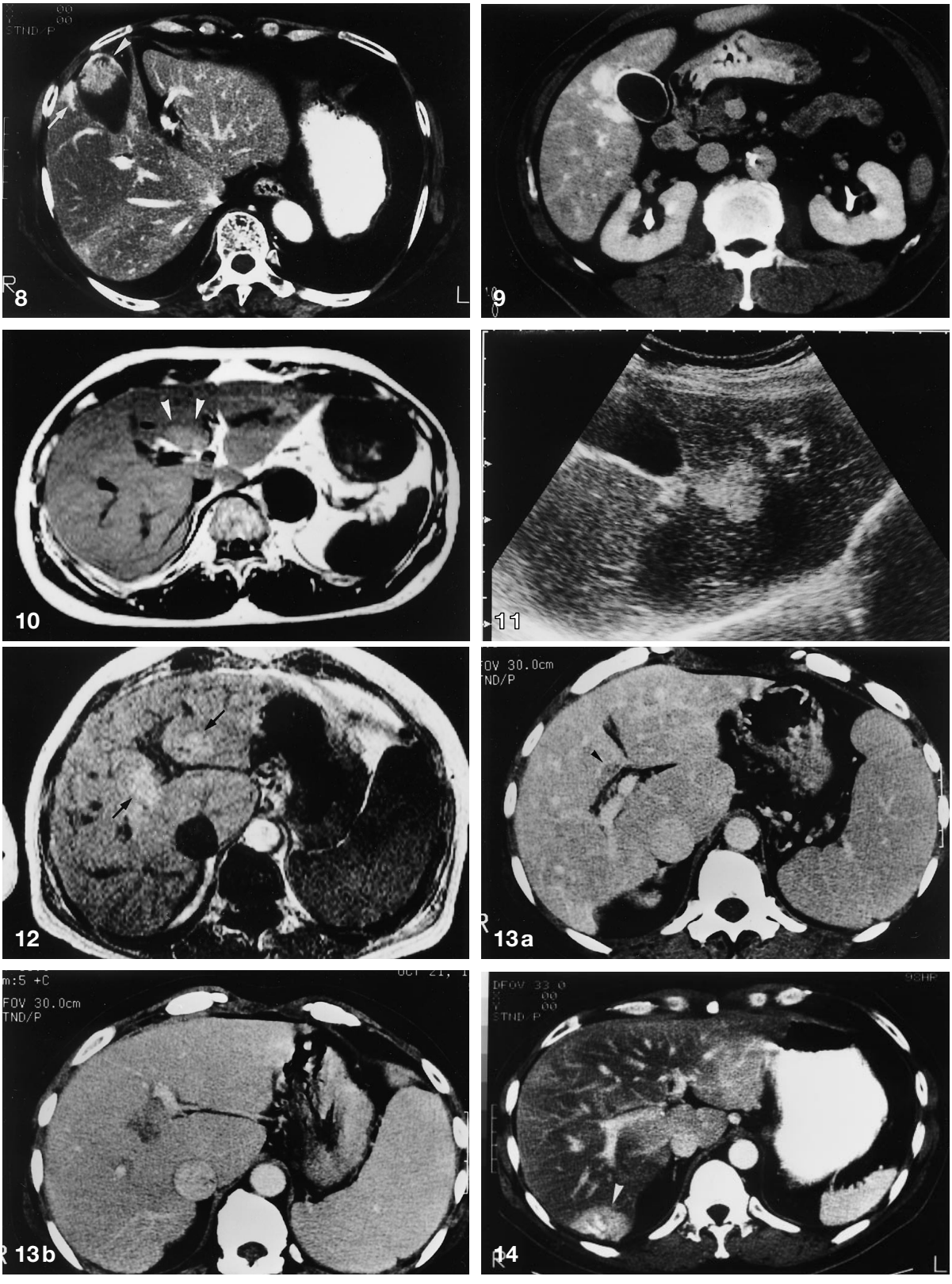


Fig. 8. Highly enhanced area of the anterior part of S_5 containing normal-appearing vessel. In a patient with gallbladder cancer *(arrowhead),* the anterior part of S_5 shows marked enhancement that contains a normal-appearing vein *(arrow)*

Fig. 9. Focal enhanced areas on CTHA. There are two areas showing marked enhancement just adjacent to the gallbladder on CTHA. One is irregular in shape but the other is almost round. Other imaging detected no mass lesion in the liver

Fig. 10. Hyperplastic change in NPSV-supplying area. There is a hyperintense area *(arrowheads)* on the T1-weighted MR image, which appears as a subtle hypointensity on the T2-weighted image (not shown). (Reproduced with permission from [13])

pared with the venous return from the intestine and spleen. Thus areas receiving NPSV flow are additionally enhanced at a relatively high concentration with contrast agent when the agent is coming through the hepatic artery [18]. The effect is most obvious on CTHA since in this case the portal blood does not contain any contrast agent.

The focal enhanced areas show a similar location and shape of corresponding perfusion defect at CTAP (usually appearing a little smaller than the enhanced area on CTHA) as well as visible normal vessels therein (Fig. 8).

Hyperplastic change

Matsui et al. [13] reported that NPSV-supplying areas in cirrhotic liver sometimes appear as hypoechoic on US, hypoattenuated on enhanced CT, and hyperintense on T1-weighted or hypointense on T2-weighted MR images (Fig. 10). In these cases histological investigation revealed hyperplastic changes of the liver [13].

Smaller areas are often regarded as abnormal; however, in a number of cases the smaller areas are actually normal as is the case with focal sparing in diffuse fatty liver. Hyperplastic change is an example of such a case when the rest of the parenchyma is cirrhotic. One should be aware that interpretation of biopsies can be difficult and biopsies from both apparently diseased and normal-looking areas can be useful or even necessary in these cases.

Focal fat deposits

The location of focal fat deposits can vary widely and they can be solitary or numerous [14, 29]. However, the two most common sites for solitary deposits are the anteromedial part of the medial segment of the left lobe (S_4) and the hilar side of S_4 (Figs. 11, 12) [14]. Other less common sites include areas around the hepatic hilum and the gallbladder neck [30]. A small number of multiple lesions are occasionally seen around the hepatic hilum and around the ligamentum falciformis (Fig. 13). In some cases of focal fat deposits the systemic venous supply and the NPSV supply can clearly be demonstrated [14, 30].

The reason why focal fat deposits are induced in specific areas is not clear. However, there seem to be two different mechanisms in the anteromedial part of S_4 and the areas around the hepatic hilum. In the latter case, venous blood from the pancreas seems to be the cause of focal fat deposits since it is seldom combined with focal fat deposits in S_4 and S_5 around the gallbladder fossa. Therefore an increased hormone level in blood from the pancreas or some dietary elements together with hormone may play an important role [30].

On the other hand focal fat deposits in the anteromedial part of S_4 just adjacent to the falciform ligament have a close relationship with venous blood supply from the systemic vein: (1) in the case of focal fat deposits there is also a perfusion defect at CTAP [14, 15]; (2) focal enhancement is not noted at CTHA or intravenous dynamic study [15]; (3) focal fat deposits are never found in cases of portal hypertension where a hepatofugal portosystemic shunt is noted [31], i.e., this area receives portal venous flow and no systemic venous flow in patients with portal hypertension [32]; and (4) thus focal fat deposits in this area may be induced by hepatic injury due to a deficit of 'portal' venous blood.

The reasons why the cystic vein-supplying area does not suffer from focal fat deposits despite a lack of 'portal' venous flow, and why the anteromedial portion of S_4 receiving the systemic vein supply does not show focal sparing in the case of fatty liver, remain to be explained.

Differential diagnosis

True tumor versus focal sparing

Any tumor or mass lesion in fatty liver may appear as relatively hyperattenuated and mimic focal sparing [33]. Differential points include the location (Fig. 14) and shape (Fig. 15) of the abnormal area: a location outside an NPSV-supplying area and quite a round shape almost deny the possibility of focal sparing.

In difficult cases located at the favorite sites of focal sparing and with similarly irregular shape, color/power Doppler may make a definite diagnosis if the hepatopetal-flowing vein coming into the abnormal area is demonstrated [22, 23]. Biopsy may be necessary for the definite differentiation of atypical focal sparing from a true tumor (Fig. 16).

NPSV-supplying area versus other perfusion defects at CTAP

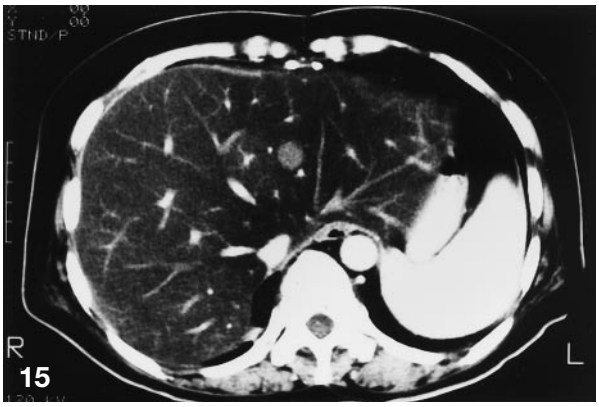
There are several types of perfusion defects on CTAP that can be due to pathological conditions as well as normal variants of hepatic vessel other than NPSV (Fig. 7).

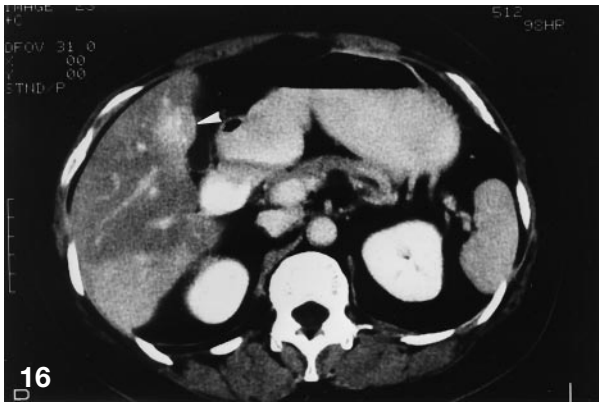
◀ **Fig. 11.** Focal fat deposit. In the posteromedial part of S_4 a multilobulated echogenic area is noted on the right subcostal ultrasound scan

Fig. 12. Multiple focal fat deposits. On the T1-weighted spin echo MR image there are two hyperintense areas *(arrows)* in S_4 and S_2 along the fissure of the ligamentum teres, which also appear as mildly hypointense on the T2-weighted image (not shown)

Fig. 13 a, b. Focal fat deposit in S_4. **a** Irregular-shaped hypoattenuated area *(arrowhead)* is noted in the posterior part of S_4. In a caudad section the hypoattenuated area is in contact with the hepatic capsule of the hilar side and is small in size. **b** In a cephalad section the hypoattenuated area is larger but is still in contact with the hepatic capsule along the fissure of the ligamentum teres

Fig. 14. Metastatic tumor mimicking focal sparing of fatty liver. The patient, who had a history of resection of colonic cancer, was suspected to have irregular fatty liver. The posterior part of S_7 *(arrowhead)* is an unusual site for focal sparing of fatty liver and turns out to be a metastatic focus confirmed by resection. (Reproduced with permission from [33])





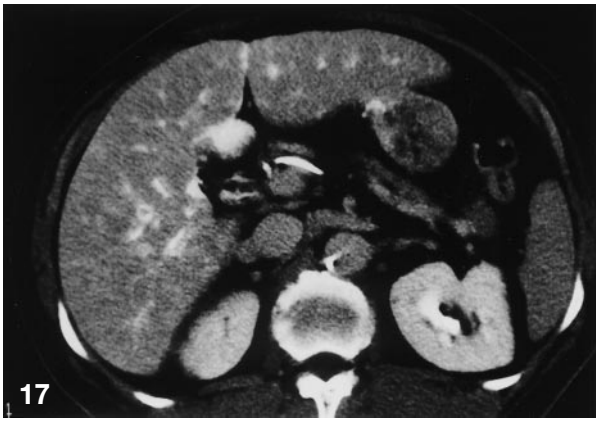


Fig. 15. Metastatic lesion in fatty liver. A round hyperattenuated mass is noted in fatty liver. The patient died of metastatic liver tumors from pancreatic cancer 6 months later

Fig. 16. Metastatic lesion mimicking focal sparing of a fatty liver. There are two foci showing relative hyperattenuation in a fatty liver. One hyperattenuated area in S_5 *(arrowhead)* is protruding medially and compresses the gallbladder. This areas is actually a metastatic focus from breast cancer, confirmed at surgery. (Reproduced with permission from [33])

Fig. 17. Highly enhanced area on CT angiography. A protruding mass-like region shows marked enhancement on CTHA in a patient with liver cirrhosis. On follow-up study this area did not show any change in size and was regarded as enhancement due to cystic venous supply. (Courtesy of M. Satake, National Cancer Center Hospital East)

Any portal venous flow depletion such as obstruction and compression of the portal/hepatic vein, arterioportal shunts related or unrelated to liver tumor as well as systemic venous supply to the liver cause perfusion defects at CTAP [1, 33]. When such a lesion or normal variant occurs on the favored site of a NPSV-supplying area, short time-interval dynamic CT/MRI will be most useful [34]: any portal venous flow depletion induces arterioportal shunting through the peribiliary plexus and shows enhancement of the involved area at the early arterial phase [1].

True hypervascular mass versus focal enhanced area due to NPSV

As mentioned above, NPSV-supplying areas show focal enhancement mainly due to early venous return, while true hypervascular masses such as hypervascular tumors and inflammation show enhancement mostly due to increased arterial flow (Fig. 17). Hence differentiation of a focal enhancing area related to NPSV from a true hypervascular mass is obtained with short time-interval dynamic CT/MRI [35] and/or the very early phase of a dynamic study as well as by the general characteristics of a NPSV-supplying area. Tumor vessels, if demonstrated, are pathognomonic for a hypervascular mass, whereas the presence of normal-appearing veins make a diagnosis of nontumor definite.

Liver-specific MRI contrast agent will be of great value in the differentiation of abnormal findings due to NPSV, since these tumor-mimicking areas have normal or almost normal liver function.

In conclusion, the advent of imaging has revealed many nontumorous abnormal imaging findings. Thorough knowledge of NPSV is very important and useful in judging whether an abnormal imaging finding is induced by a true lesion. If additional effective imaging examinations such as liver-specific contrast-enhanced MRI fail to provide a definite conclusion, biopsy should be performed without delay in order to rule out malignancy.

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