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Hepatic Perfusion Disorders: A Pictorial Review of CT and MR Imaging[1]

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The liver has a unique dual blood supply through the portal vein and the hepatic artery. There are several communications between these two vessels under various conditions such as in hepatic tumors, trauma and liver cirrhosis, vascular compromise, among others. When vascular compromise occurs, this dual blood supply system can cause changes in the volume of blood flow in individual vessels or even in the direction of blood flow. With rapid image acquisition and increased resolution available in multislice CT and MR imaging, hepatic perfusion disorders are now more frequently encountered than in the past. Familiarity with imaging findings of these perfusion disorders will be helpful in characterizing focal hepatic lesions and will also help to avoid false positive diagnoses.

Index words : Liver, blood supply
Liver, CT
Liver, MR

The liver has a unique dual blood supply through the hepatic artery and the portal vein, which comprise 25% and 75% respectively of its vascularization. When vascular compromise occurs, this dual blood supply system can cause changes in the volume of blood flow in individual vessels or even in the direction of blood flow (1 - 3).

Perfusion disorders are defined as any disturbance of homogeneous enhancement of the liver during either the hepatic arterial phase (HAP) or the portal venous phase image at dynamic contrast enhanced CT or MR image (2, 3) (Fig. 1). Perfusion disorders mimic hypervascular tumors and can cause the size and extent of a tumor to be overestimated. Knowledge about perfusion disorder patterns associated with malignant tumors can influence the recommended treatment plan. Therefore, familiarity with dynamic contrast enhanced CT and MR findings of these perfusion disorders will be helpful in characterizing focal hepatic lesions and will also help to avoid false positive diagnoses (4).

The purpose of this pictorial review is to understand the underlying mechanisms associated with hepatic perfusion disorders, to identify a variety of hepatic lesions causing hepatic perfusion disorder, and to review the imaging findings in hepatic perfusion disorders.

Pathophysiology of perfusion disorder

In general, the liver mainly takes blood from the portal vein. Hepatic parenchymal enhancement is minimal because the portal vein delivers approximately 75% at the beginning of the HAP image. Maximal enhancement is seen in the portal venous phase on normal hepatic

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parenchyma.

Functional (Fig. 2) or pathologic arterioportal (AP) shunts (Fig. 3) develop as a result of various causes (Table 1). Among them, the most prominent route is the transplexal via the peribiliary plexus (3, 5). When blood flow through the portal vein is diminished or absent, the hepatic artery takes over perfusion of the liver through the AP shunt (Fig. 4). Contrast material from high-pressure arterial blood passes into a low-pressure portal venous branch. HAP images show segmental or wedge-shaped enhancement of hepatic parenchyma.

CT and MR imaging findings of perfusion disorders

Regardless of the AP shunt mechanism, its dynamic CT findings are as follows; (a) early enhancement of the peripheral portal venous branches in the HAP before the main portal vein is opacified. (b) wedge-shaped hepatic parenchymal enhancement with straight margin on HAP image (Figs. 1, 5), defined as transient hepatic attenuation difference (THAD) (6).

MR imaging findings of AP shunts are essentially the same as those of CT findings. Unenhanced T1- and T2-weighted MR images reveal no abnormal signal intensity. In the case of an intrahepatic portal vein obstruction followed by a parenchymal injury due to hepatocellular atrophy, due to insufficient compensation from arterial flow, or tissue edema caused by extravasated plasma, it is possible that high signal intensity on T2-weighted image could be seen (6, 7).

Etiology of perfusion disorders

Hepatic perfusion disorders are caused by various conditions (Table 2).

Vascular obstruction

A compromise of any individual hepatic vessel immediately induces various changes in the blood flow surrounding vessels due to the close relationship between several of these vessels (3).

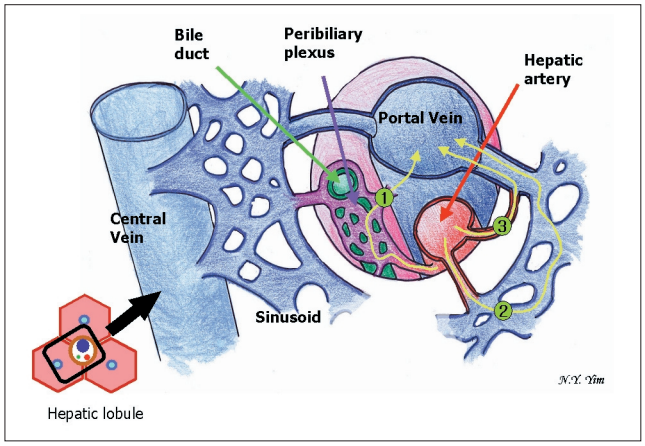
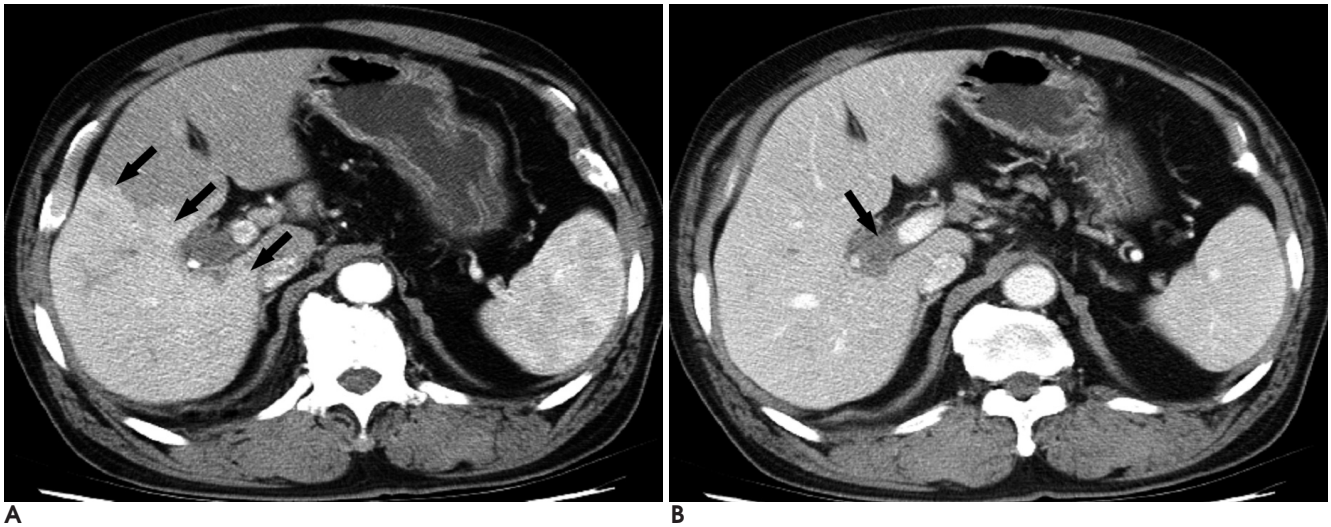


Fig. 2. Schema of a functional shunt of the liver and micorvasculature. Transplexal shunt (hepatic artery → peribiliary plexus → portal vein) Transsinuoidal shunt (hepatic artery → hepatic sinusoid → portal vein) Transvasal shunt (hepatic artery → vasa vasorum → portal vein).



A B

Fig. 1. Transient hepatic attenuation difference secondary to portal vein thrombosis.
A. Hepatic arterial phase CT scan shows area of high attenuation in the right hepatic lobe caused by a compensatory increase in hepatic arterial blood flow. The straight border sign (arrows) between the two lobes is seen.
B. On portal venous phase image, there are no attenuation differences in the entire liver and thrombosis in the right portal vein (arrow).

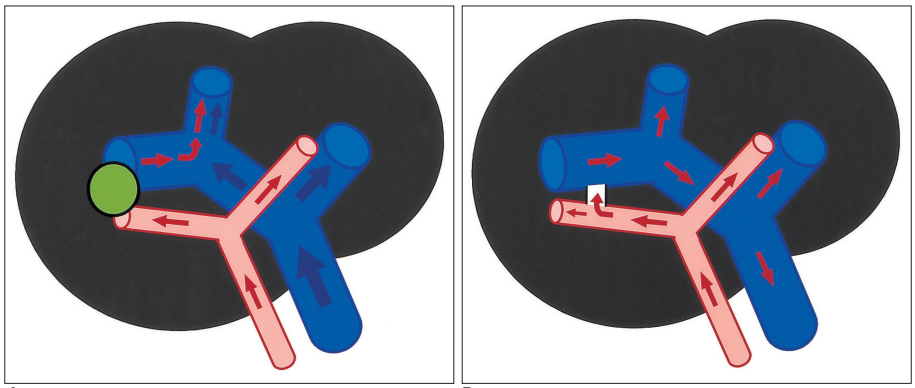


Fig. 3. Pathologic shunt.
A. Schema of a transtumoral shunt. Shunted hepatic arterial flow (red arrow) enters the portal vein branch. The shunted blood then joins normal hepatopetal blood flow in an adjacent portal vein branch (blue arrow).
B. Schema of a macroscopic shunt. Hepatic arterial flow provides the entire hepatic blood (red arrow) supply because portal venous flow does not enter the liver. This schema represents hepatopetal flow of the shunted arterial blood.

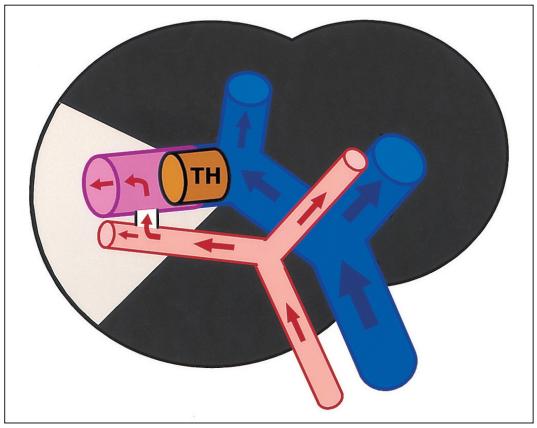


Fig. 4. Schema of a right portal vein thrombosis (TH) with change of blood flow direction. Hepatic artery blood (red arrow) enters into the regional portal vein. White-colored hepatic parenchyma represents transient high attenuation on hepatic arterial phase image.

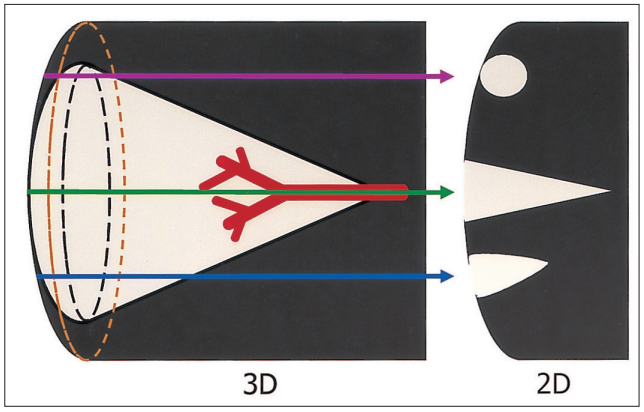


Fig. 5. Schema of pseudolesion. The three dimensional shape of an entire lesion has a different shape on two dimensional spiral CT or MR imaging.

Table 1. Route of AP Shunts

Functional shunt
Transplexal via peribiliary plexus: most prominent
Transsinusoidal
Transvasal
Pathologic shunt
Transtumoral
Macroscopical

Table 2. Etiology of Perfusion Disorders

Vascular obstruction
Portal vein, Hepatc artery, Hepatic vein obstruction
Tumor
HCC*, Hemangioma, Metastasis, Cholangiocarcinoma
Inflammatory disease
Liver abscess, Acute cholecystitis, Acute cholangitis
Iatrogenic and Trauma
Radiofrequency ablation
Percutaneous ethanol injection therapy
Transarterial chemoembolization
Liver biopsy
Trauma
Aberrant venous drainage
Miscellaneous
Liver cirrhosis, Compression of hepatic surface, Confluent fibrosis

HCC* : hepatocellular carcinoma

Portal vein obstruction

A decrease or stoppage of portal flow may occur under various conditions such as, thrombosis, increased parenchymal pressure (e.g. subcapsular hematoma), or surgical ligation of the portal vein. Portal vein thrombosis can occur under various conditions, including tumor invasion, infection (e.g. sepsis), hypercoagulative state, myeloproliferative disorder and noninfectious inflammatory process (e.g. pancreatitis) (1, 3, 5).

When blood flow via the portal vein is diminished or absent, the hepatic artery takes over the perfusion of liver. This occurs mainly through the peribiliary plexus (3, 5) (Fig. 6).

On HAP image, THAD is seen in the involved area as a result of increased arterial flow (Fig. 1). Thrombosis

within the portal vein may also be seen (Fig. 7). In the case of portal vein thrombosis, thread and streak signs along the portal vein may be seen (Fig. 8). Cavernous transformations may develop when the main portal vein is obstructed. When this occurs, collateral venous vessels continue to supply the central part of the liver (caudate lobe and the lateral segment of the left hepatic lobe), whereas the peripheral zone (right hepatic lobe and the medial segment of the left hepatic lobe) receives less portal venous flow (1). To compensate for this, hepatic arterial flow is increased in the peripheral portion of the liver. Consequently, hyperattenuation of the peripheral zone is noted (Fig. 9).

Hepatic artery obstruction

A decrease or stoppage of hepatic arterial flow may occur under various conditions such as, surgical ligation, interventional occlusion (e.g. transarterial embolization), or intimal dissection during embolization (5).

Hepatic arteries communicate with each other in the central portion of the liver, and as a result, a blockage of one of the main arteries causes a change in blood flow (3). In addition, the liver has various other potential arteries that may contribute other than the hepatic artery. The list of these potential arteries includes the inferior phrenic arteries, intercostal arteries, adrenal arteries, left gastric artery and periportal collaterals. Occlusion of the hepatic artery may exaggerate the degree of collateral circulation through these other arteries (5). As a result of this characteristic of the hepatic artery, hepatic arterial occlusion alone does not induce portal venous flow change (8) (Fig. 6).

However, hypervascular tumors (e.g. large hepatocellular carcinoma [HCC] or hypervascular metastases) may induce "hypertrophy" of hepatic arterial blood supply to a lobe or segment of the liver containing the mass. Consequently, this lesion can "steal" arterial flow from the surrounding parenchyma. As a result, the hypoattenuating area may be seen around hypervascular lesion on HAP images. This phenomenon is called "steal phenomenon or siphonic phenomenon" (1, 2, 8).

Hepatic vein obstruction

A decrease or stoppage of hepatic venous flow may occur under various conditions such as, hepatic vein obstruction by tumor or bland thrombus, extrinsic hepatic vein compression by tumor or hematoma, Budd-Chiari syndrome, right side heart failure, and mediastinal fibrosis (1, 2, 5). When the hepatic vein is obstructed, blood stasis within the hepatic sinusoid occurs and a reversal of the hepatic arterial flow to the portal vein occurs. As a result, the portal vein becomes a draining vein rather than supplying vein. Elimination of functional portal flow results in a compensatory increase in

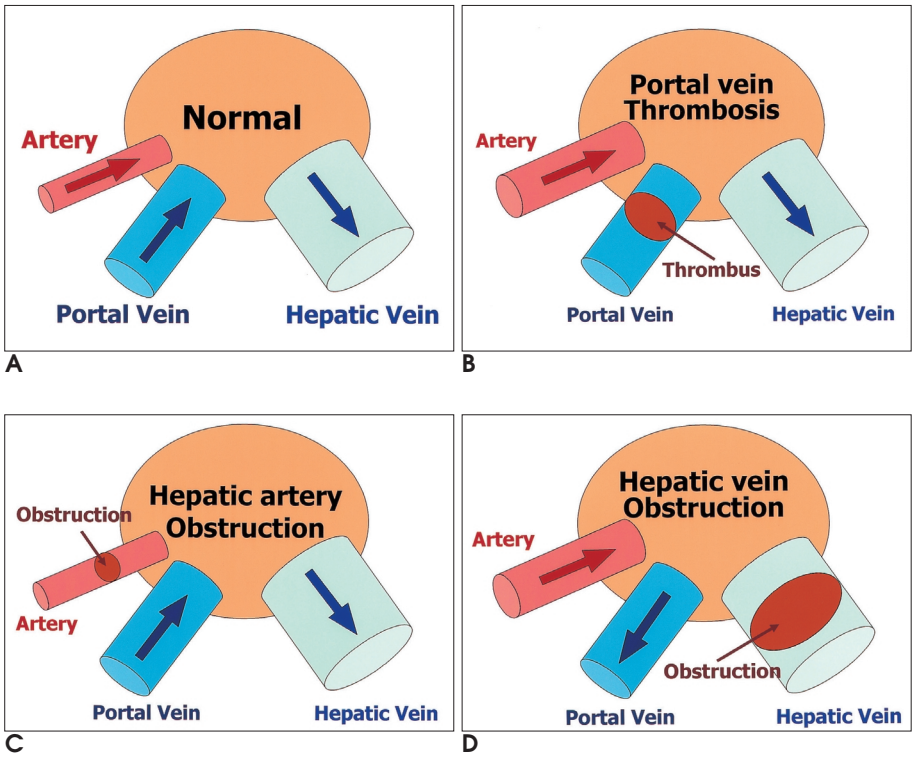
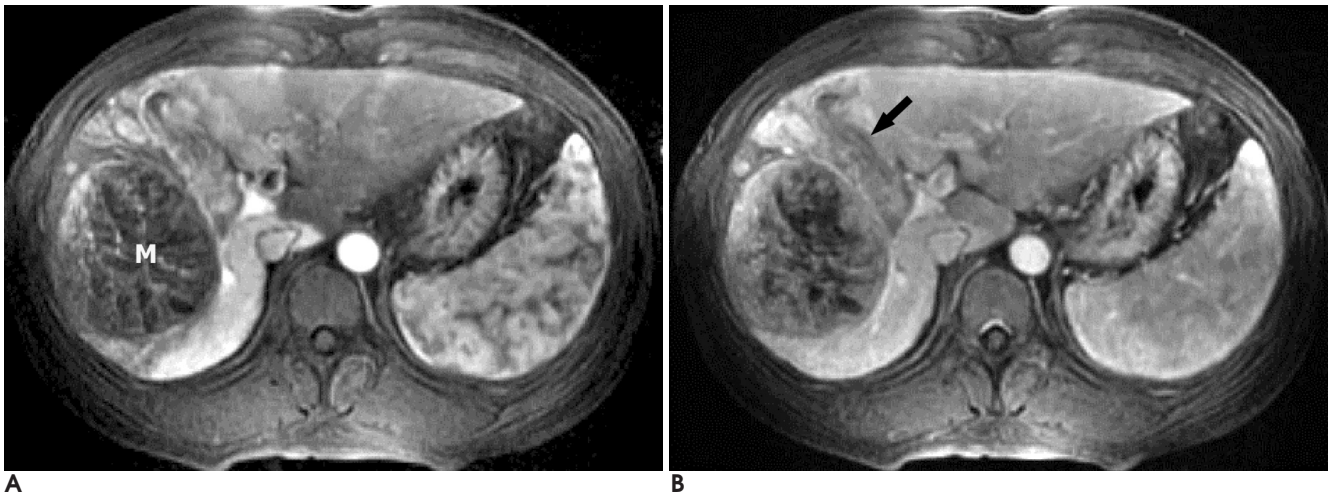


Fig. 6. Schema of normal and portal vein obstruction.
A. Normal blood flow and volume in the normal liver.
B. Hepatic arterial flow increases during portal venous obstruction.
C. Portal venous flow does not change during hepatic arterial occulusion.
D. Hepatic arterial flow increases during hepatic venous obstruction.



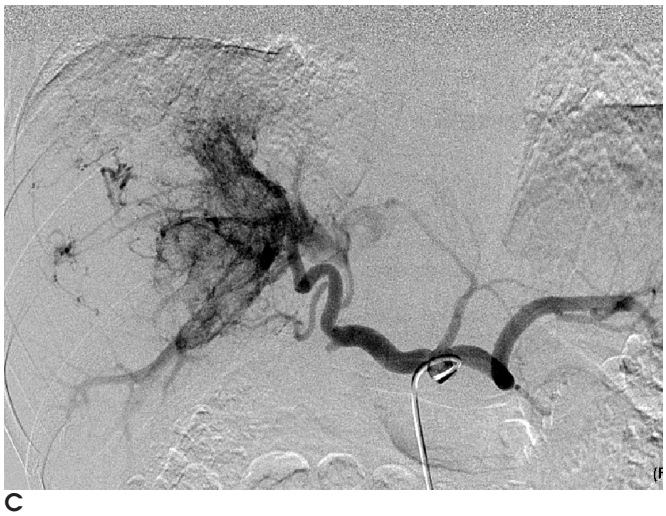


Fig. 7. Portal vein thrombosis secondary to HCC.
A. T1-weighted MR image during hepatic arterial phase shows heterogeneous enhancing HCC (M). The surrounding parenchyma is hyperperfusion compared with the contralateral lobe.
B. T1-weighted MR image during the portal venous phase reveals portal vein thrombosis (arrow).
C. Celiac angiogram demonstrates that shunted arterial flow is instantaneously drained into the portal branches in the entire liver.

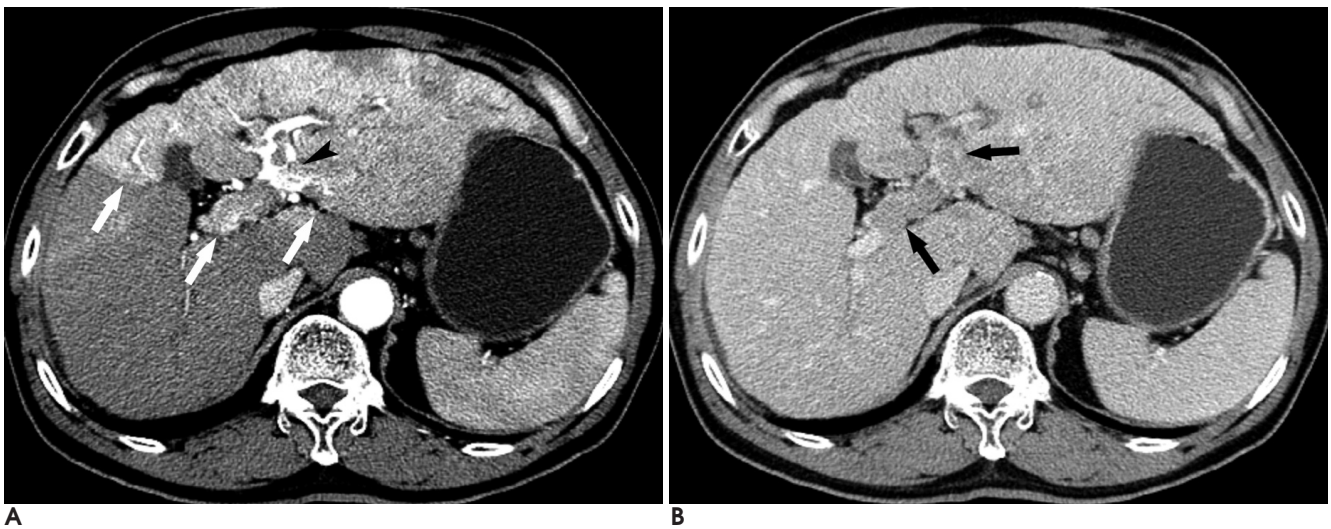
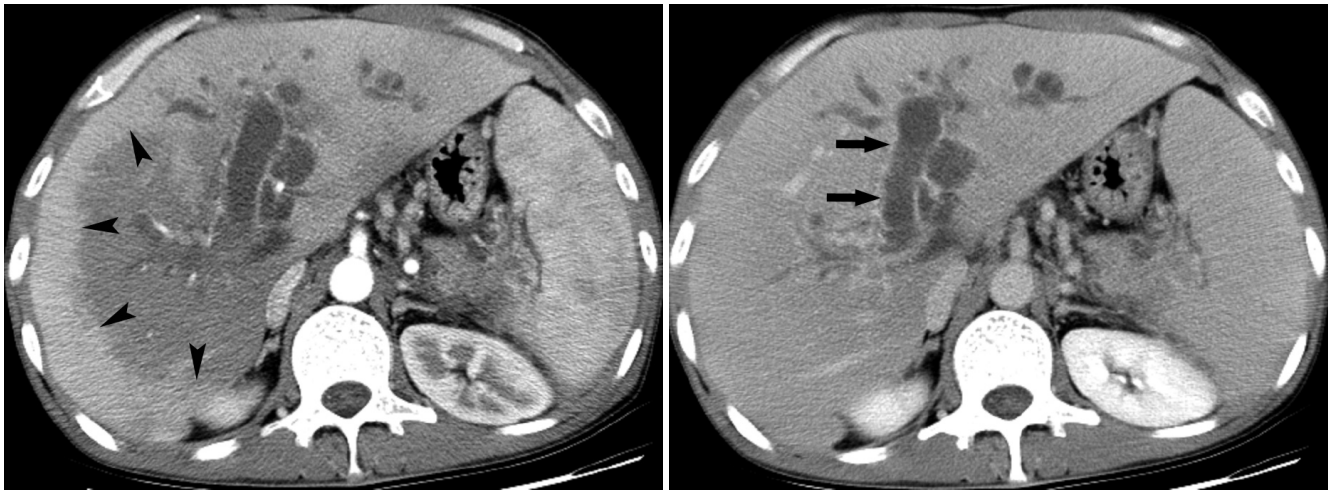


Fig. 8. Portal venous thrombosis secondary to HCC.
A. Hepatic arterial phase CT scan shows high attenuation in the right hepatic lobe caused by a compensatory increase in hepatic arterial blood flow. The straight border sign (arrows) is seen. Note that the marked enhancement along the thrombosed left portal vein represents a 'thread and streak' sign (arrowhead).
B. Portal venous phase CT scan reveals the involved area to be isoattenuating with the surrounding parenchyma and portal vein thrombosis (arrows).

hepatic arterial flow (3, 5) (Fig. 6).

In acute hepatic vein obstruction, HAP image demonstrate THAD in the area of the obstructed hepatic venous drainage similar to the findings in the case of portal venous flow stoppage (1), but THAD disappears on delayed phase image (Fig. 10).

Chronic venous obstruction has different imaging findings compared with acute hepatic venous obstruction. Budd-Chiari syndrome, right side heart failure and mediastinal fibrosis can cause chronic hepatic venous obstruction (1 - 3). HAP image shows diffuse inhomogeneous, or patchy enhancement of the hepatic parenchyma, resulting in a reticular or mosaic pattern (Figs. 11, 12). On delayed phase image, the hepatic parenchyma becomes homogeneous (2, 5). In Budd-Chiari syndrome, homogeneous enhancement and hypertrophy of the caudate lobe may be seen, because the caudate lobe has its own draining veins into the inferior vena cava (1 - 3, 5).



A B

Fig. 9. Main portal vein thrombosis.
A. Hepatic arterial phase CT scan shows an area of hyperattenuation (arrowheads) of the peripheral zone (the right hepatic lobe and the medial segment of the left hepatic lobe) and iso-attenuation of the central zone (the caudate lobe and the lateral segment of the left hepatic lobe) of liver. On arterial phase CT scan, the peripheral zone has increased hepatic arterial flow due to rich venous collateral circulation in the central zone compared with the peripheral zone.
B. Delayed phase CT shows no attenuation differences between the central and peripheral zones. There is a cavernous transformation and also a dilated intrahepatic bile duct (arrows).

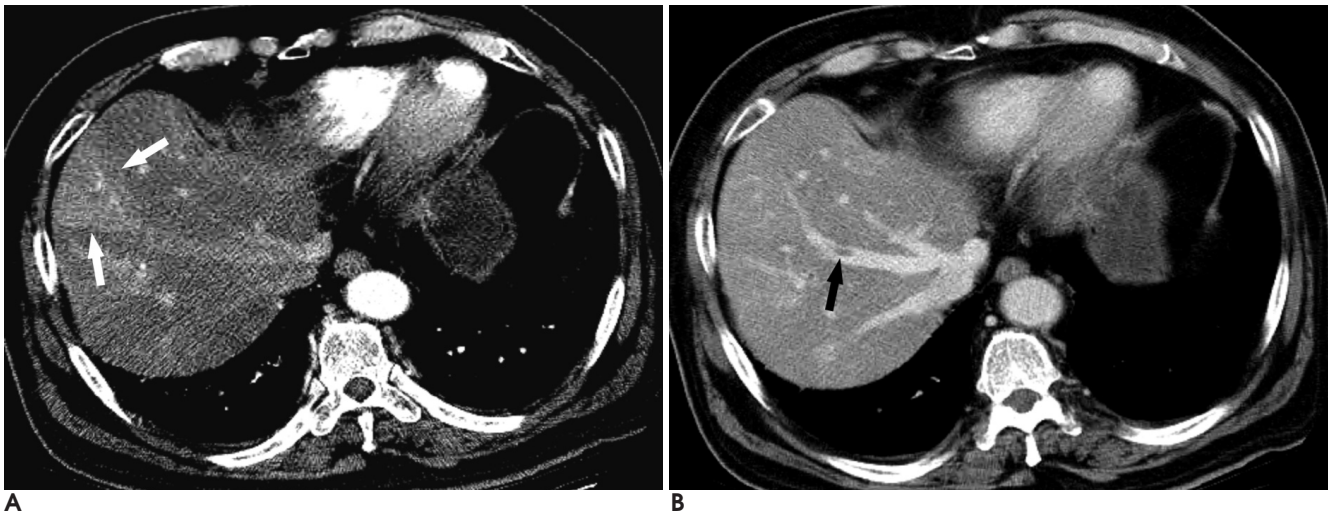


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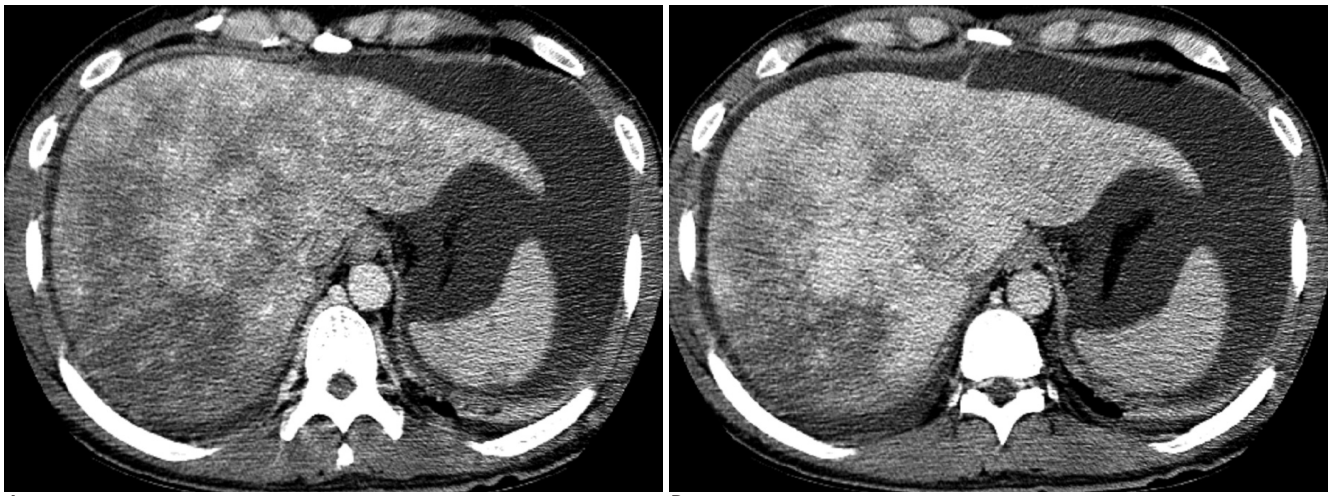
Fig. 10. Thrombus in the middle hepatic vein secondary to cholecystitis.
A. Hepatic arterial phase CT scan reveals wedge-shaped area of high attenuation (arrows) directed toward the middle hepatic vein.
B. Portal venous phase CT scan shows focal thrombus (arrow) within the middle hepatic vein.



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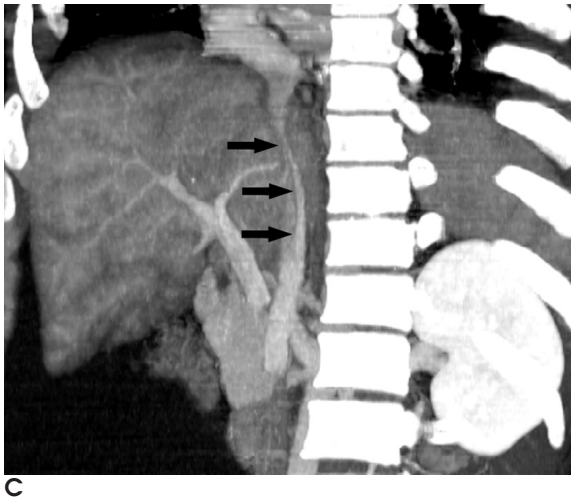
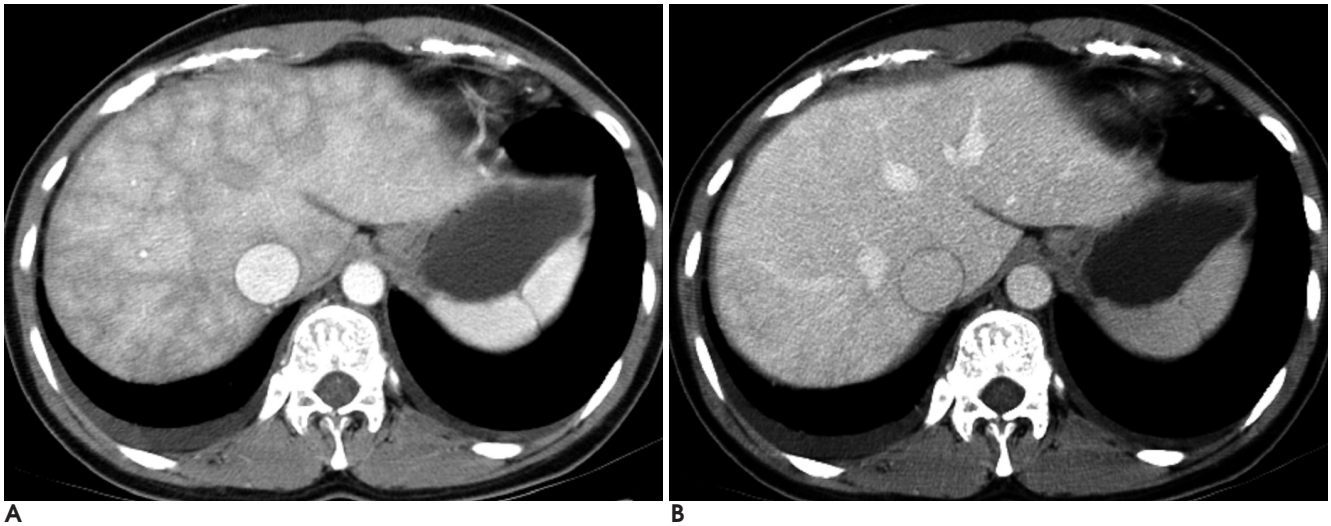


C

Fig. 11. Budd-Chiari syndrome. Hepatic arterial phase (**A**) and portal venous phase (**B**) CT scan show patchy enhancement in the entire liver. Hepatomegaly and ascites are seen.
C. Oblique coronal MIP image demonstrates slit-like narrowing of the inferior vena cava (arrows).



A B

Fig. 12. Perfusion disorder secondary to congestive heart failure.
A. Hepatic arterial phase CT scan shows geographic enhancement of the entire liver.
B. Portal venous phase CT scan reveals homogenous enhancement of the liver.

There are several differences between the findings in hepatic and portal venous obstruction (Table 3) (Fig. 13). First, the straight border at the attenuation difference within the liver intersects the portal vein during hepatic vein obstruction, whereas during portal venous obstruction, it intersects the hepatic vein. Second, the vertex of the wedge-shaped THAD points to the hepatic hilum or umbilical portion of the left portal vein during portal vein obstruction, whereas during hepatic venous obstruction it points toward the inferior vena cava. Third,

Table 3. Hepatic Vein versus Portal Vein Obstruction

	Hepatic vein occlusion	Portal vein occlusion
THAD*	Intersect portal vein	Intersect hepatic vein Hepatic hilum
Direction of the vertex of a THAD*	Inferior vena cava	Umbilical portion of left portal vein
Development of collaterals	Hepato-hepatic venous shunt	Large number of small collaterals (portal cavernoma)
Anatomic variations	Accessory hepatic vein (inferior right hepatic vein)	Systemic venous supply in periphery (Right aberrant gastric vein)

THAD*: transient hepatic attenuation difference

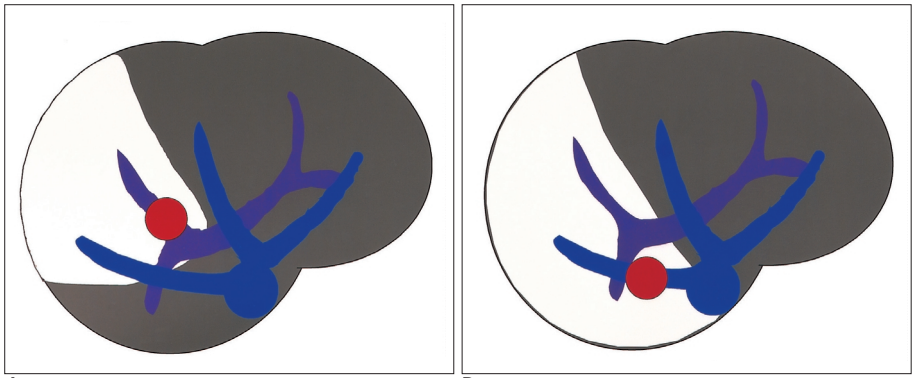


Fig. 13. Schema of portal vein obstruction (**A**) and hepatic vein obstruction (**B**). Obstruction of a given portal or hepatic vein generally causes blood flow changes of a certain extent, and these areas of blood flow change do not overlap.
A. Obstruction of the anterior portal vein due to a tumor (red) induces high attenuation (white) in the anterior segment. Apex of a highly attenuated area (white area) directed to the right portal vein is seen. Straight border almost intersects the right hepatic vein.
B. Obstruction of the right hepatic vein shows a highly attenuated area in the right hepatic lobe. Apex of lesion directed to IVC is noted. Straight border intersects the right portal vein.

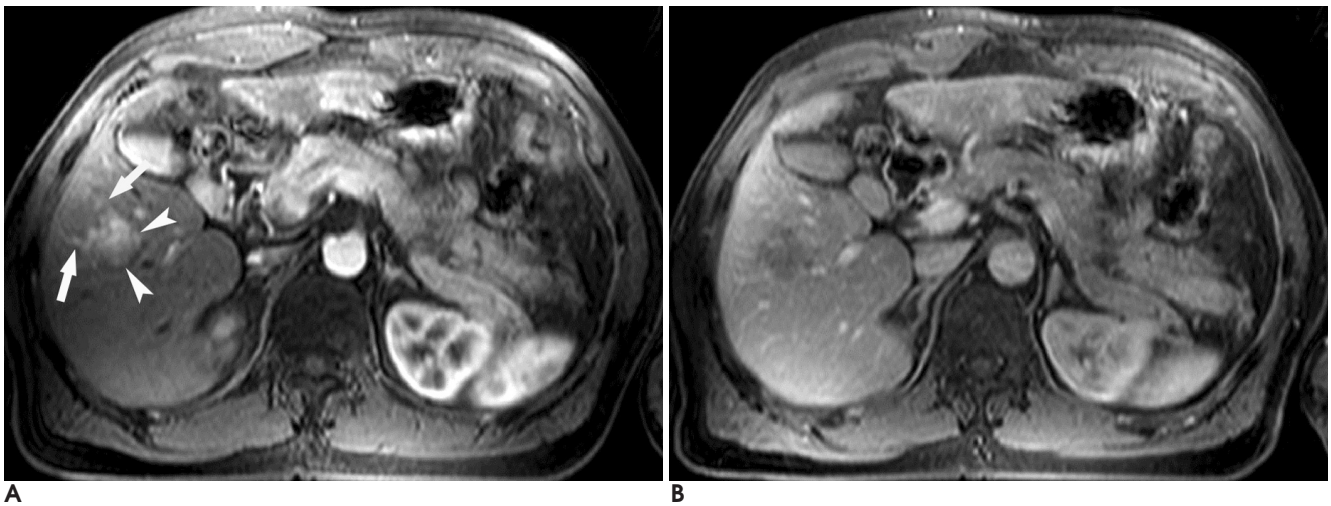


Fig. 14. Arterioportal shunt secondary to HCC.
A. T1-weighted MR image during hepatic arterial phase shows an enhancing HCC (arrowheads) in the right hepatic lobe. Wedge and tubular shaped hyperperfusion (arrows) is seen in the lateral portion of the tumor.
B. T1-weighted MR image during the portal venous phase reveals hypointense HCC. There is no difference in signal intensity between the area of hyperperfusion and normal parenchyma.

a large number of small collateral vessels may develop during portal venous obstruction (portal cavernoma), whereas the hepato-hepatic venous shunt may be conspicuous during hepatic venous occlusion. Fourth, vascular anomalies, may play an important role. During portal venous obstruction, systemic veins (esp. right aberrant gastric vein) supply the peripheral region of the liver . During hepatic vein obstruction, accessory hepatic veins (esp. inferior right hepatic vein) may be the major hepatic venous drainage route (6, 8).

Hepatic tumor

Hepatic malignant tumors, such as HCC, cholangiocarcinoma and hepatic metastasis can induce AP shunts. In addition, benign hemangioma can also induce AP shunts (5, 6). These hepatic tumors (usually HCC) are sometimes associated with portal vein compromise and less frequently hepatic vein compromise by compression or direct invasion (1, 5, 6). In the case of HCC, AP shunts are induced by several mechanisms as follows; (a) transtumoral shunt, venous drainage of the tumor in-

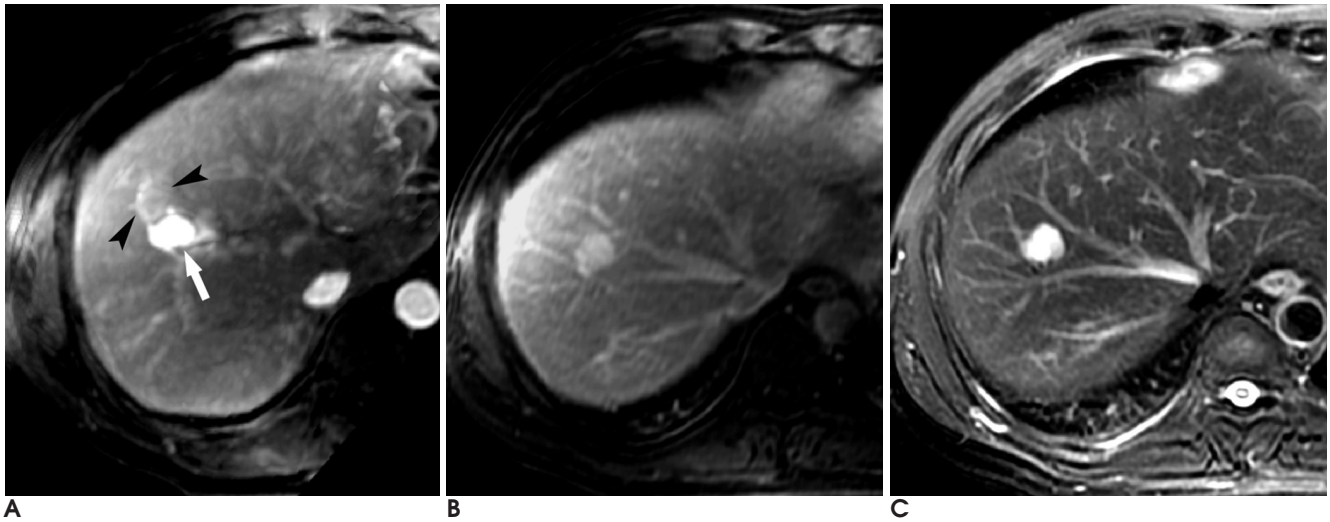


Fig. 15. Arterioportal shunt secondary to a hemangioma.
A. T1-weighted MR image during the hepatic arterial phase reveals focal wedge-shaped hyperintense area (arrowheads) due to an arterioportal shunt, as well as intense enhancing hemangioma (arrow).
B. T1-weighted MR image during the portal venous phase show a persistent enhancing hemangioma. Area of hyperperfusion returns to normal enhancement pattern.
C. T2-weighted fast spin echo MR image shows a small hemangioma with bright signal intensity (arrow).

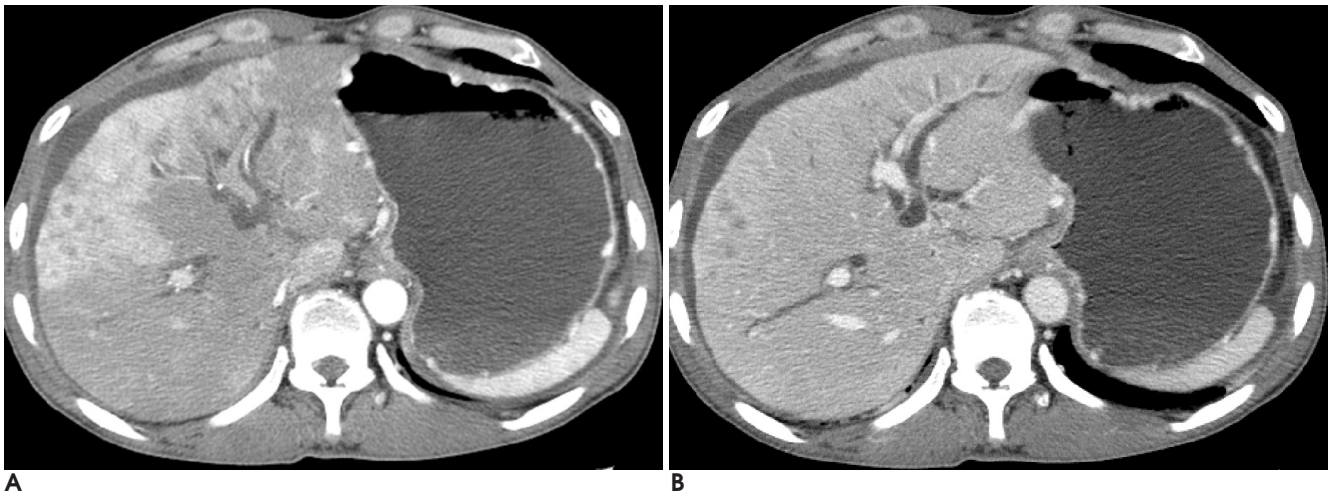


Fig. 16. Multifocal perfusion disorder secondary to metastasis from a pancreatic carcinoma.
A. Hepatic arterial phase CT scan shows a multifocal area of heterogeneous enhancement in the anterior segment and the left hepatic lobe. Ascites is seen.
B. Portal venous phase CT scan reveals the involved areas to be isoattenuating with the surrounding parenchyma.

to the peripheral portal vein. (b) transplexal shunt, when the portal vein is compromised. (c) transvasal shunt, through the tumor thrombus in the portal vein via the hypertrophied vasa vasorum. This shunt often causes a "thread and streak" sign. (d) transsinusoidal shunt, between microscopic hepatic arterioles and portal venules distal to the portal vein compression or thrombosis. (e) steal phenomenon, hypervascular tumor can "steal" arterial blood from its surrounding parenchyma. On CT, in such cases, the parenchyma surrounding a hypervascular tumor appears hypoattenuating on hepatic arterial phase imaging. (f) macroscopical AP shunt is also possible in HCC (Fig. 7) (1 - 3, 5, 6).

On HAP image, hepatic tumors can produce a fan or wedge shaped THAD (Figs. 14 - 16). Generally, fan shaped THAD are induced by portal vein compression and wedge shaped THAD by proximal tumor thrombosis or AP shunt (1, 3).

Inflammatory disease

Inflammatory lesions such as liver abscesses, acute cholecystitis and acute cholangitis can induce perfusion disorders. Local inflammation can cause hyperemia of the hepatic artery and stoppage of regional portal venous flow. This can induce the development of a functional AP shunt around the lesions. In the case of acute cholecystitis, THAD may be induced by increased blood flow from the dilated aberrant cystic vein of the diseased gall bladder or by thrombosis of the regional portal or hepatic veins (1, 6).

On HAP image, hyperattenuation is seen around the gallbladder fossa (Figs. 17, 18). This lesion becomes nor-

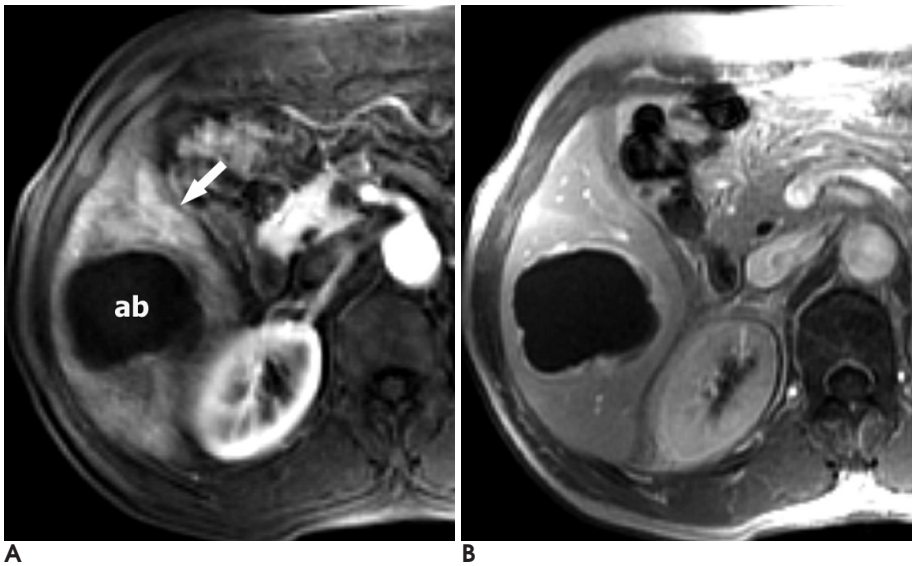


Fig. 17. Perfusion disorder secondary to a liver abscess.
A. T1-weighted MR image during the hepatic arterial phase shows a hepatic abscess (ab), with wedge-shaped area of hyperperfusion (arrow) in the adjacent hepatic parenchyma.
B. T1-weighted MR image during the hepatic venous phase depict the concentric enhancement of the abscess wall, and parenchymal enhancement has returned to normal.

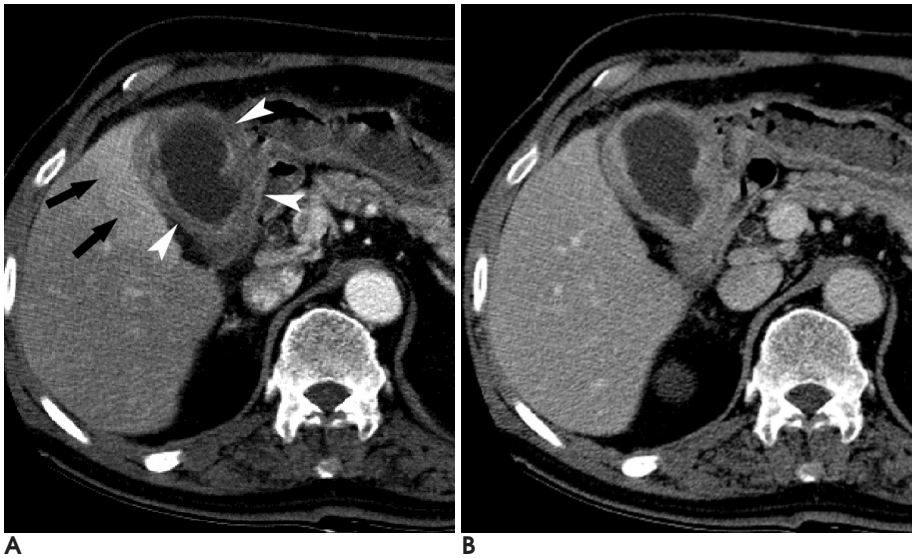


Fig. 18. Hyperpefusion secondary to an acute cholecystitis.
A. Hepatic arterial phase CT scan reveals cholecystitis (arrwoheads) with wedge shaped hyperattenuation (arrows) in the hepatic parenchyma adjacent to the gall bladder.
B. Portal venous phase CT scan reveals homogenous enhancement of the entire liver.

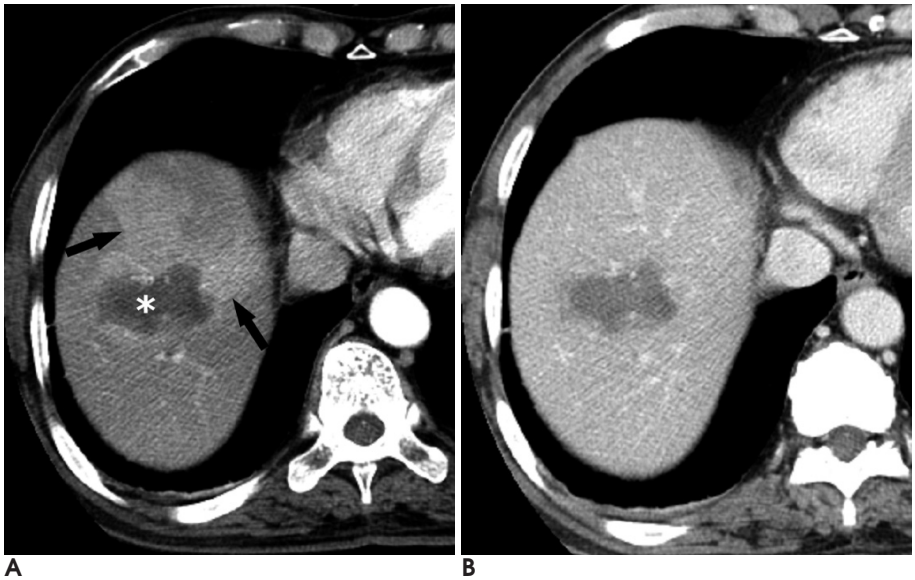
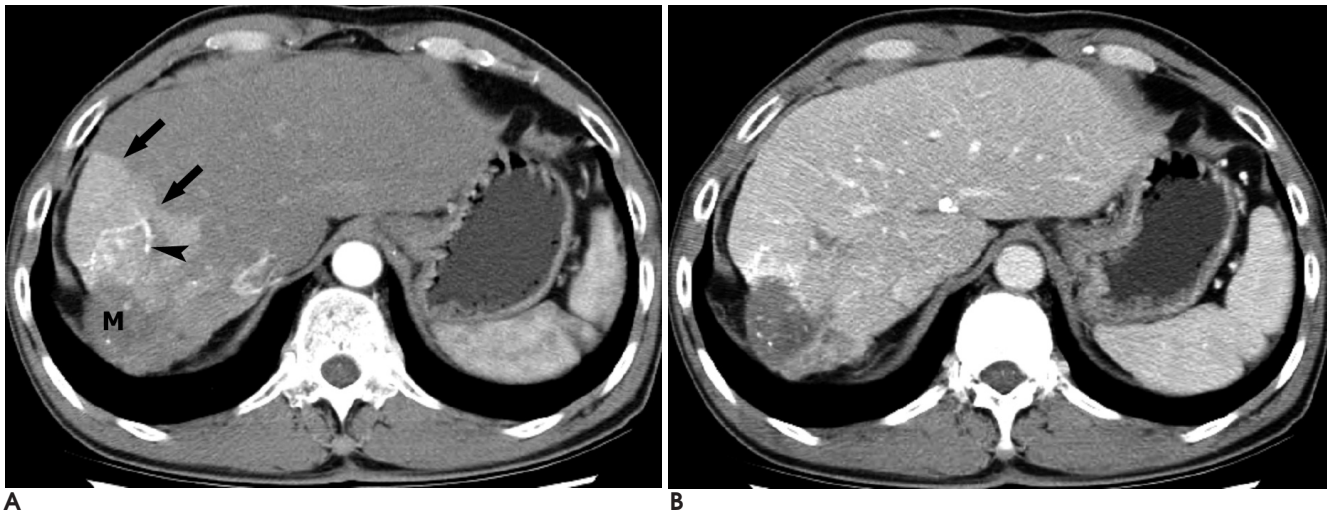


Fig. 19. Hyperperfusion after radiofrequency ablation (RFA) therapy.
A. Hepatic arterial phase CT scan shows a rectangular shaped area of hyperperfusion (arrows) adjacent to the hepatic parenchyma of RFA lesion (*) in the hepatic dome.
B. On the portal venous phase CT scan, the area of hyperpefusion returns to normal enhancement pattern.



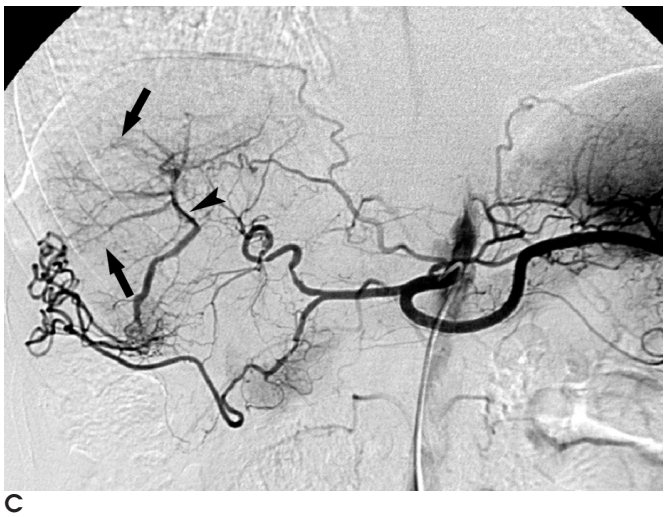


Fig. 20. Hyperpefusion secondary to Transarterial chemoembolization.
A. Hepatic arterial phase CT scan reveals a wedge shaped hyperperfusion (arrow) in the right hepatic lobe with early opacificaton of the peripheral portal vein (arrowhead). The low attenuated lesion is HCC (M).
B. Portal venous phase CT scan shows homogenous enhancement of the liver and tiny lipiodol uptake within the hypoattenuated HCC.
C. Celiac angiogram depicts wedge shaped staining (arrows) in the corresponding area and retrograde opacification of the peripheral portal vein (arrowhead).

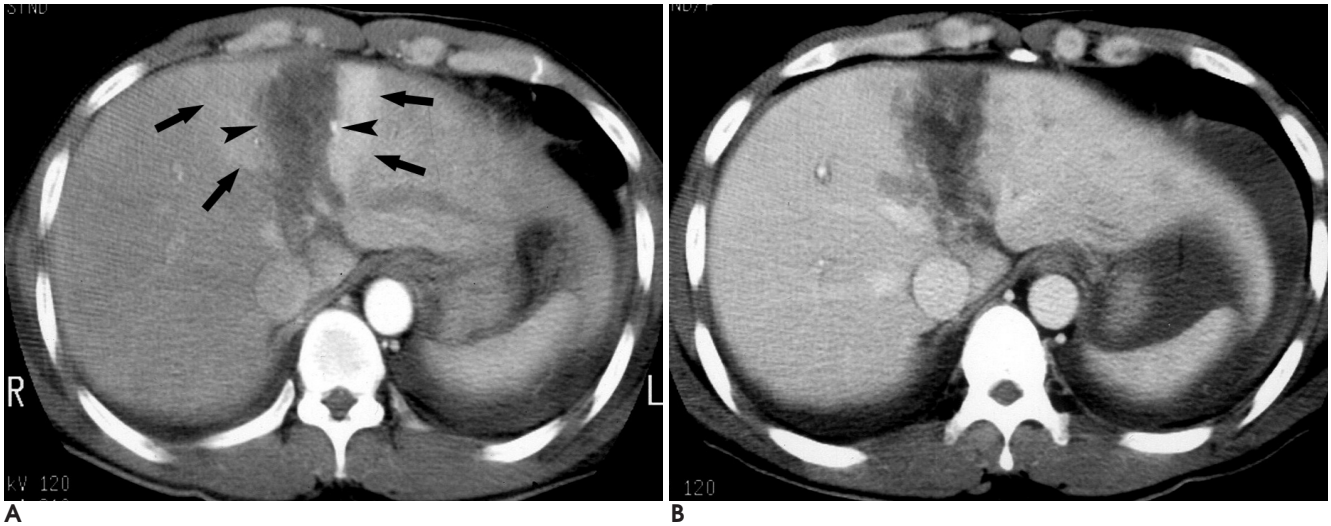
mal attenuation on portal venous phase image (1, 2, 5, 6).

Iatrogenic and Trauma

Interventional procedures (radiofrequency ablation, percutaneous ethanol injection therapy, transarterial chemoembolization, liver biopsies) and abdominal trauma sometimes cause an organic communication between the arterial and portal venous systems or a functional AP shunt. On HAP image, THAD around the therapeutic or traumatic lesion is noted (Figs. 19 - 21). Sometimes hepatic artery pseudoaneurysm may be seen, which appear as focal, round, enhancing lesions on HAP images (1).

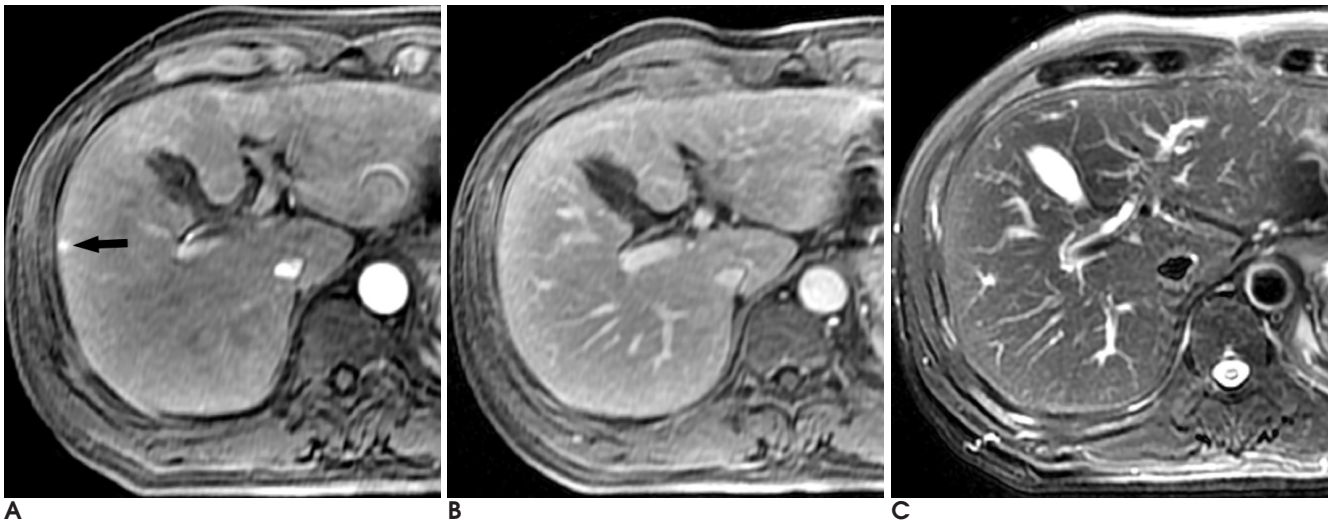
Liver cirrhosis

Liver cirrhosis is a well known cause of the alteration of normal hepatic flow dynamics, resulting in increased arterial flow and decreased portal venous flow to the liver (1). Nontumorus AP shunts in liver cirrhosis are believed to be secondary to the occlusion of small hepatic



A B

Fig. 21. Hyperpefusion secondary to a hepatic contusion with trauma.
A. Hepatic arterial phase CT scan reveals a wedge shaped hyperpefusion lesion (arrows) in the hepatic parenchyma adjacent to the hepatic contusion (arrowheads).
B. Corresponding portal venous phase CT scan shows normal attenuation of the involved area around the hepatic contusion.



A B C

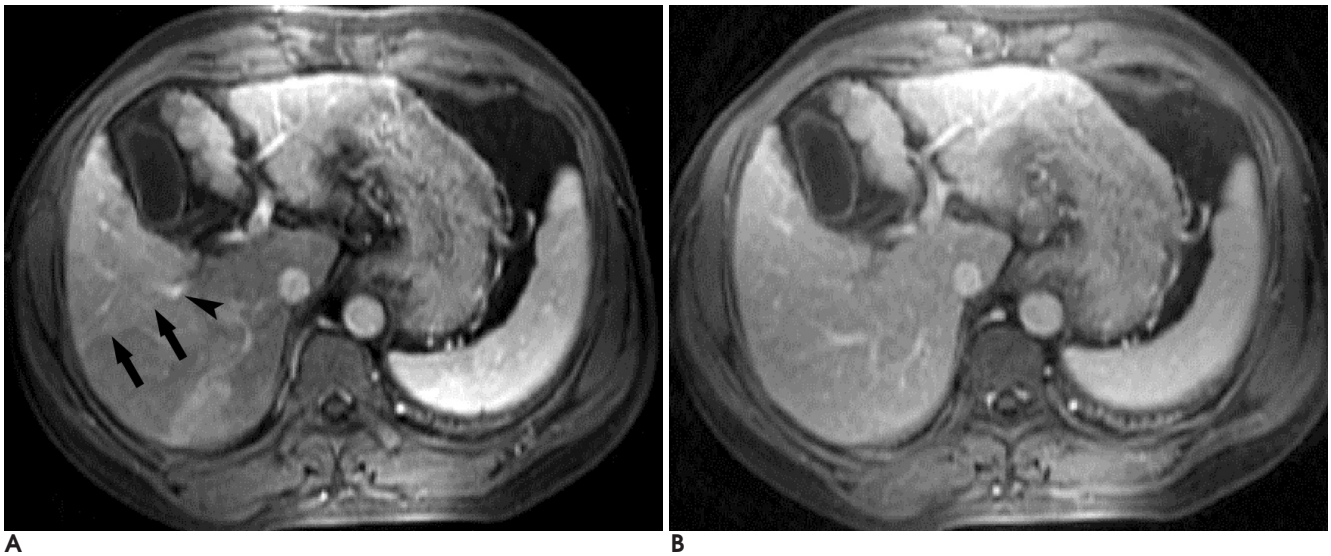
Fig. 22. Liver cirrhosis with pseudolesion.
A. T1-weighted MR image during hepatic arterial phase depicts a small nodular enhancement (arrow) on the subcapsular portion of the right hepatic lobe. This lesion mimics small HCC.
B. T1-weighted MR image during the portal venous phase shows no abnormal enhancement.
C. T2-weighted fast spin echo MR image reveals iso-signal intensity corresponding nodular lesion.

venules and retrograde filling of the small portal vein branches by way of arterioportal anastomosis (transsinusoidal shunt). As a result, the portal vein becomes the draining vein rather than supplying vein (hepatofugal flow) and it compensates by increasing hepatic arterial flow (1, 6). AP shunts in liver cirrhosis are wedge shaped, early enhancing lesions in HAP images (Figs. 22, 23), but normal enhancement during the portal venous phase and delayed phase image.

Discrimination between AP shunts and small HCC is important. AP shunts are usually wedge or triangular shaped. Homogeneous enhancements with early visualization of portal branches during HAP image and iso- or slight hypoattenuation during portal venous phase image also suggest findings of normal AP shunts (8). T1- and T2-weighted MR images show iso signal intensity in most cases of AP shunts (7).

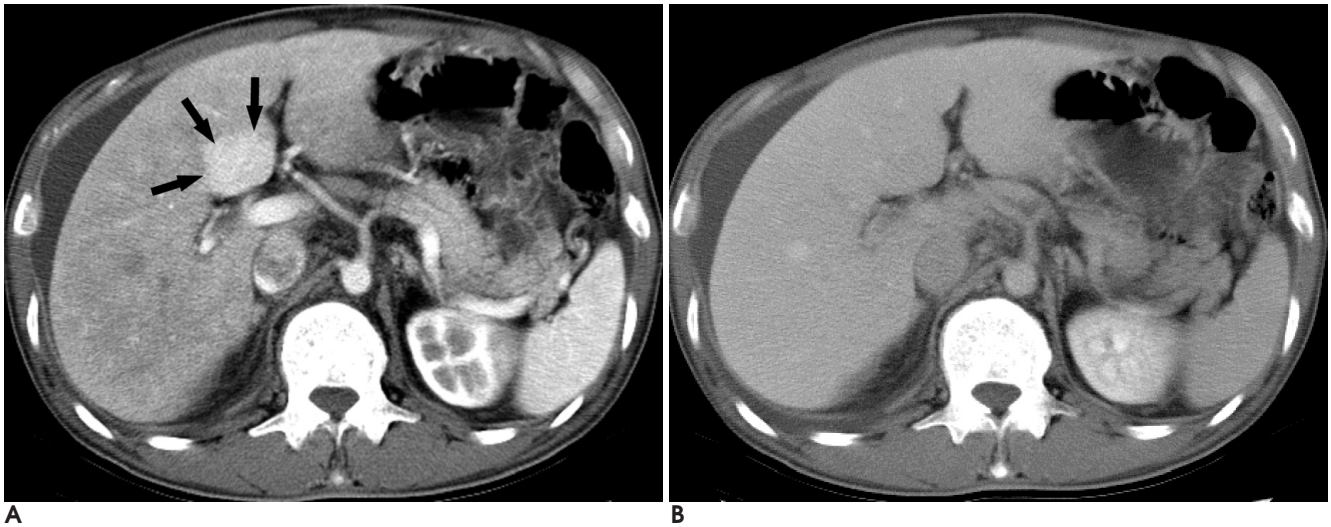
Aberrant venous drainage

The liver receives blood mainly from the portal vein



A B

Fig. 23. Liver cirrhosis with pseudolesion.
A. T1-weighted MR image during the hepatic arterial phase shows wedge shaped areas of hyperperfusion (short arrows) in the right hepatic lobe. Early opacification of the peripheral portal branches (long arrows) is seen.
B. T1-weighted MR image during the portal venous phase shows no focal enhancement.



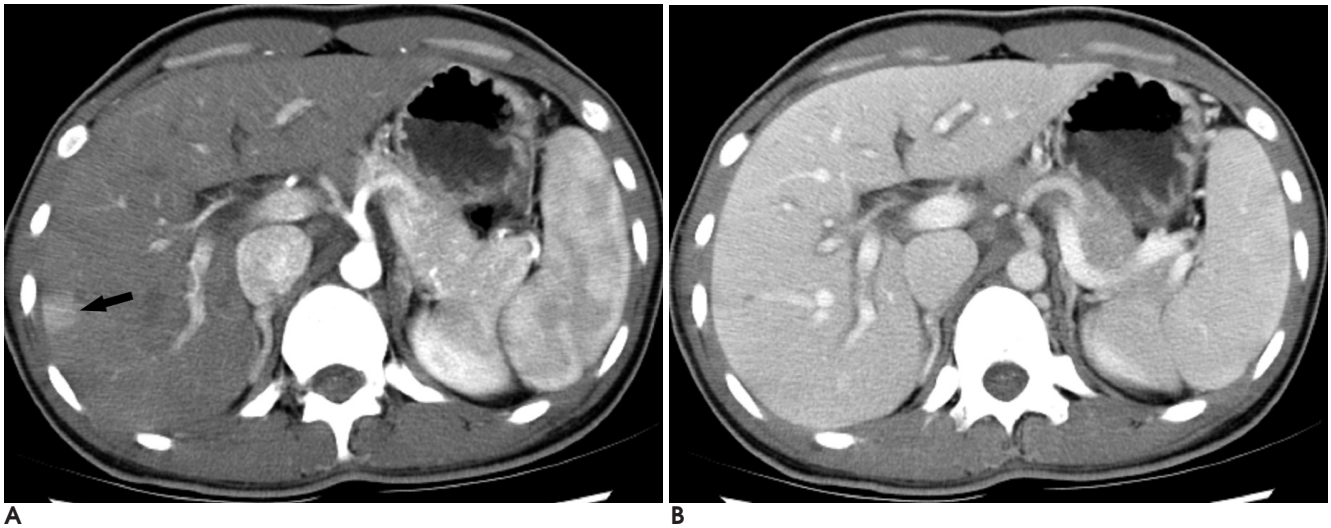
A B

Fig. 24. Hyperpefusion due to an aberrant right gastric vein.
A. Hepatic arterial phase CT scan reveals a round hyperattenuated lesion in the medial segment (IV) of the left hepatic lobe.
B. Portal venous phase CT scan reveals homogenous enhancement of segment IV compared with the adjacent hepatic parenchyma. Angiogram showed an aberrant right gastric vein.

and hepatic artery. However, some aberrant vessels supply the liver from areas other than the portal trunk and hepatic artery. This unusual pattern of vascular supply can cause some pseudolesions. Table 4 shows the typical site of common pseudolesion associated with aberrant vessels. There are two kinds of veins that supply venous blood to the liver other than from portal venous flow, veins originating from non-portal splanchnic veins, such as the cystic vein and the parabiliary venous system and systemic veins such as inferior and superior vein of Sappey (9). On HAP images, these lesions are focal hyperattenuated lesions around segment II, segment III or the subcapsular area in the hepatic parenchyma due to earlier venous return of contrast materials by

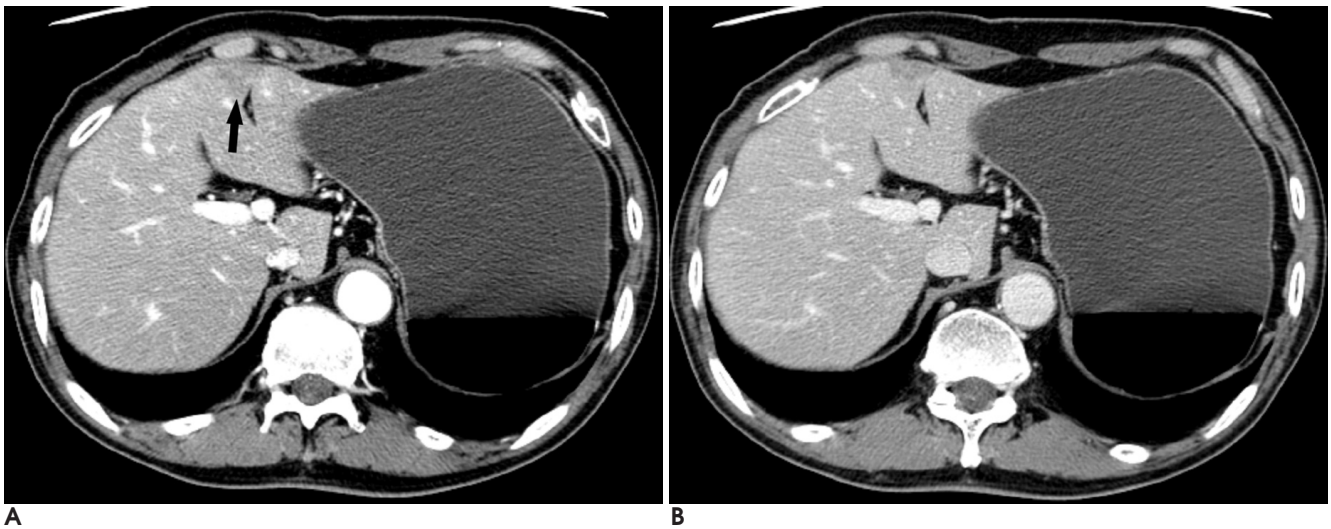
Table 4. Aberrant Vein and Common Locations of Pseudolesion

Aberrant vein	Common location
Aberrant gastric vein	Segment II and III
Parabiliary vein and cystic vein	Around gall bladder fossa
Capsular vein	Subcapsular area
Paraumbilical vein (inferior vein of Sappey)	Around falciform ligament



A B

Fig. 25. Pseudolesion due to an aberrant capsular vein.
A. Hepatic arterial phase CT scan reveals a wedge shaped hyperpefusion area (arrow) in the right hepatic lobe.
B. On portal venous phase CT scan, this lesion depicts an iso-attenuation corresponding area.



A B

Fig. 26. Perfusion defect around the falciform ligament.
A. Hepatic arterial phase CT scan reveals a wedge shaped low attenuation pseudolesion (arrow) around the falciform ligament via the the epigastric-paraumbilical venous system.
B. On portal venous CT scan, the low-attenuation pseudolesion is seen persistently due to delayed inflow by epigastric-paraumbilical vein.

non-portal splanchnic veins and systemic veins compared with portal veins from the intestine and spleen (9, 10). (Figs. 24, 25). On the contrary, nodular hypoattenuation around the falciform ligament on HAP images are also seen (Fig. 26).

Miscellaneous

Extrinsic compression of the hepatic surface, including compression by the ribs, perihepatic peritoneal implants, pseudomyxoma peritonei, and perihepatic fluid collection can cause focal increases in tissue pressure, resulting in decreased portal venous perfusion and little change in hepatic arterial perfusion. Confluent fibrosis in liver cirrhosis can cause decreased portal venous perfusion and slight increases in hepatic arterial perfusion. Congenital AP shunts, although rare, are possible including hereditary hemorrhagic telangiectasis (Osler-Weber-rendu disease), Ehlers-Danlos syndrome and others (1, 6).

Summary

With rapid image acquisition and increased resolution available in multislice CT and MR imaging, hepatic perfusion disorders are now more frequently encountered than in the past. The major cause of perfusion disorders are AP shunts. Functional or pathologic AP shunts are secondary to a decrease or stoppage of portal venous flow. As a result, hepatic arterial flow increases and flow to the regional portal venous system occurs. This can cause hyperattenuation or hyperintensity on HAP images. These are common causes of pseudolesion of hepatic imaging.

References

1. Quiroga S, Sebastia C, Pallisa E, Castella E, Perez-Lafuente M, Alvarez-Castells A. Improved diagnosis of hepatic perfusion disorders: value of hepatic arterial phase imaging during helical CT. *Radiographics* 2001;21 :65-81
2. Gryspeerdt S, Van Hoe L, Marchal G, Baert AL. Evaluation of hepatic perfusion disorders with double-phase spiral CT. *Radiographics* 1997;17:337-348
3. Itai Y, Matsui O. Blood flow and Liver imaging. *Radiology* 1997; 202:306-314
4. Yu JS, Rofsky NM. Magnetic resonance imaging of arterioportal shunts in the liver. *Top Magn Reson Imaging* 2002;13:165-176
5. Choi BI, Chung JW, Itai Y, Matsui O, Han JK, Han MC. Hepatic abnormalities related to blood flow: evaluation with dual-phase helical CT. *Abdom Imaging* 1999;24:340-356
6. Choi BI, Lee KH, Han JK, Lee JM. Hepatic arterioportal shunts: Dynamic CT and MR features. *Korean J Radiol* 2002;3:1-15
7. Matsuo M, Kanematsu M, Kondo H, Maeda S, Goshima S, Suenaga I. et al. Arterioportal shunts mimicking hepatic tumors with hyperintensity on T2-weighted MR images. *J Magn Reson Imaging* 2002;15:330-333
8. Itai Y, Murata S, Kurosaki Y. Straight border sign of the liver: spectrum of CT appearances and causes. *Radiographics* 1995;15:1089-1102
9. Itai Y, Matsui O. 'Nonportal' splanchnic venous supply to the liver: abnormal findings on CT, US and MRI. *Eur Radiol* 1999;9:237-243
10. Yoshimitsu K, Honda H, Kuroiwa T, Irie H, Aibe H, Shinozaki K, et al. Unusual hemodynamics and pseudolesions of the noncirrhotic liver at CT. *Radiographics* 2001;21:S81-S96

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간의 혈액공급은 간문맥과 간동맥을 통해 주로 이루어지며, 이들 혈관계 사이에는 여러 교통이 있다. 간암, 간외상, 그리고 간경화 등의 질환에서 이러한 혈관계의 이상이 발생할 수 있으며, 이러한 경우 간문맥계와 간동맥계를 통한 혈액공급 체계에 변화가 발생한다. 이러한 변화는 각각의 혈관계를 통한 관류양의 변화 혹은 관류방향의 변화로 발생한다. 다중절편 CT와 MRI통해 영상획득이 빨라지면서 이러한 간의 관류이상은 이전과 비교하여, 좀 더 흔히 관찰되는 소견이다. 따라서 이러한 관류이상의 영상소견을 숙지하는 것이 간의 병변을 정확하게 감별 진단하는데 도움을 준다.