Radiologic manifestation of hepatic pseudolesions and pseudotumors in the third inflow area

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Radiologic manifestation of hepatic pseudolesion and pseudotumor in the third inflow area

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Summary

We sometimes encounter hepatic pseudolesions and pseudotumors of the third inflow area on imaging. Generally, to differentiate these lesions of true hepatic neoplasm on radiological examinations are easy with the knowledge of common site in the liver and characteristic findings of these lesions. However, it occasionally mimic hepatocellular carcinoma in various imaging modalities. In this article, we are going to provide current knowledge about pseudolesions and pseudotumors in the third inflow area on imaging. To have knowledge about pseudolesions and pseudotumors in the third inflow area can aid in correct diagnosis and avoid unnecessary treatment.

Key Words

Pseudolesion, Pseudotumor, Third inflow, CT during arterial portography, CT during hepatic arteriography, Parabiliary venous system, Focal fatty liver, Focal sparing of fatty liver, Focal hyperplastic change, Aberrant gastric venous drainage

Introduction

Various types of pseudolesions (focal mass-like findings seen only on imaging) or pseudotumors (focal mass-like parenchymal change) are observed in the liver. In order to accurately diagnose true mass lesions based upon imaging, it is necessary to exclude these entities. Pseudolesions or pseudotumors observed in the third inflow area occasionally mimic hepatocellular carcinoma in various imaging modalities. In this article, we review the etiology and characteristic findings of hepatic pseudolesions and pseudotumors in the third inflow area.

Overview of liver hemodynamics

The liver has a dual blood supply consisting of the hepatic artery and portal vein. About 75% of hepatic blood supply comes from the portal vein, and 25% from the hepatic artery. Portal venous flow primarily supplies hepatic sinusoids, while terminal hepatic arterial branches form peribiliary vascular plexuses supplying the bile ducts, which then drain into hepatic sinusoids. Both vascular systems have several communications, including transsinusoidal, transvasal, and transplexal routes [1].

There are reciprocal complementary supporting mechanisms of blood supply between portal venous flow and hepatic arterial flow through these communications. The transplexal route is thought to be the dominant pathway in the setting of portal vein obstruction, occlusion, or increased sinusoidal pressure.

Due to its dual blood supply, liver infarction is uncommon under physiological conditions.

Angiography-assisted CT for analysis of hepatic hemodynamics

Matsui et al. first introduced CT with arterial portography (CTAP) for the whole liver for the precise detection of hepatic neoplasms [2].

CTAP is considered the most sensitive modality for detection of small hepatic lesions, particularly small hepatic tumors, such as hepatocellular carcinoma and metastatic tumors. CT during hepatic arteriography (CTHA) can demonstrate both hypervascular and hypovascular tumors, and can estimate the grade of malignancy of nodular hepatocellular lesions in cirrhotic livers [3-5].

These techniques are based on the different characteristic of blood supply to the liver parenchyma and hepatic neoplasm. That is, hepatic parenchyma is mainly supplied by portal venous flow, while most hepatic lesions (such as hepatocellular carcinoma and metastatic liver tumors) are supplied by hepatic arterial flow. Thus, on CTAP images, most hepatic lesions show a portal perfusion defect and appear as a hypodense lesion

within opacified normal hepatic parenchyma. Additionally, using CTHA most metastatic liver tumors and hepatocellular carcinoma show some degree of hyperdensity within the relatively hypodense hepatic parenchyma. Furthermore, some precancerous lesions and well-differentiated hepatocellular carcinomas in cirrhotic livers are depicted as a hypodense mass compared to the hepatic parenchyma (Figure 1).

Details of CTAP and CTHA procedures are as follows. From the femoral artery, 4-F catheters are inserted into the superior mesenteric artery for CTAP, and into the common, proper, or replaced right hepatic artery for CTHA.

CTAP scans were obtained in sections of 5–7-mm thick and 5–7-mm collimation to cover the entire liver in a single patient breath. To increase blood flow and decrease laminar flow in the portal vein, 5 μg of prostaglandin E1 (Palux; Taisho, Tokyo, Japan) was injected into the superior mesenteric artery prior to contrast material infusion. A total of 50-70 mL of iohexol (320-350 mg/mL iodone) (Omnipaque; Daiichi, Tokyo, Japan) was infused at a rate of 1.8 mL/sec with a power injector. Helical scanning began 25 seconds after infusion.

CTHA scans were obtained in sections of 3–5-mm thickness and 3–5-mm collimation. Helical scanning was started 7 seconds after the beginning of an iohexol infusion (320–350 mg/mL iodine) into the common, proper, or replaced hepatic artery at a rate of 1.8 mL/sec. Contrast infusion was continued until 5 seconds after early-phase CTHA scanning was completed. Scanning time varied according to the individual liver size (about 20–25 seconds). The total amount of contrast medium varied according to the following equation: (early-phase scanning time + 12 seconds) × injection rate. Thirty seconds after contrast material infusion finished (about 62–67 seconds after the infusion began), late-phase scanning started.

Because CTAP is very sensitive at detecting areas of decreased regional intrahepatic portal flow, various types of pseudolesions mimicking true space-occupying lesions in the liver have also been reported [6]. Due to the invasiveness of the procedure and the relatively high incidence of pseudolesions, the use of this technique in western countries is limited. In other words, CTAP is not a commonly performed procedure in the USA nor in many other countries. However, in Japan, CTAP and CTHA are still performed for closer examination for hepatocellular carcinoma and related nodular lesions such as dysplastic nodules, because we can estimate the grade of malignancy of nodular hepatocellular lesions in cirrhotic livers [3-5].

Pseudolesion of the liver

"Pseudolesion" is defined as a focal mass-like finding seen only on imaging studies without real parenchymal change.

The pseudolesions seen at CTAP are usually caused by obstruction of the intrahepatic portal veins by tumors or other pathologic processes, arterioportal shunting from a variety of causes, and laminar blood flow causing insufficient mixing of opacified and nonopacified portal blood [6] (Figure 2).

In the liver, these pseudolesions have been seen most frequently surrounding the gallbladder fossa, and at the posterior aspect of the medial segment, immediately anterior to the right portal vein.

Matsui et al. reported that drainage of cystic veins into the intrahepatic portal veins surrounding the gallbladder fossa may be the main cause of these pseudolesions. Areas in the liver drained by cystic veins occasionally appear as hypo-attenuating areas on CTAP because the contrast medium in the portal blood from the superior mesenteric vein is diluted by blood from cystic veins, which does not contain contrast medium [7].

Peterson et al [8] reported that there are two characteristic locations of pseudolesions in the left lobe of the liver. One is in the anteromedial aspect of the medial segment, which is a commonly recognized location of focal fatty change. The other is the posterior aspect of the medial segment, immediately anterior to the porta hepatis. Fernandez MdP, et al [9] reported that pseudolesions in the posterior aspect of the medial segment of the liver were found in 14% of CTAP examinations. This region of the medial segment of the liver is known to be focally spared from fatty change [10]. These regions of the medial segment of the liver may receive collateral circulation and show portal perfusion defect on CTAP [8,9].

Classification of third inflow vessels and imaging findings of pseudolesion of the liver in third inflow area

Usually, splanchnic venous flow, such as the drainage from the spleen and intestines, forms the portal vein and flows into the liver at the hepatic hilum.

However, some of the venous flow does not join the portal vein in the extrahepatic portion and directly enters the liver independently, then flows into the hepatic

sinusoids.
These are called “third inflow” vessels to the liver (Figure 3).
Darnert defined the “third inflow” to the liver as follows: aberrant veins supplying small areas of liver tissue and communicating with intrahepatic portal vein branches [11].
There are two kinds of veins that supply venous blood to the liver: veins originating from a digestive organ, such as the cholecystic vein and parabiliary venous system, and systemic veins, such as the epigastric-paraumbilical venous system, and capsular veins [12].

1) Pseudolesion in the drainage area of parabiliary venous system

Matsui et al [6] summarized the previous descriptions about the parabiliary venous system as follows; In 1859, Sappey described a case in which the right gastric vein ascended parallel to the main portal vein and drained into the left lobe of the liver. Because the right gastric vein branched out within the liver in the same manner as the portal vein, he regarded it as an accessory portal vein [13]. Michels also described the same type of large, aberrant right gastric vein draining into segment IV [14]. Couinaud described a venous network ascending along the arterial and biliary components of the main portal vein and supplied by vessels originating from the pyloroduodenopancreatic veins. This was frequently seen in the hilum of the liver, which was called the parabiliary venous system [15].
Yoshimitsu et al [16] mentioned that, from an embryologic standpoint, the development of bile ducts, the parabiliary venous system, the hepatic artery, and segment I and IV of the liver occurs later then the major portion of the liver and the portal venous system. This difference may account for why aberrant drainage of the parabiliary venous system occasionally occurs in segments I and IV.
Although pseudolesions in the posterior aspect of segment IV of the liver on CTAP [8,9] have been reported, the drainage vessel flow into the area had not been revealed. In 1994, Matsui et al first reported that the pyloroduodenopancreatic vein (so called “aberrant right gastric vein”) drains directly into the posterior aspect of segment IV and causes a portal perfusion defect in this area on CTAP [6] (Figure 4).

Matsui et al reported that the frequency of aberrant right gastric venous drainage into the posterior aspect of segment IV detected at imaging diagnosis is about 6%-14% [6,9]. Pseudolesions from aberrant right gastric venous drainage are observed not only in the

posterior aspect of segment IV, but also in liver segments II and III of the liver [17,18].

In most cases, pseudolesions in the drainage area of the parabiliary venous system exhibit hypodensity on CTAP. However, Yoshikawa et al [19] reported a case of high-density pseudolesion in the posterior aspect of segment IV on CTAP. This pseudolesion appeared as high density on CTAP because contrast media flowed into the pancreatic head via the dorsal pancreatic artery arising from the superior mesenteric artery. Dense contrast material from the head of the pancreas flowed directly into the liver parenchyma via the pyloroduodenopancreatic veins, so the posterior aspect of segment IV appeared to be higher density compared with the surrounding hepatic parenchyma. Although it is rare, pseudolesions of the liver in the third inflow area may appear hyperdense on CTAP.

2) Pseudolesion in the drainage area of epigastric-paraumbilical venous system

This venous system consists of small veins around the falciform ligament which drain the anterior part of the abdominal wall directly into the liver. This group is divided into the following groups: superior vein of Sappey, inferior vein of Sappey, and vein of Burow [20]. The superior vein of Sappey drains the upper portion of the falciform ligament and medial part of diaphragm. It enters the peripheral left portal vein branches and communicates with the superior epigastric and internal thoracic veins. The inferior vein of Sappey drains the lower portion of the falciform ligament. It enters the peripheral left portal vein branches and communicates with branches of the inferior epigastric vein around the umbilicus.
The vein of Burow terminates in the middle portion of the collapsed umbilical vein, and communicates with branches of the inferior epigastric vein around the umbilicus.
The inferior vein of Sappey and vein of Burow also have interconnecting veins.

Genchellac et al [21] described that hepatic pseudolesions around the falciform ligament are frequently encountered on portal-dominant phase MDCT images, and the prevalence of pseudolesions around the falciform ligament was 20% (Figure 5). The presence of an inferior vein of Sappey supplying these pseudolesions was found to be 27%. Fatty infiltration was found in 29%.
Ohashi et al [22] described that pseudolesions were seen on 64 (14%) of 472 helical CT scans, and 96% correlated with portal perfusion defects on CTAP. They were not more

enhanced than the surrounding liver parenchyma on CT arteriography with hepatic artery injection, but were enhanced in two patients on CT arteriography with internal thoracic artery injection.
The prevalence of pseudolesions around the falciform ligament (segment IV and/or III) is about 13-20% on biphasic contrast enhanced CT [23] and Gd-enhanced MRI [24].
Irie et al [25] compared the prevalence of pseudolesions in cirrhotic and non-cirrhotic livers on CTAP, and found that pseudolesions seen around the falciform ligament are significantly low in cirrhotic livers, but pseudolesions around the gallbladder and the posterior aspect of the segment IV showed no difference between the two groups. It suggests that in cirrhotic livers, since the parabiliary venous system (inferior vein of Sappey) acts as a collateral pathway for portal hypertension and shows hepatofugal flow, contrast material in the sinusoids around the falciform ligament are not diluted by "the third inflow."

3) Pseudolesion in the drainage area of cholecystic vein through the liver bed

Some parts of small cholecystic venous branches enter the liver directly through the liver bed (Segment IV and V) and drain the liver parenchyma around the body and fundus of the gallbladder, and communicate with peripheral intrahepatic portal branches. They dilute the portal perfusion at these sites, resulting in the appearance of pseudolesions [16].

In 1987, Matsui et al [7] reported that the increased cystic venous drainage to the intrahepatic portal vein causes staining in the non-diseased gallbladder bed on hepatic arteriography.
Yamashita et al [26] also reported that transient focal increased attenuation of the liver may occur on CT scans in patients with acute cholecystitis. This increased attenuation associated with acute cholecystitis has a typical location and pattern, and is probably attributed to hepatic arterial hyperemia and early venous drainage from the adjacent inflamed gallbladder. This finding should be differentiated from hypervascular hepatic tumors (Figure 6).
Ito et al [27] reported that, in gallbladder disease, the incidence of transiently increased attenuation around the gallbladder fossa and segments IV and V of the liver during arterial-phase helical or incremental CT is significantly increased compared to patients without gallbladder disease. Moreover, on hepatic angiography, 10 of the 22 patients showed early depiction of the dilated cystic vein (8 patients) and direct communication

with the portal branches (2 patients).
Such increased cystic venous drainage to the gallbladder bed causes a portal perfusion defect on CTAP.

Parenchymal changes caused by liver hemodynamic alteration (pseudotumor)

"Pseudotumor" is defined as a focal mass-like parenchymal change of the liver without timorous change, such as, focal spared area, focal fatty change and focal hyperplastic change in cirrhotic liver.

1) Focal spared area

Various types of focal areas of decreased fatty infiltration in fatty liver (focal spared area) are well visualized by sonography and CT. One of the most common sites is the posterior edge of segment IV, just anterior to the right side of the hepatic hilum [28,29] (Figure 7).
Matsui et al found a strong correlation between an aberrant right gastric venous drainage and the spared area at the posterior aspect of segment IV occasionally seen in fatty livers [30].
Focally decreased blood flow from the main portal vein, associated with aberrant right gastric venous drainage, is a likely cause of the focal spared area.

2) Focal fatty change

Fatty infiltration of the liver is a well-recognized entity in both diffuse and focal forms. In the focal form, various types of fatty infiltration have been described, and the anteromedial portion of segment IV, adjacent to the falciform ligament, is one of the most commonly involved locations [31].
The posterior aspect of segment IV is commonly spared from diffuse fatty infiltration, and is rarely involved with focal fatty infiltration (Figure 8).
Kawamori et al [32] and Fukukura et al [33] reported that focal fatty infiltration at the posterior aspect of segment IV is also related to the presence of aberrant right gastric venous drainage.
However, the reason why similar variations in blood supply of the posterior aspect of segment IV cause focal fatty infiltration in some patients, and focal spared area in others is unknown. Kawamori et al [32] suspected that differences in the ways various hormones, nutritional elements, and other factors act on the aberrant right gastric

venous drainage and the main portal vein may influence metabolism or nutrition in the posterior edge of segment IV. These variations may lead to different results among individual cases.

3) Hyperplastic change in cirrhosis

Matsui et al [34] reported that aberrant gastric venous drainage in cirrhotic livers sometimes appears as hypoechoic on ultrasound, hypoattenuated on enhanced CT, and hyperintense on T1-weighted or hypointense on T2-weighted MR images. On dynamic CT and dynamic MRI, these areas showed early enhancement, which is thought to be caused by early venous return compared with that of the surrounding liver parenchyma (Figure 9). Histopathologically, these lesions showed hyperplastic changes.

The imaging findings, with the exception of early enhancement, were very similar to those of dysplastic nodule or well-differentiated hepatocellular carcinoma.

Similar hyperplastic changes are occasionally seen around the gallbladder fossa, which is the drainage area of cholecystic veins.

It is not known why hyperplastic change relative to the surrounding regenerative nodules was seen at the focal areas with aberrant right gastric venous drainage and cystic venous drainage area.

Matsui et al [34] suspected that these changes are probably due to differences in various kinds of hormones, nutritional elements, factors such as hepatotrophic factors, among others, between the blood from the main portal vein and that from the gastric or cystic vein.

Gabata et al [35] reported a giant hyperplastic change of the caudate lobe in a patient with liver cirrhosis, and they suspected that caudate hyperplastic change may be correlated to an anomalous caudate portal vein branch. They speculated that according to the laminar flow, the caudate branch receives less blood flow from the superior mesenteric vein than from another segment of the liver, and more blood flow from the splenic and gastric veins. The differences in hormones, nutritional elements, and hepatotrophic factors in the portal blood flow between the caudate lobe and the segment of the liver may correlate with the hyperplastic change of the caudate lobe. Insulin and glucagon have been proposed as hepatotrophic hormones, which would be present in the splenic vein [36].

Since the flow direction of the inferior vein of Sappey is hepatofugal in a cirrhotic liver, focal hyperplastic changes are not observed in the anteromedial portion of segment IV around the falciform ligament.

Hepatobiliary phase images of Gd-EOB-DTPA enhanced T1-weighted MRI is useful tool for detection of hepatic tumors. In focal fatty change and focal spared lesion cases, the lesion usually do not show attenuation difference in hepatobiliary phase of Gd-EOB-DTPA enhanced MRI compared to surrounding hepatic parenchyma, and we can easily diagnose them as pseudolesions. However, on focal hyperplastic change in cirrhotic liver, "hyperplastic" area sometimes show increase uptake of Gd-EOB-DTPA similar to other cause of hyperplasia on hepatobiliary phase image and it may confuse our diagnosis.

Imaging confirmation of the third inflow

Usually, diagnosing pseudolesion and pseudotumor in the third inflow area is not difficult if attention is paid to the typical occurrence site of the liver (such as the posterior aspect of segment IV, the anteromedial portion of segment IV around falciform ligament, and gallbladder fossa) and typical findings such as focal fatty infiltration or focal sparing of the fatty liver. But in some cases, especially in cirrhotic livers, focal hyperplastic change in the third inflow area sometimes shows findings similar to those of hypervascular HCC or hypovascular well-differentiated HCC in some modalities [34]. In such cases, the presence of third inflow to the hepatic area should be confirmed for correct diagnosis.

In some cases, it may be possible to obtain direct confirmation of aberrant right gastric venous drainage to the posterior aspect of segment IV, or cystic venous drainage to the gallbladder fossa in venous phase images of celiac, right gastric arterial, or cystic arterial angiography. For confirmation of drainage of the inferior vein of Sappey, it seems difficult to opacify the venous drainage of inferior vein of Sappey on venous phase images of arteriography.

Since it is easy to recognize the drainage area in the liver on CT obtained during selective arteriography, in order to obtain imaging confirmation of third inflow to the liver, CT during selective right gastric artery [34, 37], CT during internal thoracic artery [38], and CT during cystic arteriography [39] are performed. However, angiography and CT during selective angiography are invasive and are not performed routinely.

Recent advances in multidetector-low CT enable the demonstration of hepatic collateral vessels clearly using CT-angiographic techniques [40], so we can depict third inflow vessels draining into the specific liver area, such as the posterior aspect of segment IV, the anteromedial portion of segment IV around falciform ligament, and gallbladder

fossa with CT-angiographic, MPR, or 3D reconstruction techniques. In addition, the presence of third inflow to the liver can be confirmed using color- and power-Doppler ultrasound technique [41-43] (Figure 10).

Future perspective

Pseudolesions and pseudotumors in third inflow area are important not only in differential diagnosis of HCC but also consideration of the cause of benign nodular hepatocellular lesions.

There are many kinds of benign regenerative and / or hyperplastic lesions, such as focal nodular hyperplasia (FNH), nodular regenerative hyperplasia (NRH) and focal hyperplastic changes in cirrhotic liver observed in third inflow area.

Precise etiology of such benign regenerative and / or hyperplastic lesions is unknown. However, recently, from the histopathological points of view, Kondo [44] introduced a hypothesis that congenital vascular anomaly is the origin of some kinds of the benign hepatocellular hyperplastic nodules and hypothesized that change in the blood supply can cause hyperplastic nodule.

Physiologically, vascular anomaly in focal hepatic area might cause imbalance of portal venous and hepatic arterial flow between the hepatic parenchyma within vascular anomaly area and surrounding normal vascular area, and result in the differences in various kinds of hormones, nutritional elements, factors such as hepatotrophic factors perfuse in these area.

This is the same situation with pseudotumor in third inflow area. So elucidation of the etiology of hepatic parenchymal change observed in third inflow area might have potential clue to resolve the etiology of some kinds of benign regenerative and / or hyperplastic lesions such as FNH and NRH.

Conclusion

We reviewed hepatic pseudolesions and pseudotumors of the third inflow area on imaging. It should be noted that some pseudotumors in cirrhotic livers may have findings similar to those of hepatocellular carcinoma or dysplastic nodules and confuse diagnosis. Knowledge of pseudolesions and pseudotumors in the third inflow area can aid in correct diagnosis and avoid unnecessary treatment.

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Figure legends

Figure 1. Angiography assisted CT of hepatocellular carcinoma
A, On CT during arterial portography (CTAP), hepatocellular carcinoma shows portal perfusion defect (arrow). B, On early phase image of CT during hepatic arteriography (CTHA), hepatocellular carcinoma shows well attenuation compared to background liver parenchyma (arrow). C, On late phase image of CTHA, peritumoral hepatic parenchyma around the hepatocellular carcinoma shows corona like enhancement (arrowheads) which represent drainage flow from the tumor.

Figure 2. An example of pseudolesion in angiography assisted CT

A, On CT during arterial portography (CTAP), wedge-shaped portal perfusion defect is observed in the medial edge of right posterior segment (arrow). B, On CT during hepatic arteriography (CTHA), medial edge of right posterior segment show well attenuated both on early phase (B, arrow) and late phase image (C, arrow) and did not show corona like staining in late phase image. On, T1- (D), and T2- (E) weighted MR images, the area shows no intensity difference compared to surrounding hepatic parenchyma. These findings indicate typical early enhancing pseudolesion.

Figure 3. Schematic diagram of common sites of hepatic pseudolesions in third inflow area.

Figure 4. Pseudolesion in the drainage area of parabiliary venous system
A, On CTAP, liver parenchyma in posterior aspect of segment IV shows portal perfusion defect. B, On venous phase image of right gastric arteriography, venous flow from stomach directly drain into hepatic hilum (arrow); which represents so called aberrant gastric venous drainage. C, On CT during right gastric arteriography, we can confirm that liver parenchyma in posterior aspect of segment IV, where showed portal perfusion defect on CTAP, is well opacified by gastric venous flow not connected to the main portal trunk (arrow).

Figure 5. Pseudolesion in the drainage area of inferior vein of Sappey
On multiphasic contrast enhanced CT, liver parenchyma in anteromedial portion of

segment IV adjacent to the falciform ligament shows hypodense on portal phase image (B, arrow) and equilibrium phase image (C, arrow). however, on pre-contrast enhanced CT, the area shows no attenuation difference compared to the surrounding hepatic parenchyma (A; pre-contrast CT, B; portal phase, C; equilibrium phase contrast enhanced CT).

Figure 6. Pseudolesion in the drainage area of cholecystic vein
On multiphasic contrast enhanced CT, liver parenchyma in segment IV adjacent to the gallbladder shows early enhancement on arterial phase image (B, arrow), however, on pre-contrast and equilibrium phase contrast enhanced CT, the area shows no attenuation difference compared to the surrounding hepatic parenchyma (A; pre-contrast CT, B; arterial phase, C; equilibrium phase contrast enhanced CT).
Coronal re-construction of arterial phase enhanced CT shows inflow of cystic vein into the hepatic parenchyma in segment IV adjacent to the gallbladder (D, arrow).

Figure 7. Focal spared area of fatty liver in third inflow area
A, On US, posterior aspect of the segment IV of the liver shows hypo-echo compared to surrounding liver parenchyma. B, On pre-contrast enhanced CT, posterior aspect of the segment IV of the liver shows hyperdense compared to surrounding liver parenchyma. On T1 weighted MRI, Liver parenchyma, including posterior aspect of the segment IV, shows uniform intensity on in-phase image (C), and liver parenchyma except the posterior aspect of the segment IV shows hypointense on opposed-phase image (D, arrow). These images indicate focal sparing of fatty liver in posterior aspect of the segment IV. Venous flow from stomach directly drain into the posterior aspect of segment IV of the liver on coronal re-construction of portal phase enhanced CT (E).

Figure 8. Focal fatty change in third inflow area
Liver parenchyma in posterior aspect of segment IV shows hyperintense on in-phase image (A, arrow) and hypointense on opposed-phase image (B, arrow) of T1 weighted MRI, which indicate focal fat deposit in the area. On coronal re-construction portal phase image of Gd-enhanced T1 weighted MRI, venous flow from pyloric area directly drain into the posterior aspect of segment IV of the liver (C, arrow; so called " aberrant gastric venous drainage") .

Figure 9. Hyperplastic change in cystic venous inflow area in cirrhotic liver
Hepatic parenchyma in segment IV adjacent to the gallbladder shows hyperintensity On T1 weighted MR image (A, arrow) and hypointensity on T2 weighted MR image (B, arrow). On hepatobiliary phase of Gd-EOB-DTPA enhanced T1 MR image, the lesion shows more hyperintensity compared to background liver parenchyma (C, arrow).
On CTAP, the lesion shows portal perfusion defect (D, arrow). Coronal re-construction of early phase CTHA image shows inflow of cystic vein into the hepatic parenchyma in segment IV adjacent to the gallbladder (E, arrow).

Figure 10. Ultrasonographic confirmation of the third inflow in the drainage area of inferior vein of Sappey
Because of SVC obstruction caused by large mediastinal tumor (not shown), inferior vein of Sappey show hepatipetal flow as collateral pathway of obstracted SVC.
On multiphasic contrast enhanced CT, liver parenchyma in anteromedial portion of segment IV adjacent to the falciform ligament shows contrast enhancement on arterial phase (B, arrow) and slightly hypodense on equilibrium phase image (C, arrow). On pre-contrast enhanced CT, the area shows no attenuation difference compared to the surrounding hepatic parenchyma (A; pre-contrast CT, B; portal phase, C; equilibrium phase contrast enhanced CT). Hepatopetal venous flow toward the liver parenchyma in anteromedial portion of segment IV adjacent to the falciform ligament is confirmed by color Doppler ultrasonography (D, arrow).

Executive Summary

Definition of pseudolesion and pseudotumor

Pseudolesion is defined as focal mass-like findings visualized only on imaging without parenchymal change.

Pseudotumor is defined as focal mass-like parenchymal change of the liver with preserved internal hepatocytes, angioarchitecture and function of the normal liver.

Third inflow to the liver

Aberrant veins supplying small areas of liver tissue and communicating with intrahepatic portal vein branches.

Classified into the following groups;

Cholecystic vein (Drain into gallbladder bed)

Parabiliary venous system (Mainly drain into posterior aspect of segment IV)

Epigastric-paraumbilical venous system (Mainly drain into anteromedial portion of the segment IV)

Pseudotumor in third inflow area

Focal spared area of fatty liver

Focal fatty liver

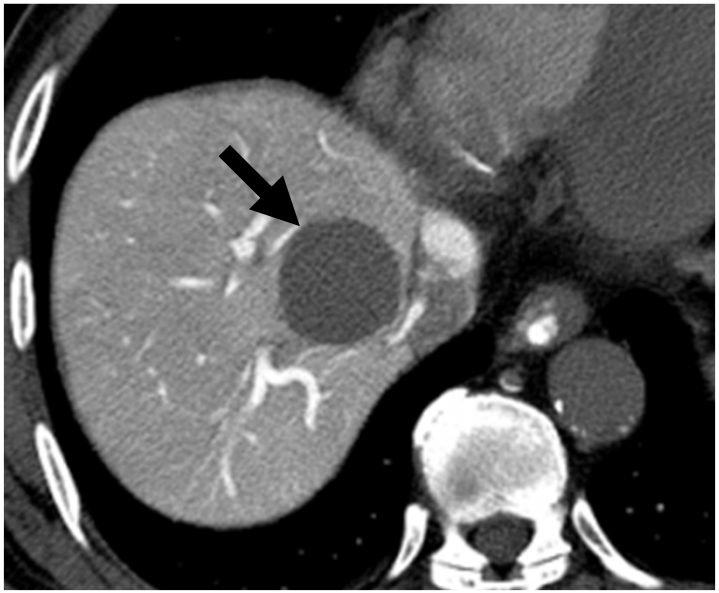
Focal hyperplastic change in cirrhotic liver

Focal hyperplastic change in third inflow area sometimes show similar imaging findings with well-differentiated hepatocellular carcinoma.

Imaging confirmation of the presence of third inflow is vital for correct diagnosis of focal hyperplastic change in third inflow area.

Fig1

A



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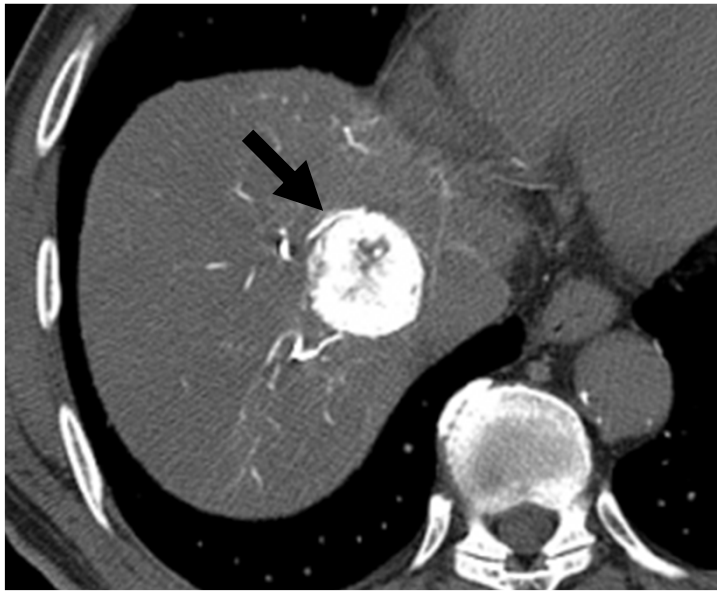

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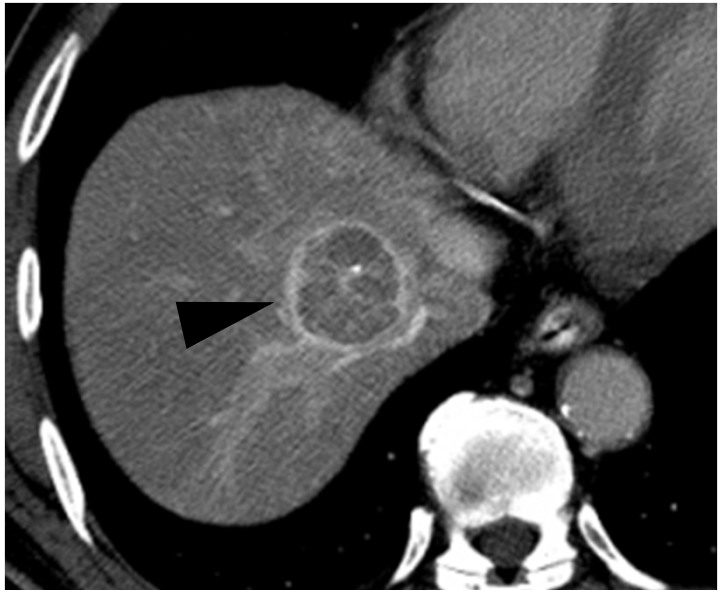


Fig2

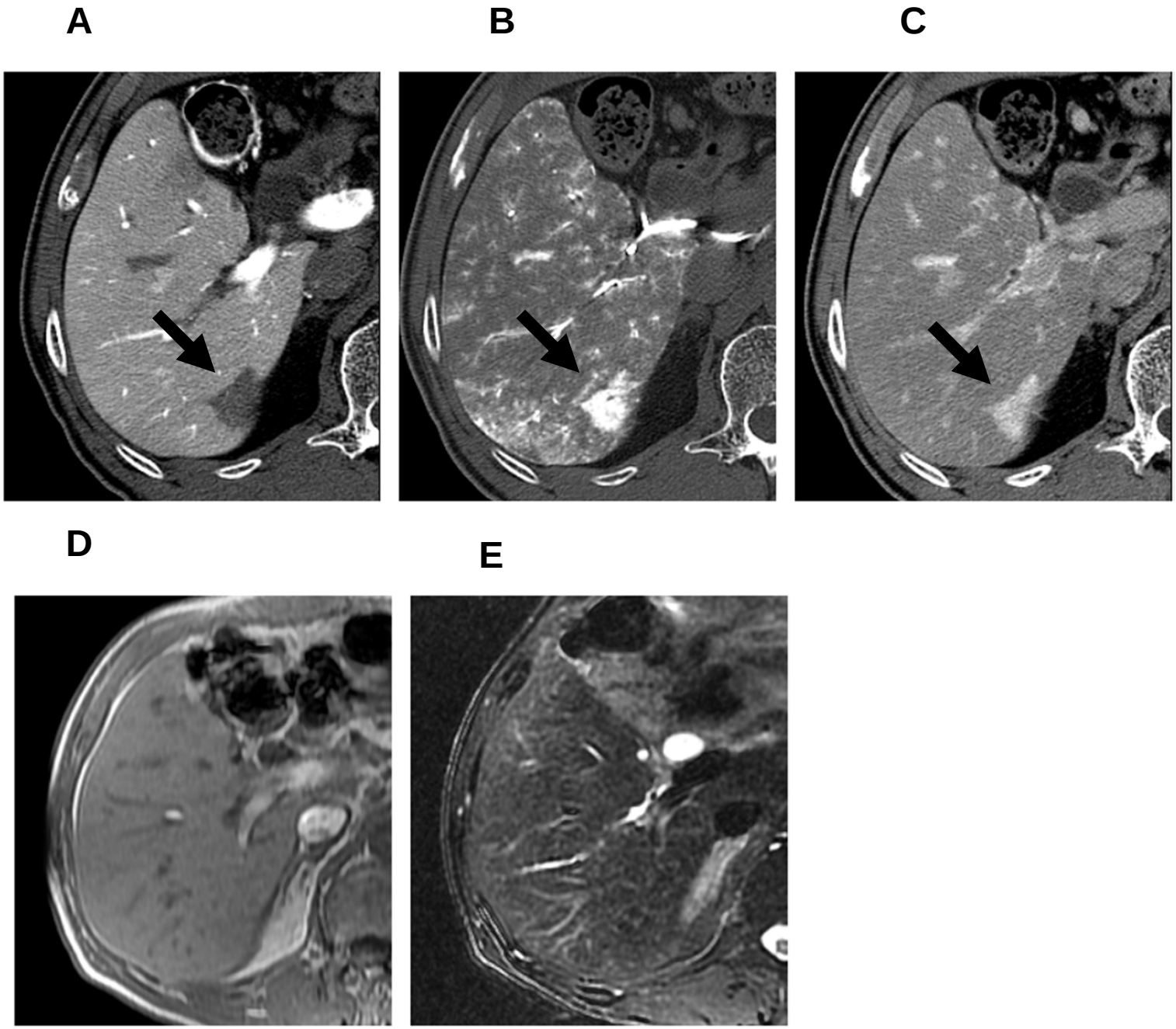


Fig.3

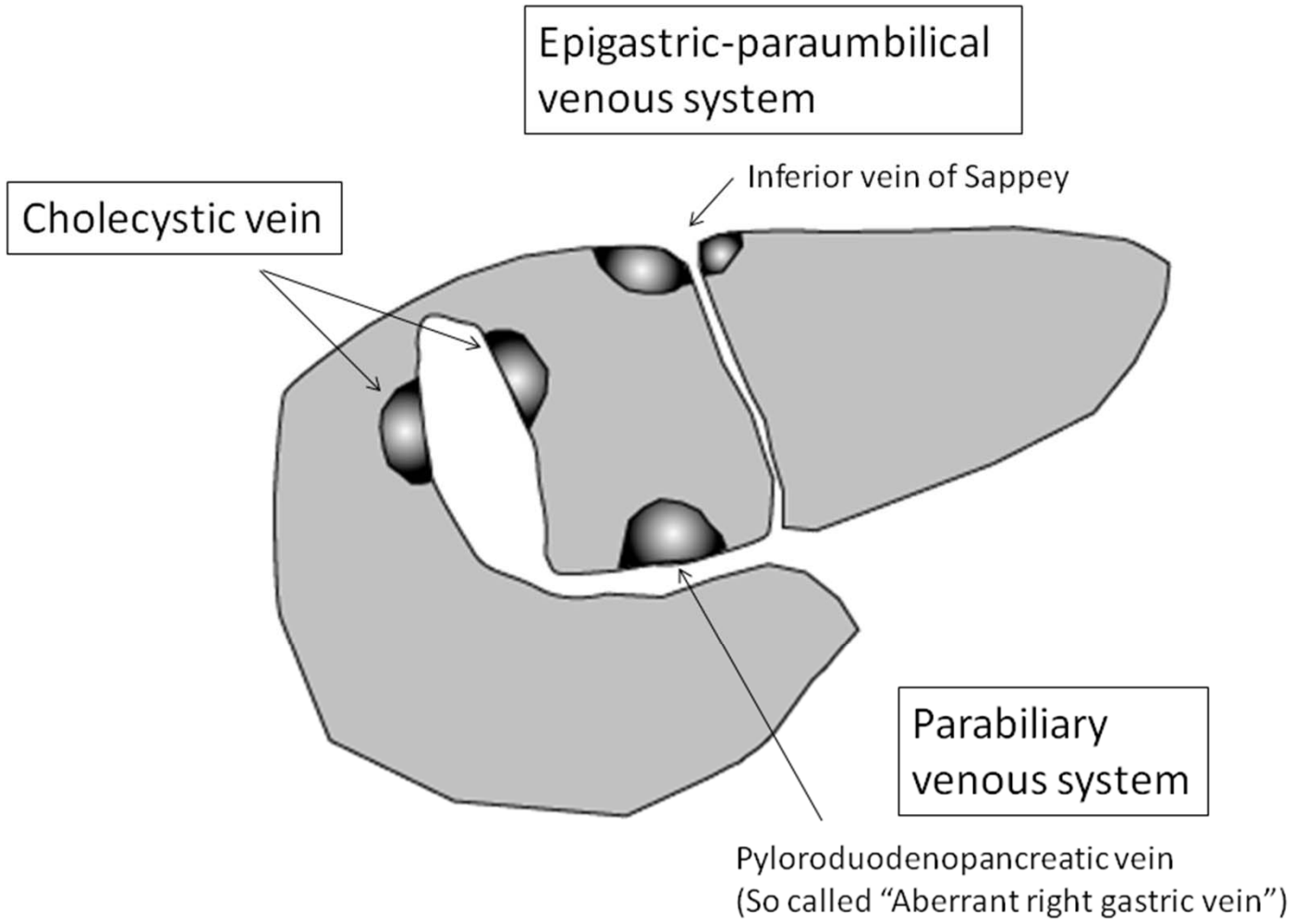
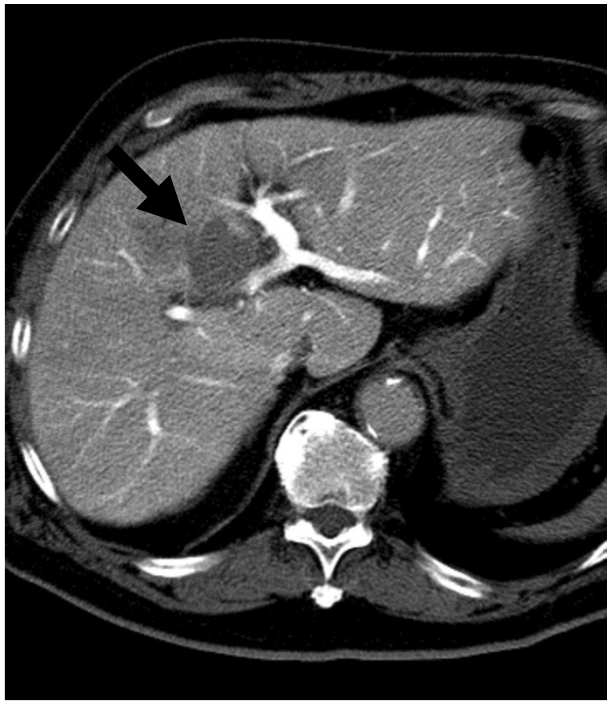

Epigastric-paraumbilical venous system
Inferior vein of Sappey
Cholecystic vein
Parabiliary venous system
Pyloroduodenopancreatic vein
(So called "Aberrant right gastric vein")

Fig4

A



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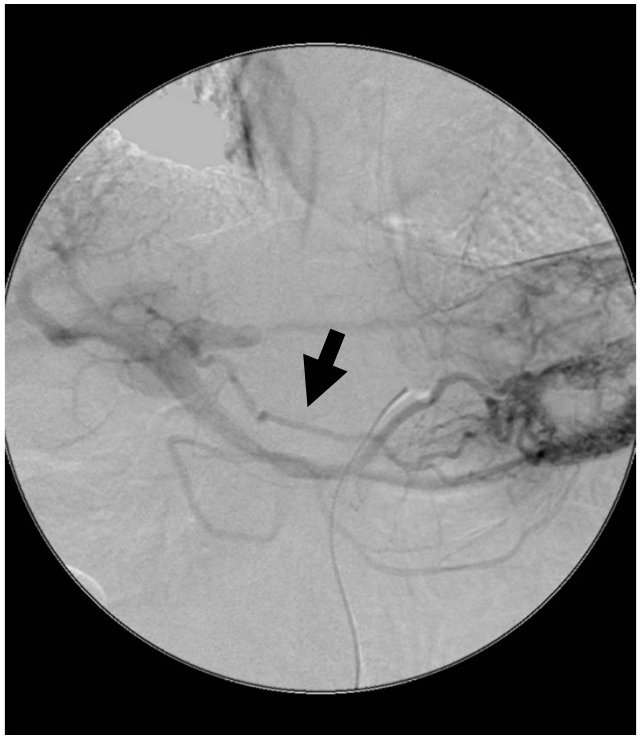


C

Fig.5

A

B

C

Fig.6

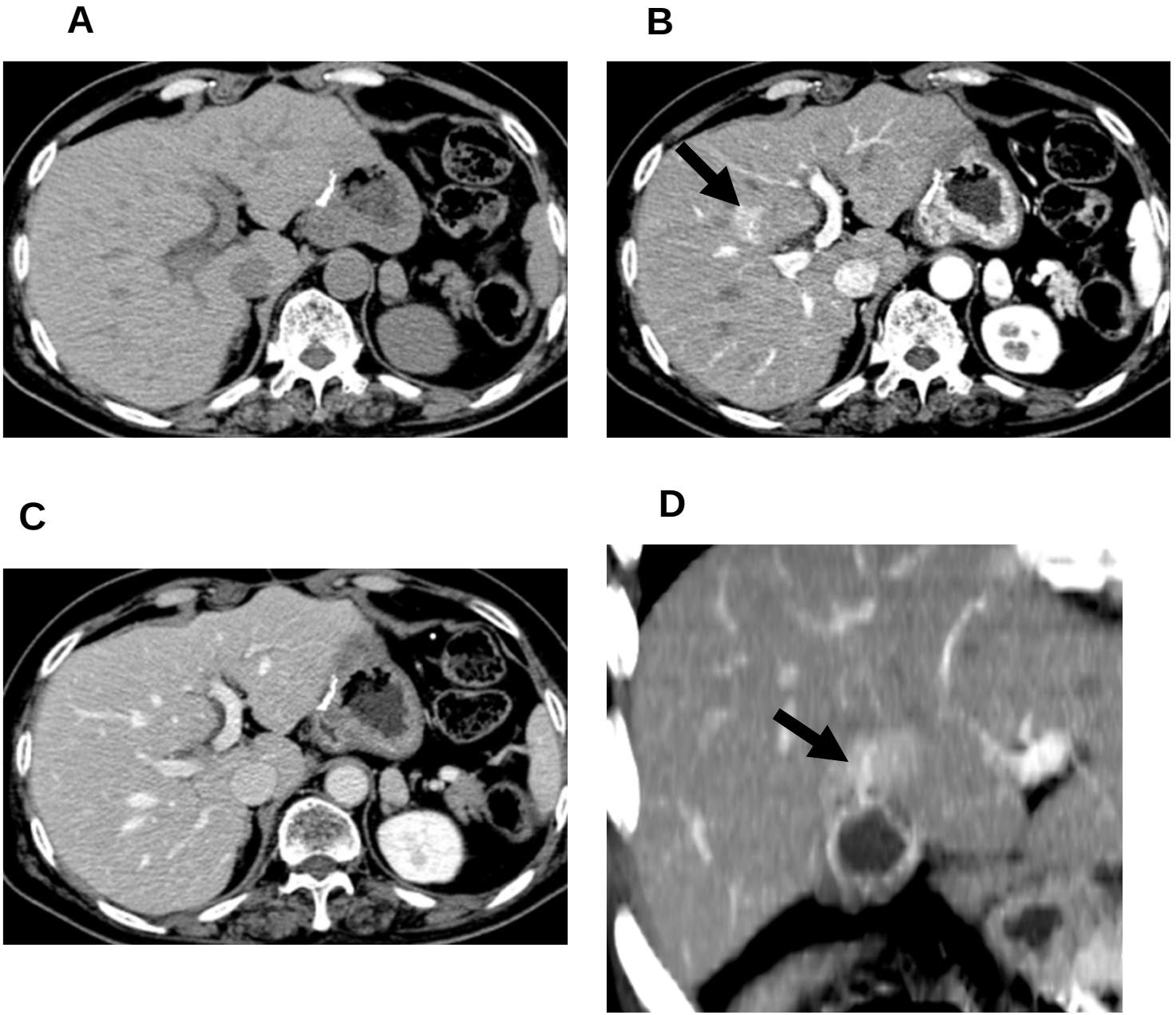
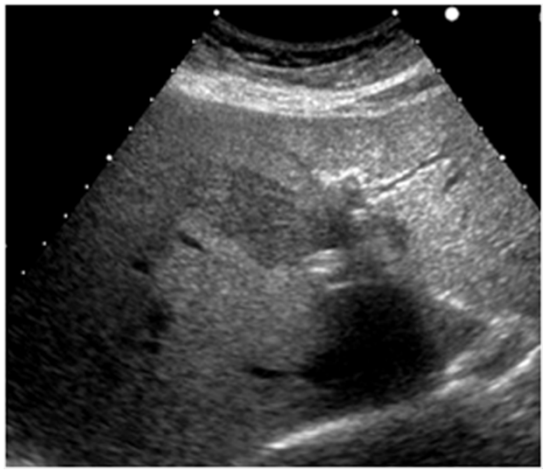


Fig 7

A



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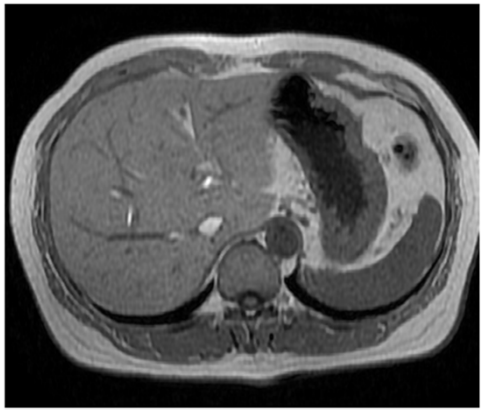
C



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E

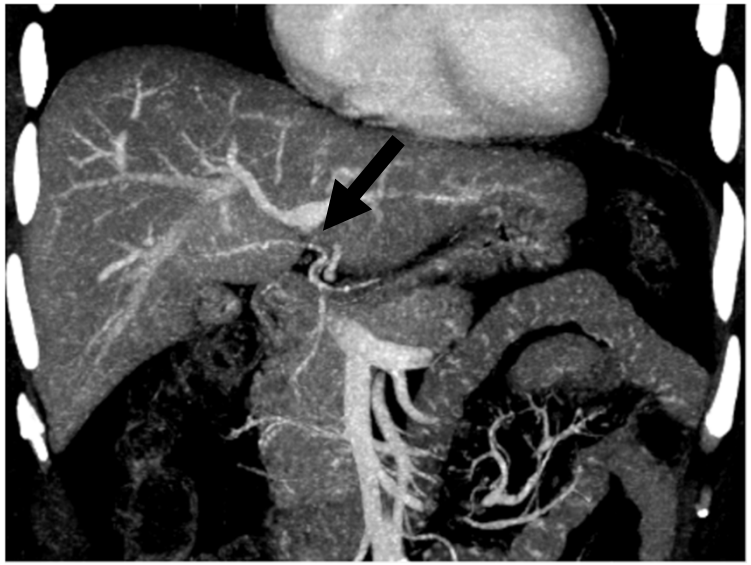

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Fig.8

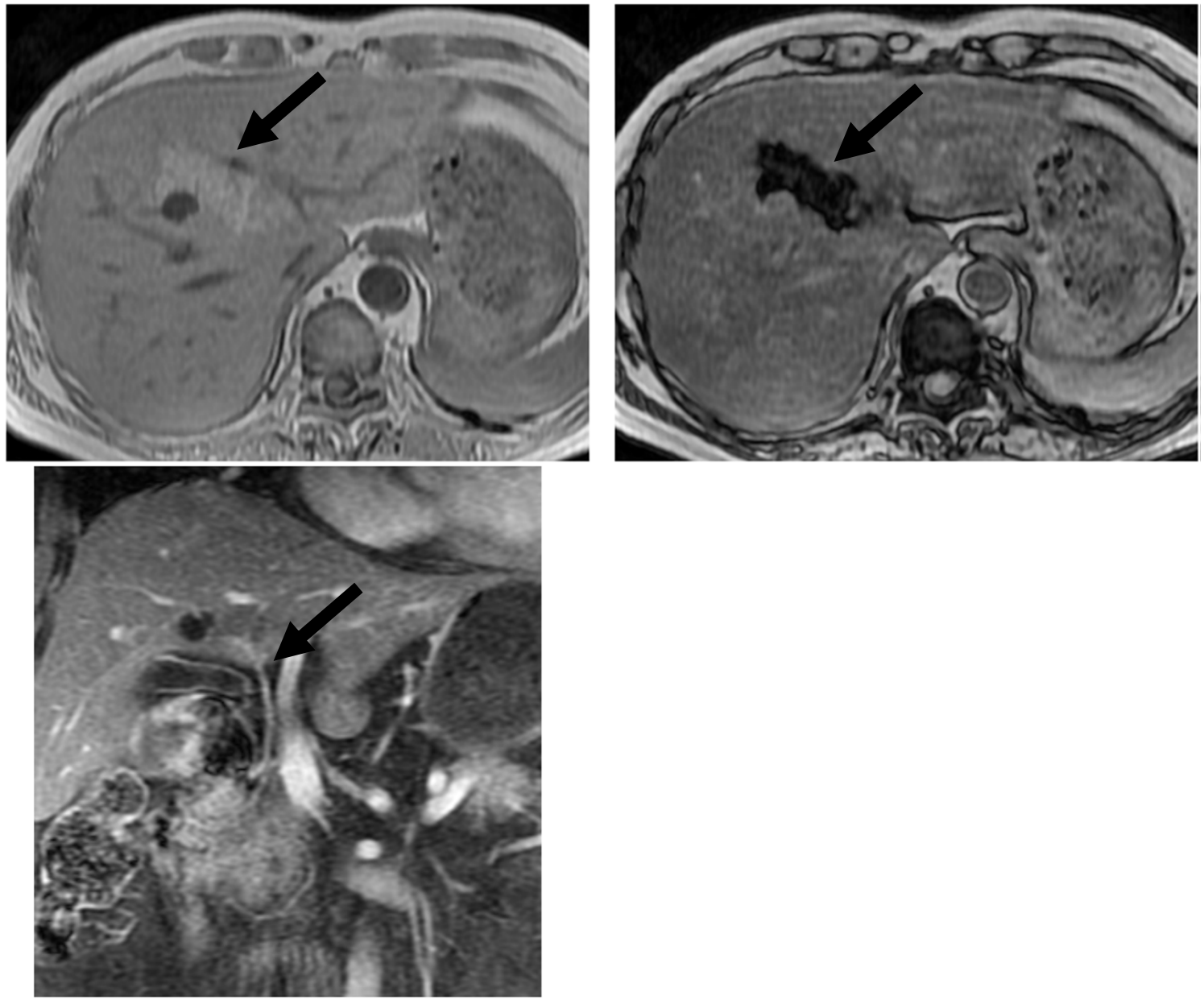

Fig.9

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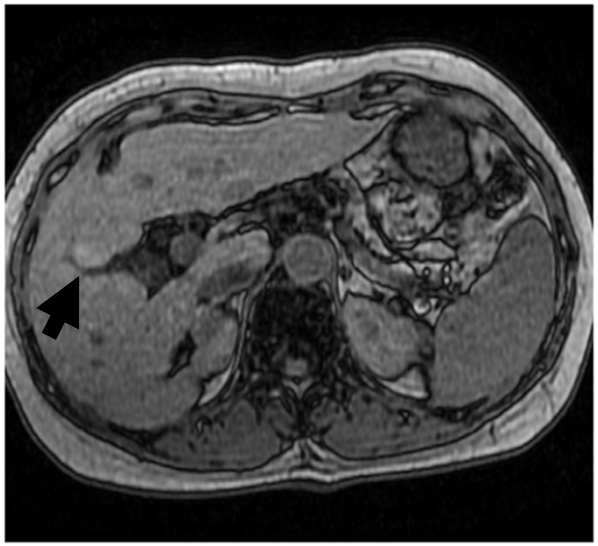

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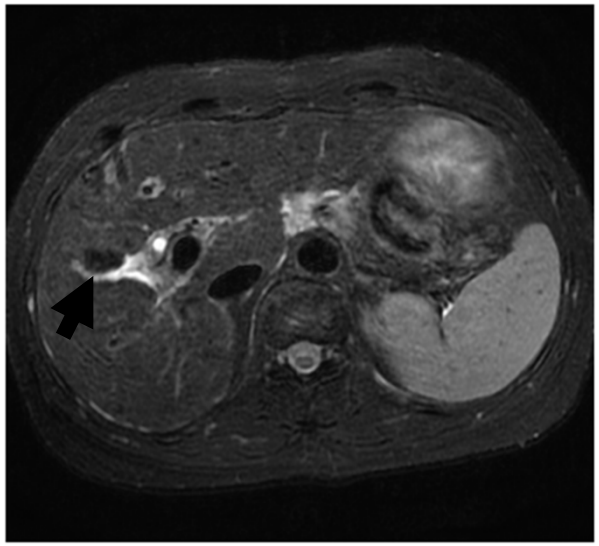

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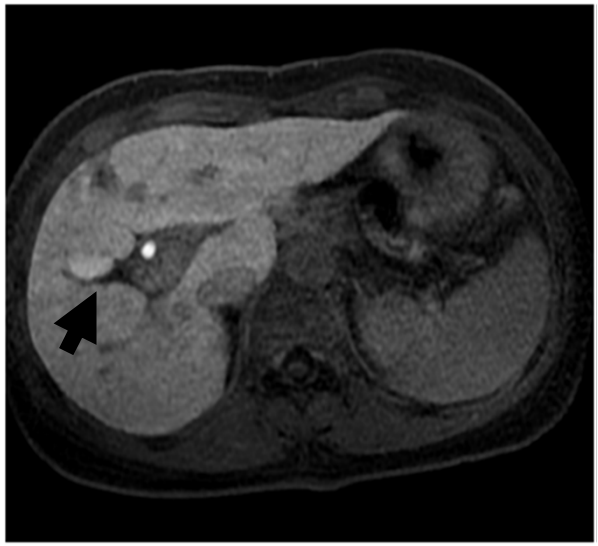

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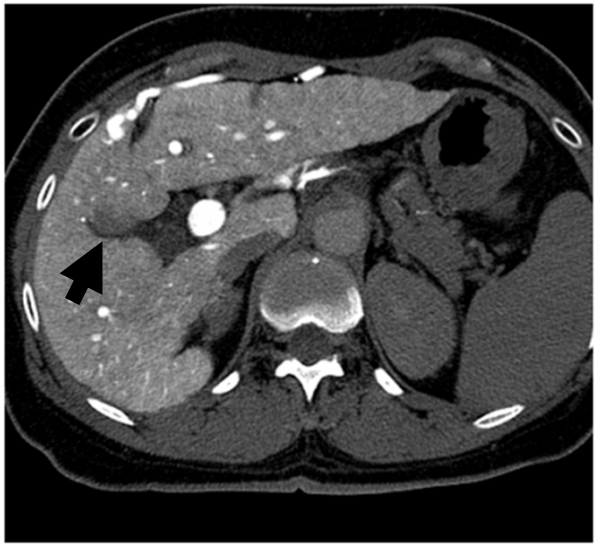

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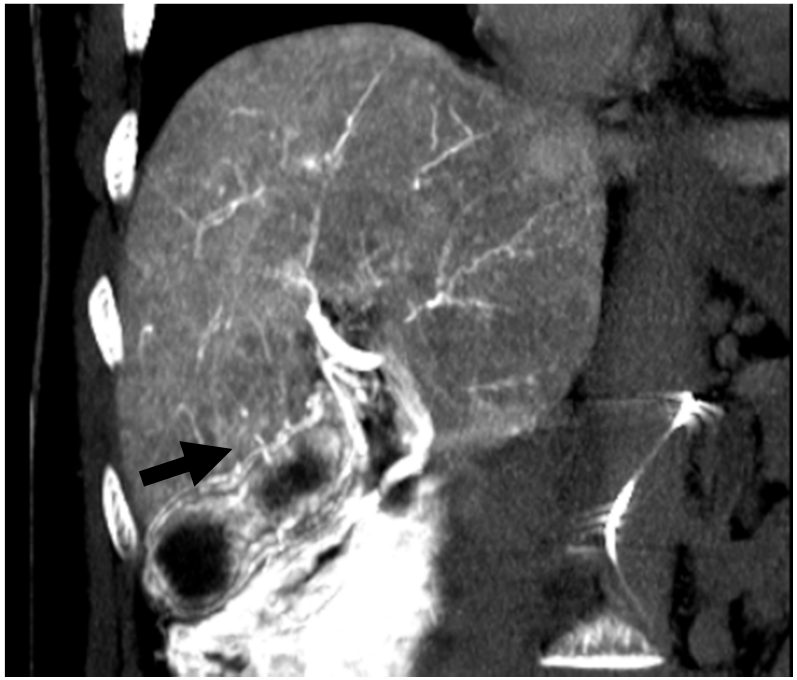
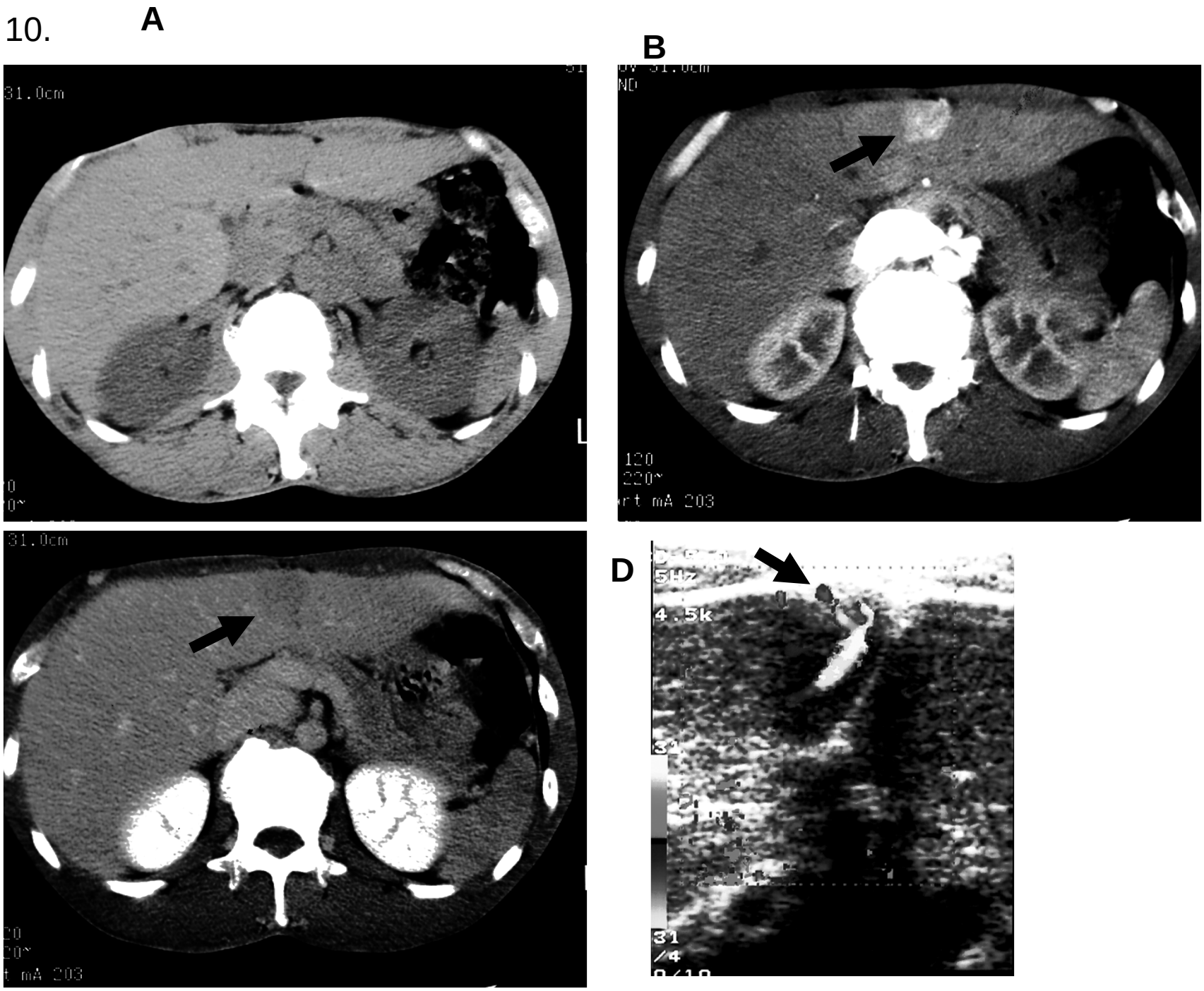
Fig 10.