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ORIGINAL ARTICLE

The significance of the right hepatic artery originating from the superior mesenteric artery in patients with cavernous transformation of the portal vein

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PURPOSE
Dilatation of the hepatic artery in response to decrease in portal vein flow is known as hepatic arterial buffer response (HABR). In this study, the effect of HABR on variant hepatic arterial anatomy is investigated by analyzing the frequency of the right hepatic artery originating from the superior mesenteric artery (variant artery) and by determining the diameters of variant artery and common hepatic artery (CHA) in patients with cavernous transformation of the portal vein.

MATERIALS AND METHODS
Forty-one patients who were referred for contrast-enhanced abdominal magnetic resonance angiography were retrospectively evaluated in two groups: group 1 (n = 15), cirrhotic patients with cavernous transformation of the portal vein; and group 2 (n = 26), cirrhotic patients without cavernous transformation of the portal vein.

RESULTS
The frequency of the variant artery was significantly higher (53%) in patients with cavernous transformation of the portal vein ($P < 0.01$) than those without cavernous transformation (11.5%). The mean diameters of the CHA and the variant artery in 2 groups were not significantly different.

CONCLUSION
Vasodilatation at the level of intrahepatic arterioles (HABR) in response to diminished portal flow may be a factor that increases the frequency of the variant hepatic artery.

Key words: • *thrombosis, portal vein* • *hepatic artery* • *variations, anatomic*

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Experimental studies show that the hepatic artery dilates in response to reduction of portal venous perfusion (1). This observation is explained by Lautt as an intrinsic regulatory mechanism called the hepatic arterial buffer response (HABR), which maintains total hepatic blood flow at a constant level (1). The mechanism of the HABR is based on adenosine washout. Adenosine, which dilates the hepatic artery, is produced at a constant rate and secreted into a fluid compartment around the hepatic arterial system. If the flow in portal vein decreases, less adenosine is washed away into the portal venous blood, and its accumulation causes hepatic arterial dilation (1). Attempts have been made to confirm the observations of Lautt in patients with portal vein thrombosis, using Doppler ultrasonography (2–4). Controversy remains, however, regarding hepatic artery resistance indices in portal vein thrombosis. This controversy is partially explained by the background conditions of the liver (presence or absence of cirrhosis).

Dilatation of the main hepatic artery is not a direct effect of the portal vein thrombosis. Vasodilatation occurs first at the level of the arterioles (precapillaries); the caliber of the main hepatic artery increases indirectly as a consequence of increased blood flow. Because the hepatic artery takes over the entire hepatic blood supply in portal vein thrombosis, the demonstration of high-frequency arterial Doppler signals within the liver may be considered an indirect sign of portal thrombosis (5). It is also known from dynamic computed tomography (CT) examinations that venous compromise (portal or hepatic veins) results in increased arterial flow, which has been shown to be a transient alteration of hepatic perfusion (6). To the best of our knowledge, there is one published report on magnetic resonance (MR) angiography confirming the pathophysiological changes in HABR (7).

During multiphasic MR angiographic studies of the abdomen in our institution, we were able to predict the presence of the portal vein thrombosis from the first phase of the examination by observing the prominent hepatic arterial dilatation. We also realized that a variant hepatic artery arising from superior mesenteric artery was frequent in cases of portal vein thrombosis in which the common hepatic artery was not significantly dilated. To test the validity of our observations, we aimed to determine the frequency of this specific variant hepatic artery in cirrhotic patients with portal vein thrombosis.

Patients and methods

Patient population

The patient selection for this retrospective study was based on the availability of contrast-enhanced MR portographic data on compact

discs recorded between June 2000 and June 2005. Duplicate data on individual patients (n = 5) and data from patients with hepatic venous outflow obstruction (n = 81) were not reviewed. In addition, the following cases identified during the retrospective MR angiographic image analysis were not included in the study: isolated intrahepatic portal vein branch thrombosis (n = 3), occlusion of the celiac trunk associated with dilation of the pancreaticoduodenal arcade (n = 1), hepatic alveolar *Echinococcus* invading hepatic veins (n = 1), metastatic liposarcoma in the liver (n = 1), hepatic surgery (n = 3), cholangiocellular carcinoma (n = 1), pancreatic carcinoma (n = 6), and duodenal carcinoma (n = 1). An additional 5 patients with hepatocellular carcinoma were excluded since it was shown that hepatic arterial blood flow may increase in such cases (4). Patients without liver cirrhosis as an underlying cause of the cavernous transformation of the portal vein (n = 20; prothrombotic disorders, pancreatitis, pancreatic carcinoma, hepatic metastasis) and patients with idiopathic (non-cirrhotic) portal hypertension were omitted from the study.

A total of 41 patients with advanced cirrhosis and hypertension were included in the study. The diagnosis of cirrhosis was made by histopathologic examination. The diagnosis of portal hypertension was made according to the presence of splenomegaly, ascites, or portosystemic collateral vessels on ultrasonographic and MR angiographic examinations, and/or presence of esophageal and fundal varices on upper gastrointestinal endoscopy. The patients were divided into two groups according to the presence or absence of cavernous transformation of the portal vein.

Group 1: Cirrhotic patients with cavernous transformation of the portal vein

This group included 15 patients (9 men and 6 women; age range, 27–56 years; mean age, 43 years). The cause of the portal vein thrombosis was viral hepatitis-induced chronic liver disease in 13 patients; the cause was unknown in the other two patients. All patients had cavernous transformation of the portal vein due to portal vein thrombosis.

Group 2: Cirrhotic patients without portal vein thrombosis and cavernous transformation

This group included 26 patients (17 men and 9 women; age range, 19–75 years; mean age, 45 years). The etiologies of underlying cirrhosis were viral hepatitis (n = 15), alcohol-induced liver disease (n = 1), and primary biliary cirrhosis (n = 1). In 9 patients, the cause of cirrhosis was obscure.

MR angiography technique

At our institution, quadriphasic MR angiography is the standard examination for the evaluation of the hepatic vasculature and portal venous system. The examination was performed with a 1.0 Tesla system (Signal LX Horizon; General Electric Medical Systems, Milwaukee, USA) equipped with a four-element torso coil.

Just before data acquisition, approximately 0.2 mmol per kilogram of body weight of gadolinium chelate was injected through a 20-gauge intravenous cannula in the antecubital vein. Following a determined delay time (estimated on the basis of age and cardiac status of the patient; range, 13 to 17 seconds), data acquisition of the MR angiography study was started.

Contrast-enhanced MR angiography was performed using fast spoiled gradient echo recalled sequence. Data were acquired in a coronal plane during breath-hold. TR/TE/FA (repetition time/echo time/flip angle), and bandwidth were 4–6 ms, 1.2 ms, 20 degrees, and 31.2 or 62.5 kHz, respectively. Image matrix was 256 × 160 or 256 × 128. Field of view (FOV) varied from 40 to 58 cm. Section thickness was 3.0 to 4.0 mm. Acquisition time ranged between 12–24 seconds (mean, 16 seconds). If the predicted acquisition time was beyond a reasonable breath-hold duration, phase encoding steps were decreased (to 128), or phase FOV was reduced (to 0.70–0.60). Data acquisition was repeated four times consecutively to obtain dynamic information at early arterial and late portal venous and hepatic venous phases. One total arterial phase and three venous phase data sets were acquired in approximately 2 minutes. Postcontrast fat-suppressed fast spoiled gradient echo images (TR, 120 ms; TE, 6.3 ms; FA, 90 degrees; BW, 20.83; FOV, 32–40; slice thickness, 7 mm; spacing, 1.5 mm; matrix, 256 × 160; NEX [number of excitations], 1) of the upper abdomen were also obtained in the axial plane as a complementary sequence to overview of the portal venous system and its tributaries.

Image analysis

All MR angiographic studies retrieved from the compact discs were retrospectively analyzed at a workstation (Advantage Windows, version 3.1, GE Healthcare) by the same experienced radiologist. Source images of the arterial and portal venous phases were reconstructed using a target maximum intensity projection (MIP) technique to depict the hepatic arterial and portal venous anatomy. When reconstructing the MR angiograms, four images (target MIP, reformatted axial, reformatted sagittal, and coronal source images) were simultaneously displayed on the screen of the workstation. The origin and the course of the vessels could be assessed more readily using this configuration of the system.

The diameter measurements were repeated at least three times for each vessel by using electronic calipers and magnification tool. The average values were recorded on a standard form and used as statistical data. The diameter of the common hepatic artery (with classical and variant anatomy) was measured approximately 1.5 cm distal to its origin at the celiac trunk. The maximum diameter of the superior mesenteric artery was measured 0.5–0.8 cm from its origin dorsal to the pancreas. The variant hepatic artery arising from the superior mesenteric artery, if present, was followed from its origin to the portal hilum. Its diameter was measured at its proximal third. The accessory (Michels type III) and the replaced (Michels type VI) types of this variation (8) were not determined because the identification of intrahepatic distribution of small arterial branches was not possible in each case. For the purpose of our study, both types of variation were assumed to represent the variant hepatic artery in a fashion similar to the Hiatt classification (grouped as type 3) (9).

The diameter of the main portal vein was measured at 1.5–2 cm distal to the portal hilum. The vessel was assumed to be normal when it was patent with an average caliber (upper limit, 13 mm) (5). Nonvisualization and nonenhancement of the main portal vein were considered to be signs of portal vein thrombosis. Depiction of a hy-

pointense, thrombotic material filling the whole lumen was also considered to be consistent with occlusion. A network of numerous enhancing serpiginous vascular channels at the expected region of the portal vein was considered to be cavernous transformation.

Statistical analysis

The homogeneity of sex distribution of the groups was analyzed with the chi-square test. All results of the measurements were expressed as mean ± standard deviation (SD). The differences between the mean values of hepatic artery, superior mesenteric artery, and variant hepatic artery diameters in the two groups were compared using the t-test. The frequency of the variant artery between 2 groups was assessed with the Fisher exact test. A parametric t-test was used to compare the mean diameters of common hepatic artery in patients with and without variant artery in two groups. The nonparametric Mann-Whitney *U* test was used to evaluate the significance of the difference in diameters of the variant artery between the groups.

Results

Chi-square analysis revealed no significant gender difference between the groups. The mean values ± SD and ranges of the diameters of the common hepatic artery, superior mesenteric artery, and variant artery in two groups of patients are shown in Table. No significant difference was found between groups ($P = 0.07$) in mean common hepatic artery diameter. The mean diameter of the superior mesenteric artery and variant artery were also not significantly different between groups.

A significant difference ($P < 0.01$) was found between groups in the frequency of the variant artery; it was present in 53.3% of the patients in Group 1 and 11.5% of the patients in Group 2. In Group 1, the mean diameter of the common hepatic artery was not significantly different ($P = 0.05$) in patients with and without variant artery (7.3 ± 0.5 mm vs. 6.2 ± 0.5 mm). Examples of our cases are presented in Figs. 1 and 2.

Discussion

Portal vein patency and anatomic variations in the arterial blood supply of the liver are critical considerations in the preoperative assessment of patients who are candidates for liver surgery. Most of our knowledge regarding the variant hepatic artery anatomy is based upon angiographic data and derives mainly from the surgical and transplantation literature (8–13). The incidence of replaced and accessory right hepatic artery originating from the superior mesenteric artery in studies using different investigation methods varies ranging from 10.6% to 18% in various subject groups (8–13).

Gadolinium-enhanced MR angiography is a highly sensitive technique for the evaluation of the dual blood supply of the liver and provides important diagnostic information necessary for pretreatment work-up of liver disease (14–18). The anatomic variations in the hepatic artery have also been studied by using 3D MR angiography (18). Ishigami et al. studied variant hepatic anatomy in 84 patients awaiting orthotopic liver transplant (18). They found a higher posttransplant complication rate in patients with variant hepatic arterial anatomy than patients with classic anatomy. They also reported a smaller diameter of the distal common hepatic artery in patients with variant anatomy than in patients with classic anatomy. The authors suggested that the smaller caliber of the native common hepatic artery may contribute to the higher risk of arterial complications after transplantation of the liver (18). In our cirrhotic patients without cavernous transformation of the portal vein, the variant hepatic artery was visualized in 11% of the cases.

The diameter of the common hepatic artery increases in portal vein thrombosis (5). In our study, the mean diameter of the common hepatic artery in cirrhotic patients with cavernous transformation of the portal vein due to thrombosis was not significantly larger than that of the cirrhotic group. However, we also found that a variant hepatic artery pattern (right hepatic artery originating from the superior mesenteric artery) was more frequent in patients with cavernous transformation of the portal vein. In the embryogenic life arterial blood supply to the liver comes from different sources (19, 20). We speculate that such a variant vessel may become prominent in portal vein thrombosis when demand for the arterial blood flow increases. According to our data, half of the patients with portal cavernoma had a variant hepatic artery. Cavernous transformation is an important physiologic phenomenon because it can efficiently reestablish circulation after thrombosis. Biliary (cystic and paracholedochal veins) and gastric (left and right gastric veins) branches of the portal vein are the most frequently visualized collateral vessels contributing to the cavernous transformation (21).

HABR in patients with advanced cirrhosis may exist (22). It is reported that hepatic arterial vasodilatation, which occurs in response to reduced portal venous flow, provides substantial functional benefit in patients with cirrhosis (23).

There is a limitation of our study. Although we observed variant hepatic arteries originating from the left gastric artery during image analysis, we did not evaluate the frequency and significance of arterial variations supplying the left hepatic lobe. It is of interest that compensation afforded by the HABR was different in each lobe. Increase in arterial flow is an independent response to ipsilateral portal flow diminution and should not be accepted as a steal of the flow from one lobe to the other lobe (24). During our image analysis, a left hepatic artery originating from the left gastric artery was observed in 2 patients who had thrombosis in the left intrahepatic branch of the portal vein. In addition, we observed a right hepatic artery originating from the superior me-

Table. Mean values, standard deviations, and the ranges of diameters of the common hepatic artery, superior mesenteric artery, and variant artery in patient groups

Group	CHA (mm)	SMA (mm)	VA (mm)
1 (n = 15)	6.7 ± 1.0 (5–8.5)	8 ± 1.3 (5.6–10)	5.1 ± 1.7 (2.6–7)
2 (n = 26)	5.9 ± 1.5 (2.3–8.9)	7.6 ± 1.4 (4.8–10.3)	4 ± 0.4 (3.5–4.3)

CHA, common hepatic artery; SMA, superior mesenteric artery; VA, variant artery.
Numbers in parentheses represent the ranges of diameters.

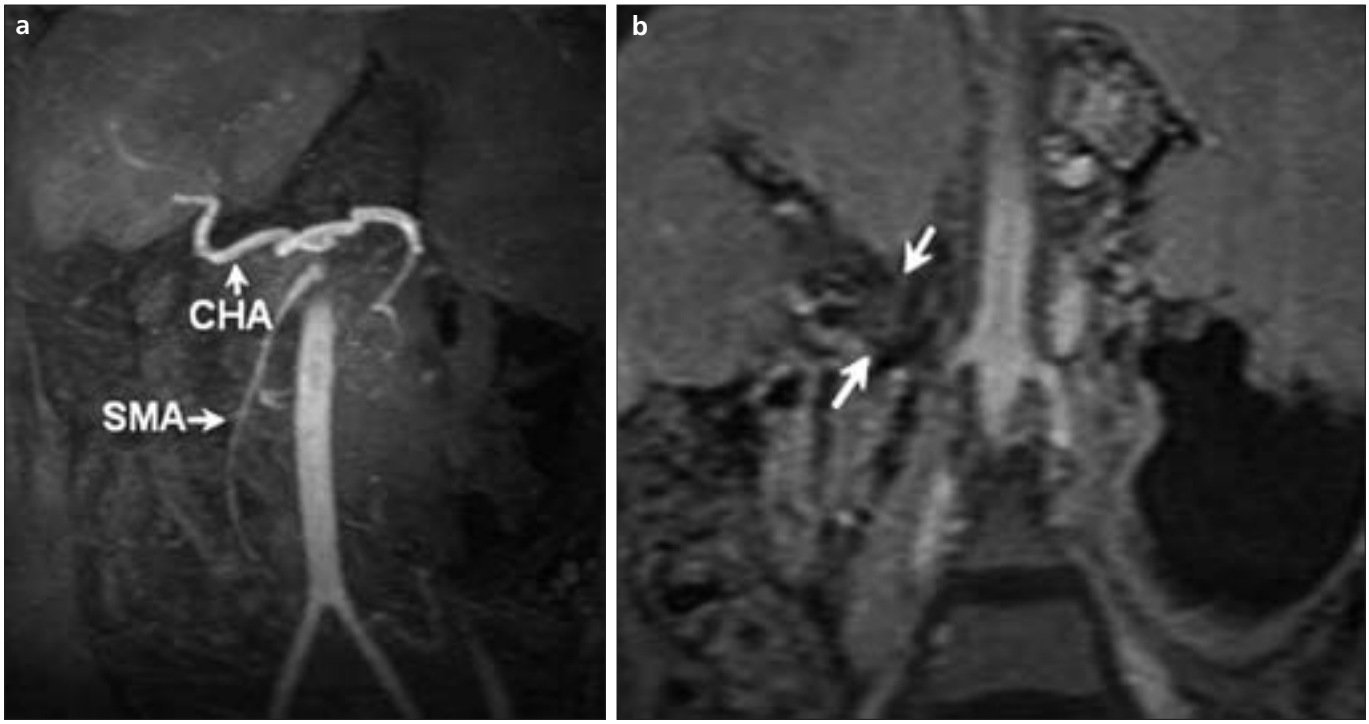

Figure 1. a, b. Dilated common hepatic artery (CHA) in a patient with cavernous transformation of the portal vein. Coronal maximum-intensity projection MR image obtained at arterial phase **(a)** shows that the CHA is larger than the superior mesenteric artery (SMA). Coronal portal venous phase source image obtained from MR angiography **(b)** in the same patient shows no enhancement in the main portal vein, consistent with thrombosis *(arrows)*. Serpiginous thin vessels at the portal hilum consistent with cavernous transformation of the portal vein can also be seen.

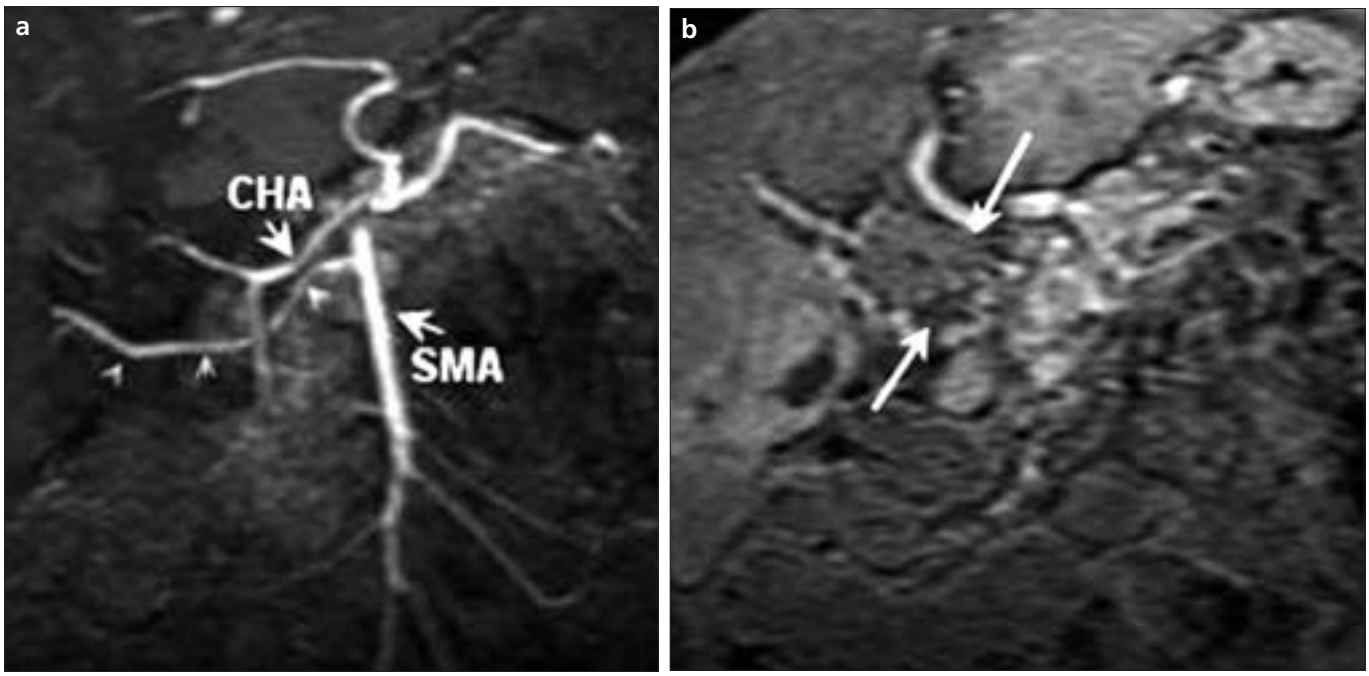

Figure 2. a, b. Coronal maximum-intensity projection MR image obtained at arterial phase **(a)** shows the right hepatic artery *(arrowheads)* originating from superior mesenteric artery (SMA) in a patient with cavernous transformation of the portal vein. The common hepatic artery (CHA) as well as the left gastric artery supplying the left hepatic lobe are also shown **(a)**. Coronal portal venous phase source image **(b)** obtained in the same patient demonstrates thrombus *(arrows)* in the main portal vein. Note also the thin, irregular vessels of cavernous transformation around thrombosed vein and enhancement differences in liver parenchyma.

senteric artery in a patient with right portal vein thrombosis. Although this was an interesting observation, the patients had patent main portal vein trunks and thus did not fulfill inclusion criteria for our study. This observation regarding the visualization of HABR on MR angiography in thrombosis of intrahepatic portal branches

was consistent with the findings in the case described by Gulberg and Schoenberg (7).

Viewed from a surgical standpoint, presence of a variant hepatic artery is an important consideration that could change the course of the surgical procedure in patients with portal vein thrombosis (which is no longer considered a definite contraindication for the liver transplantation). In our cirrhotic patients without portal vein thrombosis, a variant hepatic artery was present in 11.5% of the cases. This ratio was 53% in cirrhotic patients with portal cavernoma. In conclusion, vasodilatation at the level of intrahepatic arterioles (in a sense, HABR) in response to diminished portal flow may increase the frequency of detection of the variant hepatic artery.

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