The Cause of Nontumorous Defects of Portal Perfusion in the Hepatic Hilum Revealed by CT During Arterial Portography

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OBJECTIVE. We investigated the cause of nontumorous defects of portal perfusion in the hepatic hilum revealed by CT during arterial portography (CTAP).

MATERIALS AND METHODS. One hundred sixty patients who simultaneously underwent CTAP and CT during hepatic arteriography of the common hepatic artery formed the basis of our study. The frequency, site, and shape of nontumorous defects of portal perfusion in the hepatic hilum on CTAP and the findings on CT during hepatic arteriography were determined. In 13 patients in whom nontumorous portal perfusion defects were observed on CTAP, CT was performed during selective angiography via the gastric artery, pancreaticoduodenal artery, or both.

RESULTS. Nontumorous defects of portal perfusion were detected in 49 regions in 33 of the 160 patients (dorsum of segment IV, $n = 30$; dorsum of the lateral segment, $n = 11$; segment I, $n = 8$). Of the 33 patients, 16 had two defects each. Of the 49 nontumorous defects of portal perfusion, 38 showed enhancement on CT during hepatic arteriography. In the 13 patients who underwent CT during selective arteriography, enhancement due to nonportal venous inflow was seen in 16 of the 19 areas of decreased nontumorous portal perfusion (dorsum of segment IV, nine of 11; dorsum of the lateral segment, four of five; segment I, three of three).

CONCLUSION. The main cause of nontumorous defects of portal perfusion in the hepatic hilum revealed by CTAP is decreased portal inflow due to nonportal supply via the parabiliary venous system. Thus, such lesions were also enhanced at a high frequency on CT during hepatic arteriography.

Although use of noninvasive techniques such as dual-phase helical CT [1, 2] and MR imaging [3] has been increasing, CT during arterial portography (CTAP) has also found widespread acceptance as one of the most precise methods to detect hepatic tumorous lesions [2, 4]. Recently, its combined use with CT during hepatic arteriography has been attempted to further enhance diagnostic accuracy [5, 6]. Also, these examinations have become easier to perform because of the development of systems combining angiography equipment and CT [5]. On the other hand, paralleling the increase in the number of CTAP examinations, many reports have described areas of decreased nontumorous portal perfusion [7–12], most commonly in the hepatic hilum [10–12] and adjacent to the falciform ligament [9, 10]. Decreased areas of nontumorous portal perfusion appearing on CTAP have also been reported to be enhanced on CT during hepatic arteriography [13, 14]. Some nontumorous defects of portal perfusion in the hepatic hilum, such as the dorsum of segment IV and segment I, have been reported to be caused by nonportal venous return from gastric arterial systems [12, 15]; however, to our knowledge, the cause of all such decreased areas of nontumorous portal perfusion in the hepatic hilum has not been elucidated [14]. In surgical anatomic work, the presence of a venous system entering the liver parenchyma independent of the portal vein was first described by Sappey in 1859 [16] and has since been described by another group of researchers [17]. The present study was undertaken to characterize decreased areas of nontumorous portal perfusion seen on CTAP in the hepatic hilum and to clarify their cause radiologically.

Materials and Methods

The study cohort of 160 patients simultaneously underwent CTAP and CT during hepatic arteriography from the common hepatic artery for the detection of liver lesions between January 1994 and December 1997 at our institution and also satisfied the following study admission criteria: no stenosis or obstruction of the portal vein;

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none, or a few (<10), nondiffuse hepatic masses; no marked cirrhosis in clinical findings (not belonging to type C of Child-Turcott criteria); no developed hepatofugal portosystemic venous collateral or severe atrophic liver on diagnostic imaging; one hepatic artery supplying all hepatic segments; and no pathologic circulation in the liver such as arterioportal venous shunting. All patients underwent the study for the purpose of establishing angiographic diagnosis or treatment. The underlying diseases were metastatic liver tumors (n = 81 patients), hepatocellular carcinoma (n = 70), focal fatty change (n = 3), liver hemangioma (n = 3), focal nodular hyperplasia (n = 2), and hepatic angiomyolipoma (n = 1). The final 13 patients with decreased areas of nontumorous portal perfusion in the hepatic hilum revealed by CTAP also underwent selective catheterization consecutively to further identify the cause of portal venous perfusion defects. The vessels catheterized in these 13 patients were gastric artery (n = 3), posterosuperior pancreaticoduodenal artery (n = 2), and both (n = 8). Among 11 patients catheterized selectively from the gastric artery, five approaches were via the left gastric artery, five were via the right gastric artery, and one was via both left and right arteries.

Imaging Methods

CTAP was performed first with the tip of a 5-French angiography catheter inserted by Seldinger's method from the femoral artery to the origin of the superior mesenteric artery. Sixty to 80 ml of contrast medium (iopamidol, 150 mg I/ml) was injected at a rate of 2 ml/sec, with the CT scanning begun 30 sec after initiation of the contrast medium injection. As a rule, CT during hepatic arteriography was performed by placing the catheter tip in the common hepatic artery after digital subtraction angiography of the celiac and superior mesenteric arteries was completed, with 20 ml of iopamidol (150 mg I/ml) injected at a rate of 1 ml/sec and the imaging begun 5 sec after the start of the injection. A unified CT and angiography system (Interventional-CT System; Toshiba Medical System, Tokyo, Japan) was used. The unified CT and angiography system consisted of a CT unit (X-force before 1995 and X-vigor thereafter; Toshiba Medical System) and an angiographic unit also equipped with film-screen angiographic capacity (DFP-60A with KXO-80C; Toshiba Medical System). In the case of both CTAP and CT during hepatic arteriography, images were obtained during a single breath-hold with the entire liver imaged using a helical CT system (130 kV, 150 mAs). The beam width was 7 or 10 mm, table feeding speed was 7 or 10 mm/sec, and image reconstruction was performed with a width of 5 or 10 mm and a matrix of 512 × 512. CT during selective arteriography was performed by injecting 6–15 ml of iopamidol (150 mg I/ml) at a rate of 0.6–1 ml/sec from a catheter placed in the right or left gastric artery or posterosuperior pancreaticoduodenal artery, with the imaging begun 10–15 sec afterward. These conditions of contrast medium injection and the time of commencing CT under selective arteriography were determined with reference to the digital subtraction angiography performed immediately before. For digital subtraction angiography, iopamidol (370 mg I/ml) was injected at 24–30 ml, 5–6 ml/sec, in the case of superior mesenteric arteriography; 16–20 ml, 4–5 ml/sec, for common hepatic arteriography; and 6–15 ml, 0.6–1.0 ml/sec, for right or left gastric or posterosuperior pancreaticoduodenal arteriography. The total iodine dose used for this study ranged from 43.2 to 60.9 g I (mean, 46.9 g I). Informed consent was obtained from all patients.

Investigated Parameters

The following parameters were retrospectively investigated together by three radiologists experienced in abdominal diagnostic imaging with discussion continued until a consensus was reached: the frequency, site, and length of transverse diameter and shape on the largest transverse section of nontumorous defect of portal perfusion seen in the hepatic hilum on CTAP; the findings on CT during hepatic arteriography of decreased areas of nontumorous portal perfusion revealed by CTAP; and the findings of the patients in whom CT during selective arteriography was performed. Sixty-seven patients underwent intraoperative sonographic correlation. Ninety patients underwent follow-up imaging with CT, sonography, or MR imaging for a mean of 25.0 months (range, 7–55 months). Three patients with focal fatty change in the liver underwent needle biopsy of the portion showing decreased nontumorous portal perfusion on CTAP.

Results

Decreased areas of nontumorous portal perfusion were detected in the hepatic hilum on CTAP in 49 regions in 33 (21%) of 160 patients (Table 1). The site of appearance was the dorsum of segment IV in 18.8% (30/160) (Figs. 1A, 2A, and 3A), dorsum of the lateral segment of the left hepatic lobe in 6.9% (11/160) (Fig. 1A), and segment I in 5% (eight of 160) (Fig. 2A). In 16 of 33 patients, decreased areas of nontumorous portal perfusion were described in two areas. Their sizes ranged from 5 to approximately 35 mm (mean, 22.6 mm) (<10 mm, n = 18; ≥10 to <20 mm, n = 12; ≥20 mm, n = 19). The areas were broadly divided into wedge-shaped and serpiginous-shaped lesions, with the frequency of these shapes listed according to site in Table 2.

TABLE 1 Patients with Nontumorous Defects of Portal Perfusion Revealed by CT During Arterial Portography

Nontumorous Defects of Portal Perfusion	No. of Patients
Seen in one area	17
Segment IV dorsum	16
Lateral segment dorsum	1
Segment I	0
Seen in two areas	16
Segment IV dorsum and lateral segment dorsum	8
Segment IV dorsum and segment I	6
Lateral segment dorsum and segment I	2
Total	33

On CT during hepatic arteriography, 78% (38/49) of decreased areas of nontumorous portal perfusion were enhanced relative to the surrounding hepatic parenchyma: dorsum of segment IV, 73% (22/30) (Fig. 3B); dorsum of the lateral segment of the left lobe, 82% (nine of 11); and segment I, 88% (seven of eight). The border of the enhanced area was distinct in 29% (dorsum of segment IV, n = 8; dorsum of lateral segment, n = 3) and unclear in the remaining cases.

On CT during selective arteriography via the gastric or pancreaticoduodenal artery (or both), 16 (84%) of 19 decreased areas of nontumorous portal perfusion in 13 patients showed a definite enhancement (Figs. 1B, 1C, 2B, and 3C) due to venous drainage (Figs. 1D and 1E) showing a course differing from that of the portal vein on digital subtraction angiography (dorsum of segment IV, nine of 11; dorsum of the lateral segment, four of five; segment I, three of three). In one patient with nontumorous defect of portal perfusion in segment IV alone, the area was not enhanced on CT during either arteriographic method. Of nine enhanced areas at the dorsum of segment IV, five were revealed on CT during gastric arteriography and six were revealed during posterosuperior pancreaticoduodenal arteriography, with two areas overlapping during both gastric and pancreaticoduodenal arteriography (Figs. 1B, 1C, and 3C). All enhancements at the dorsum of the lateral segment and segment I were

TABLE 2 Decreased Areas of Nontumorous Portal Perfusion Revealed by CT During Arterial Portography: Shape and Location

Shape	Segment IV Dorsum	Lateral Segment Dorsum	Segment I
Wedge	22	6	5
Serpiginous	8	5	3
Total	30	11	8

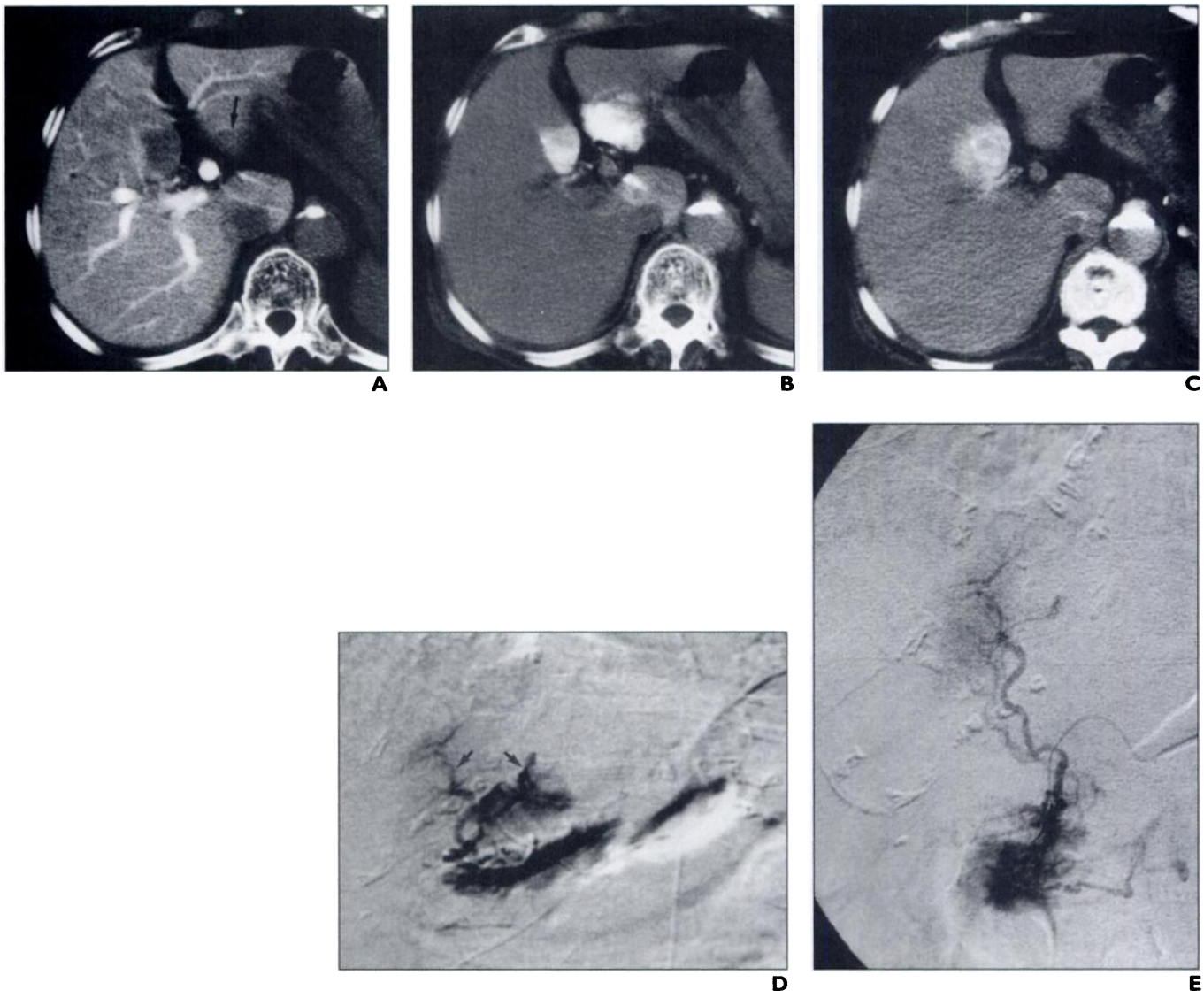

Fig. 1.—80-year-old woman with liver metastasis of colon cancer.
A, CT during arterial portography shows wedge-shaped regions of decreased portal perfusion in dorsum of lateral segment of left lobe (*arrow*) and dorsum of segment IV. Note that liver parenchyma adjacent to falciform ligament also shows decreased area of nontumorous portal perfusion.
B, CT during right gastric arteriography shows enhancement of wedge-shaped regions of decreased portal perfusion in dorsum of lateral segment and dorsum of segment IV. Part of segment I without decreased portal perfusion is also enhanced.
C, CT during posterosuperior pancreaticoduodenal arteriography shows enhanced area of dorsum of segment IV with extent of enhancement wider than decreased area of portal perfusion revealed by CT during arterial portography.
D, Right gastric arteriography shows two vessels to flow in direction of hepatic hilum in portal phase (*arrows*).
E, Posterosuperior pancreaticoduodenal arteriography shows nonportal vessels to flow in direction of hepatic hilum in portal phase.

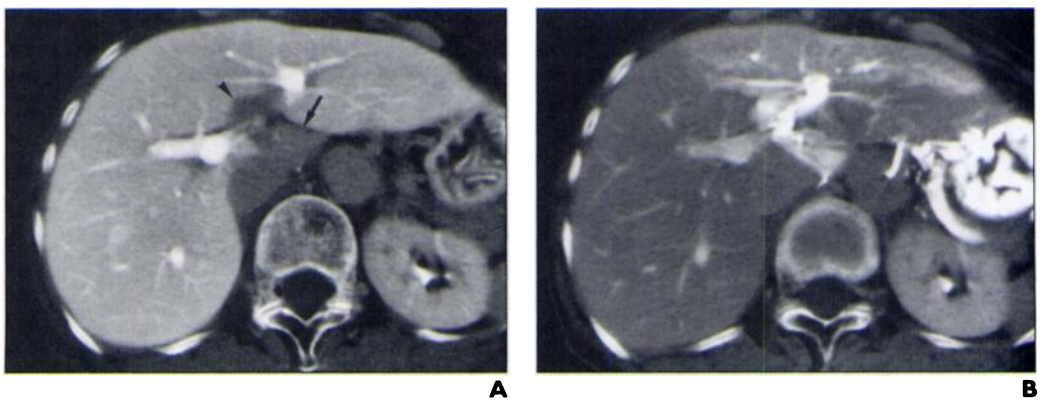

Fig. 2.—57-year-old woman with liver metastasis of pancreatic cancer.
A, CT during arterial portography shows serpiginous-shaped regions of decreased portal perfusion in segment I (*arrow*) and dorsum of segment IV (*arrowhead*).
B, CT during left gastric arteriography shows enhanced regions of decreased portal perfusion in segment I and dorsum of segment IV.

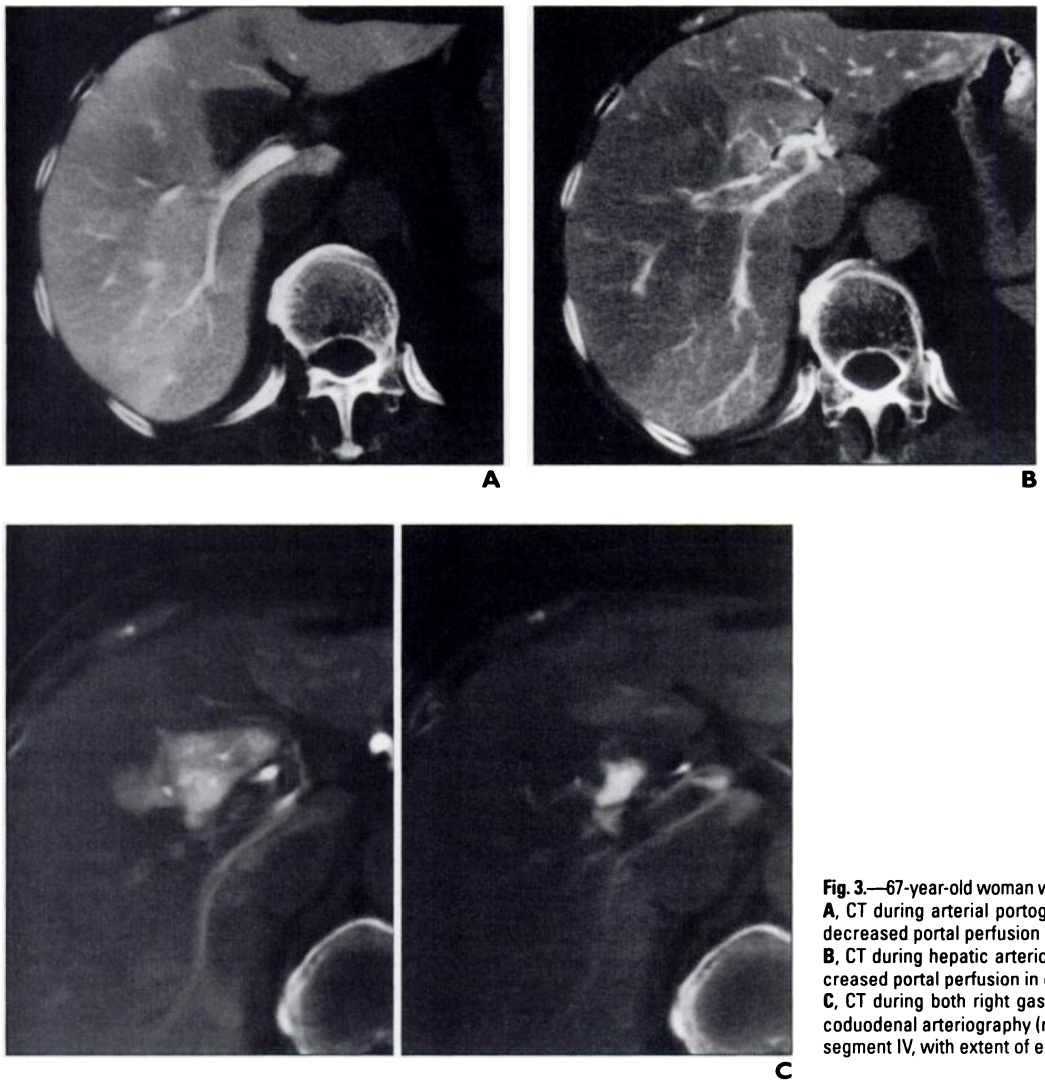

Fig. 3.—67-year-old woman with fat-spared area in diffuse fatty liver.
A, CT during arterial portography shows wedge-shaped region of decreased portal perfusion in dorsum of segment IV.
B, CT during hepatic arteriography shows enhanced region of decreased portal perfusion in dorsum of segment IV.
C, CT during both right gastric arteriography (left) and pancreaticoduodenal arteriography (right) shows enhancement in dorsum of segment IV, with extent of enhancement greater in former.

obtained on CT only during selective catheterization of the gastric artery. In four patients, nontumorous defects of portal perfusion showed simultaneous enhancement in two areas (Figs. 1B and 2B). Also, 56% (nine of 16) of decreased areas of nontumorous portal perfusion showing enhancement on CT during selective arteriography also showed enhancement on CT during hepatic arteriography (Fig. 3B).

Discussion

Nontumorous defects of portal perfusion have been revealed by CTAP in the hepatic hilum [7–12], with a frequency of 6–39% described in the dorsum of segment IV [10, 12, 14] and 1–7% in the dorsum of the lateral segment [9, 10]. Their sizes range from 5 to 40 mm [10, 12]. Most have been described as wedge-shaped or worm-shaped (described as serpiginous-shaped in the present study) [10, 12], whereas in cirrhotic livers the defects have been noted to be occasionally round [14]. When the present results were compared with those of earlier studies, the frequency and site of development, size, and shape were similar, suggesting that our subjects did not differ appreciably from those of the earlier studies [7–12]. To our knowledge, no previous reports have noted the frequency of decreased areas of nontumorous portal perfusion in segment I; we found a frequency of 5%. Some previous studies have reported that decreased areas of nontumorous portal perfusion in the hepatic hilum occasionally show partial nontumorous enhancement on CT during hepatic arteriography or early enhancement on dynamic CT with IV contrast injection [13–15]. Irie et al. [14] reported that on CT during hepatic arteriography 21 of 23 defects of nontumorous portal perfusion in the dorsum of segment IV revealed by CTAP showed enhancement; in our study a similarly high frequency of appearance of enhanced areas was noted: 73% in the dorsum of segment IV and 78% in the entire hepatic hilum. Many such decreased areas of nontumorous portal perfusion in the liver hilum revealed by CTAP are considered to be caused by direct nonportal venous supply into this area [12, 14, 15]. On the other hand, nontumorous enhancement on CT during hepatic arteriography or early enhancement on dynamic CT of these decreased areas of nontumorous portal perfusion has been explained by the relatively early venous inflow into decreased areas of nontumorous portal perfusion through nonportal vessels compared with that of the surrounding liver parenchyma [14, 15] and by concomitantly increased hepatic arterial blood supply [13].

Regarding the route of this venous inflow, Matsui et al. [12, 15] provided imaging evidence of drainage from the gastric vein to the dorsum of segment IV and segment I, which they termed "aberrant gastric venous drainage." However, in postgastrectomy cases as well, decreased areas of nontumorous portal perfusion

have been noted in the dorsum of segment IV [14, 18], suggesting the existence of venous inflow other than the main portal vein, the route of which is not through aberrant gastric venous drainage. In our study of the 13 patients who underwent CT during selective arteriography, in six of 11 patients in whom decreased areas of nontumorous portal perfusion were found in the dorsum of segment IV, enhancement of nontumorous defects of portal perfusion on CTAP was seen on CT during posterosuperior pancreaticoduodenal arteriography, confirming the existence of decreased areas of nontumorous portal perfusion other than aberrant gastric venous drainage due to nonportal venous supply. Also, because enhancement was found in 16 of 19 decreased areas of nontumorous portal perfusion in 13 patients, it was believed that most decreased areas of nontumorous portal perfusion in the hepatic hilum noted on CTAP derives from venous supply from the gastric arterial and pancreaticoduodenal arterial systems. Furthermore, 56% (nine of 16) of these decreased areas of nontumorous portal perfusion showing enhancement on CT during selective arteriography were also enhanced on CT during hepatic arteriography of the common hepatic artery.

The venous inflow from the pancreaticoduodenal arterial system confirmed in the present study is thought to correspond to the parabiliary venous system reported by Couinaud [17]. Aberrant gastric venous drainage is also thought to represent one communication between the gastric venous system and parabiliary venous system and, considered with our present finding of venous return from the pancreaticoduodenal arterial system, most nontumorous defects of portal perfusion in the hepatic hilum revealed by CTAP can be understood as originating from nonportal venous inflow via the parabiliary venous system, as a result of which they are enhanced by contrast medium injected into the gastric or pancreaticoduodenal artery on CT during hepatic arteriography of the common hepatic artery. If it is accepted that the parabiliary venous system is made up of a venous network extending to the parabiliary region [17] (Fig. 4), the existence of cases showing enhancement on CT during arteriography of both the gastric and the pancreaticoduodenal arterial systems and the existence of cases showing enhancement of multiple regions on CT during arteriography from a single route can be explained without contradiction. The fact that all nontumorous perfusion defects on CTAP in the dorsum of the lateral segment and segment I received venous return from the gastric arterial system in the present study shows that in these regions inflow from the gastric arterial system is probably predominant among the various veins in the parabiliary venous system. The enhanced areas of nonportal venous inflow on CT during selective arteriography with normal portal perfusion (Figs. 1B and 1C) are believed to be caused by anastomoses between the parabiliary venous system and peripheral portal branches.

As a practical application, other than its importance as a cause of nontumorous perfusion abnormalities on CTAP and CT during hepatic arteriography, the parabiliary venous system occasionally causes focally fat-spared areas in diffuse fatty liver [19], focal fatty change in the liver [18, 20], or focal hyperplastic change of the liver parenchyma [15] and might be developed as a collateral channel for the portal vein resulting from portal hypertension or obstruction [17]. Moreover, this venous system might be a pathway of direct hematogenous spread of metastasis from the gastric and pancreaticoduodenal areas to the hepatic hilum selectively.

This relationship between nontumorous defects of portal perfusion in the dorsum of segment IV and aberrant gastric venous drainage that is one part of the parabiliary venous system is well known [12, 15, 19, 20]. However, there are few reports about the relations between other parts of the parabiliary venous system, especially aberrant pancreaticoduodenal vessels, and few reports about other regions of the hepatic hilum supplied by this venous inflow, such as the dorsum of the lateral segment [18, 21]. To our knowledge, findings on CT during selective arteriography of the lateral segment influenced by the parabiliary venous system have not been described. In addition, few reports of CT during hepatic arteriography findings in the hepatic hilum with nonportal venous supply have been described, except for that seen at the dorsum of segment IV.

The limitation of the present study lies mainly in the small number of patients who underwent CT during selective angiography. The lack of selective arteriography of other arteries originating in the parabiliary venous system (except for the gastric and pancreaticoduodenal arteries), such as the cholecystic artery [17, 22], derives from difficulties in the selection of patients. Moreover, because selective arteriography of the gastric and pancreatic region, with or without CT, was not performed on patients without nontumorous defects of portal perfusion, we do not know if a patient can have anomalous venous inflow via the parabiliary venous system without a nontumorous perfusion abnormality on either CTAP or CT during hepatic arteriography.

In conclusion, decreased portal inflow due to nonportal blood supply via the parabiliary venous system can cause areas of decreased nontumorous portal perfusion in the hepatic hilum, as revealed by CTAP. Thus, this nonportal venous inflow can also frequently influence the nontumorous enhancement of such lesions on CT during hepatic arteriography of the common hepatic artery.

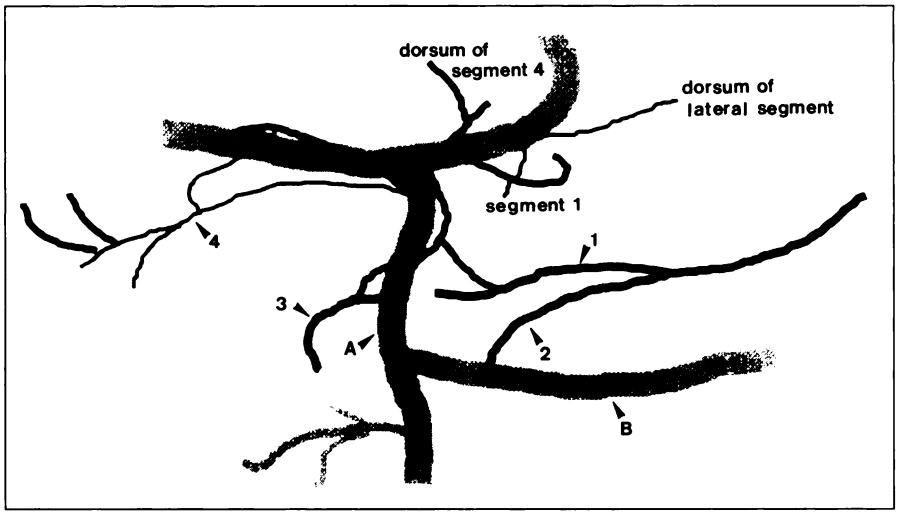

Fig. 4.—Schematic diagram of parabiliary venous system. A = portal vein, B = splenic vein, 1 = right gastric vein, 2 = left gastric vein, 3 = posterosuperior pancreaticoduodenal vein, 4 = cholecystic vein. (Reprinted [with modification] with permission from [17])

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