Cavernous Transformation of the Portal Vein: Patterns of Intrahepatic and Splanchnic Collateral Circulation Detected with Doppler Sonography

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OBJECTIVE. Cavernous transformation of the portal vein is defined as the formation of venous channels within or around a previously thrombosed portal vein. The purpose of this work was to study the hemodynamic consequences of cavernous transformation of the portal vein in a group of afflicted patients by use of Doppler sonography. We wished to study the evolution from portal vein thrombosis to the formation of cavernous transformation, the extent of resulting extrahepatic collateral channels, and the patterns of splanchnic collateral circulation.

MATERIALS AND METHODS. Seventy-five patients (48 adults and 27 children) with cavernous transformation of the portal vein were studied with color and/or pulsed Doppler sonography. Blood flow in the extrahepatic portal vein, in its segmental branches, in the hepatic veins and artery, and in the splanchnic veins was examined. Collateral pathways were sought. For nine patients with acute thrombosis of the portal vein, serial examinations were performed during the formation of cavernous transformation.

RESULTS. In nine patients, a fresh thrombus filled and distended the portal vein and became recanalized within a few days. Tortuous vessels appeared at the porta hepatis. These were characterized as veins or arteries with Doppler sonography. Soon the portal vein could no longer be identified within the mass of tortuous vessels. The cavernous transformation developed within 6–20 days of the acute thrombosis. A spongelike mass of collateral vessels around the main portal vein was seen in all but two patients. Intrahepatic extension of the cavernous transformation was seen in 57 patients (76%) and involved one or more intrahepatic portal veins. Two types of collateral circulation were observed: portosystemic, mainly through the left gastric and the perisplenic veins (the caput medusae, i.e., the paraumbilical-to-abdominal venous route, was never seen); and portoportal, from the periportal or pericholecystic venous channels to the intrahepatic portal veins. In nine patients, flow within unaffected intrahepatic branches of the portal vein was reversed and directed toward the cavernous transformation surrounding other, thrombosed intrahepatic segments of the portal vein.

CONCLUSION. After thrombosis of the portal vein, portoportal venous channels may form not only at the porta hepatis but also within the liver. Intrahepatic blood may be shunted from one segmental portal vein to another. In addition, portosystemic collateral channels are formed, suggesting that, despite extensive hemodynamic adaptations, portal hypertension ensues.

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The entity now known as cavernous transformation of the portal vein (CTPV) was first described by Balfour and Stewart [1] in 1869 as thrombosis and varicose dilatation of the portal vein leading to splenomegaly and ascites. Köbrich coined the term "cavernoma" to describe a pathologic condition in which the portal vein and lesser omentum present a peculiar spongy appearance because numerous fine blood vessels have penetrated these sites [2]. He also coined the phrase "cavernomatous transformation of the portal vein" to imply that this condition followed thrombosis of the portal vein.

The morphology of CTPV has been amply described in pathology [1, 2], surgery [3, 4], and radiology [5–14] publications. However, little is known about the evolution of cav-

ernous transformation after acute portal vein thrombosis, about the intrahepatic extension of the cavernous transformation, or about the intrahepatic and extrahepatic blood flow modifications that follow as an adaptation to the obstructed portal vein.

The purpose of this work was to study the hemodynamic consequences of CTPV in a group of afflicted patients by use of Doppler sonography. We wished to study the evolution from portal vein thrombosis to the formation of cavernous transformation, the extent of resulting extrahepatic collateral channels, and the patterns of splanchnic collateral circulation.

Materials and Methods

Between January 1986 and December 1992, 131 patients with sonographic findings of CTPV or thrombosis of the portal vein were examined at three university-affiliated institutions with pulsed (and, after 1990, also with color) Doppler sonography. The diagnosis of portal vein thrombosis was made as follows: echogenic material filled and distended the portal vein lumen completely (acute thrombosis) or partly (subacute or chronic thrombosis). No venous Doppler signals were detected in acute thrombosis. In chronic thrombosis, Doppler signals were detected around the clot. The diagnosis of cavernous transformation was made when a mass of tortuous vessels (with Doppler flow signals) was found at the porta hepatis or within the liver.

Patients with acute portal vein thrombosis had at least one follow-up examination up to 6 weeks later. If cavernous transformation developed, the patient was included in the study. In 56 of 131 patients, no cavernous transformation developed after portal vein thrombosis. These patients were excluded from the study. Of the remaining 75 patients, 66 had a cavernoma at the first examination and nine had an acute thrombosis of the portal vein. Cavernomas were noted at the first follow-up examination. For the latter nine patients, five or more serial examinations were performed to study the evolution from portal vein thrombosis to cavernous transformation.

The study group included 37 females and 38 males, from 10 months to 85 years old. Twenty-seven were children less than 16 years old. The mean age of the children was 11 years, and that of the adults was 36 years. The diagnosis of cavernous transformation, made on the basis of sonographic findings [5, 6, 9–12], was supported by the results of contrast-enhanced CT imaging for 49 patients, angiography for 17, and MR imaging for seven. Some patients had several examinations; 15 patients had sonography only.

To ascertain a possible cause of portal vein thrombosis that would have led to the formation of cavernous transformation in 75 patients, we reviewed the clinical history for all of these patients as well as previous medical records when available. We found the following causes of portal vein thrombosis in the 27 children: umbilical vein catheterization (n = 11), dehydration and shock (n = 7), coagulopathy (n = 2), and liver transplantation (n = 2); the cause was unknown in five. In adults, the causes included the following: coagulopathy (n = 19), cirrhosis (n = 5), pylephlebitis or pancreatitis (n = 4), splenectomy (n = 2), and congenital hepatic fibrosis (n = 1); the cause was unknown in 17. No patients with CTPV had an underlying tumor of the liver, bile ducts, or pancreas. The time that elapsed between the event and the discovery of cavernous transformation ranged from 2 days to more than 30 years. Presenting symptoms and signs included intestinal hemorrhage, abdominal pain, splenomegaly, and ascites. Three patients had asymptomatic cavernous transformation.

We used the following commercially available sonographic machines: Toshiba SSA-270A (Tokyo, Japan), Acuson-128 (Mountain View, CA), ATL Ultramark 5 and 8 (Advanced Technology Laboratories, Seattle, WA), and Siemens Quantum II (Jassaquah, WA). Technical factors were adjusted to obtain the highest Doppler shift from the blood flow in the vessels examined: Doppler beam/vessel angle of less than 60°, low pulse repetition frequency, low wall filter, Doppler transducer frequency of 3 or 5 MHz, and sample volume of 3-mm diameter. Gray-scale spectral display and color images were recorded on film or photographic paper.

All sonographic examinations were done and interpreted by one of three radiologists experienced in hepatic and Doppler sonography. (Because of the complexity of the technique involved, none of the examinations was performed by a sonographer.) The examination technique was as follows: the fasting patients (8 hr for adults and 2–4 hr for children) had a real-time sonogram of the entire abdomen and then a Doppler examination of the splanchnic veins, the main portal vein and its right and left intrahepatic branches and their segmental divisions, and the hepatic veins [15]. The direction of blood flow and the spectral flow patterns [16–18] within each vessel were studied. Collateral veins were sought around the gallbladder and in the liver, the liver hilum, the lesser omentum, the renal and splenic hila, the pancreas, the pelvis, and the anterior abdominal wall. Hepatic arterial signals were sought at the porta hepatis and were described as normal, low, or high.

Results

In the nine patients with acute portal vein thrombosis, the vein was first filled and then distended by an echogenic clot. Fenestrations of the clot occurred within a few days. These were later observed to be filled with flowing blood (Fig. 1). Concurrently, tortuous vessels appeared at the porta hepatis (Figs. 2 and 3). Both venous blood flow and arterial blood flow were observed within these vessels. The formation of this spongelike mass (cavernoma) at the porta hepatis occurred within 6–20 days of the acute event. The previously normal-caliber portal vein could no longer be detected within the cavernoma.

In the remaining 66 patients, cavernous transformation was seen in all but two, in whom the main portal vein appeared normal, and cavernous transformation was limited to the right and left intrahepatic portal veins and a few segmental branches. Intrahepatic extension of the cavernous transformation was seen in 57 patients (76%) and involved one or more intrahepatic portal veins. The cavernous transformation extended into the liver around both right and left portal veins in 53 patients (Fig. 1) and exclusively into the right portal vein in four patients.

Two types of collateral circulation were observed: portosystemic and portoportal. In 65 patients (87%), extrahepatic portosystemic collateral veins were found. Forty-nine patients had hepatofugal flow in a dilated left gastric vein or dilated veins in

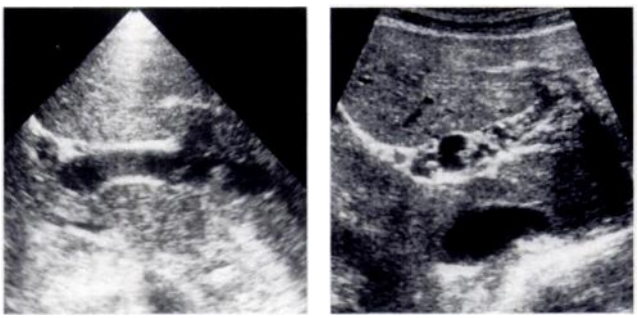
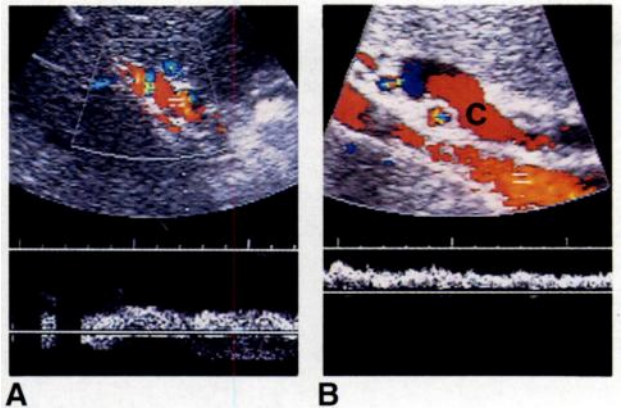


Fig. 1.—Formation of cavernoma and extension into left portal vein.
***A* and *B*, Oblique sonograms of left side of liver show that left portal vein is distended with fresh thrombi (*A*) and that, a few days later, "spongy appearance" of cavernoma has replaced left portal vein (*B*).**



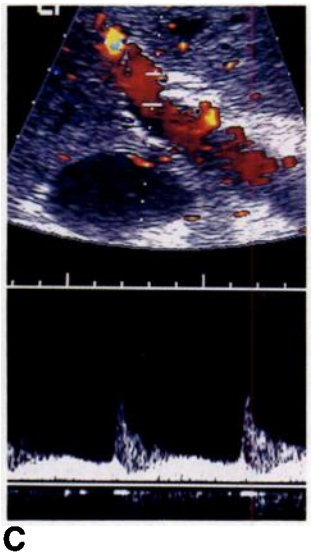


Fig. 2.—Comparison of Doppler shifts from two cavernomas with those from normal portal vein.

***A*, Right parasagittal color Doppler sonogram at porta hepatis shows typical "spongelike" appearance of portal vein cavernoma.**

***B*, Portal vein cavernoma with prominent choledochal vein (c).**

***C*, Normal pulsatile flow is synchronous with hepatic arterial signals. Note that Doppler spectra in *A* and *B* are rather flat in comparison with that shown here.**

the lesser omentum. Other collateral routes included splenogastric, splenorenal, mesentericogonadal, peripancreatic, and hemorrhoidal veins. None of our patients had a dilated paraumbilical vein, and no flow signals were obtained from within the round or falciform ligaments. Two patients had an intrahepatic portosystemic shunt. Blood flowed from the extrahepatic cavernous transformation into a newly formed vein that crossed the liver parenchyma and entered the middle hepatic vein (Fig. 3D).

Portoportal collateral channels served as a communication between the extrahepatic portal circulation and the intrahepatic portal veins. Dilated veins around the common bile duct or the main portal vein, seen in 73 patients, constituted the cavernous transformation. In 57 patients, the cavernous transformation extended into the liver, following the course of the right, the left, or both portal veins. In nine patients, flow within unaffected intrahepatic branches of the portal vein was reversed and directed toward the cavernous transformation surrounding other, thrombosed intrahepatic segments of the portal vein; for example, blood from the healthy left portal vein nourished the right lobe through the cavernous transformation surrounding the thrombosed right portal vein (Fig. 4). In 11 patients, dilated pericholecystic veins shunted blood into a patent branch of the right portal vein (Figs. 3A–3C). We were able to demonstrate hepatopetal flow in pericholecystic veins in three of these patients. In the other eight, venous flow was demonstrated in these tortuous vessels, but entry into the right portal vein was not directly outlined. Another portoportal collateral route involved the extrahepatic portal vein via a newly formed vein that traversed the liver parenchyma and entered a hepatic vein. This route was seen in two patients.

Flow in the venous channels at the porta hepatis was hepatopetal in all patients. In most patients, flow at the porta hepatis and in intrahepatic portal veins was steady and of low velocity, without the heart-modulated, slightly pulsatile flow observed in healthy persons [15, 18] (Fig. 2).

Doppler examination showed that the cavernous transformation contained not only veins but also the hepatic artery. Hepatic arterial Doppler shifts (both systolic and diastolic) were prominent in nine patients during acute portal vein thrombosis and in 35 of the 66 patients with established cavernous transformation. Results of Doppler examination of the hepatic veins were normal in all patients.

Discussion

Three theories explain the pathogenesis of CTPV: (1) a cavernoma is a congenital malformation that replaces a nondevel-

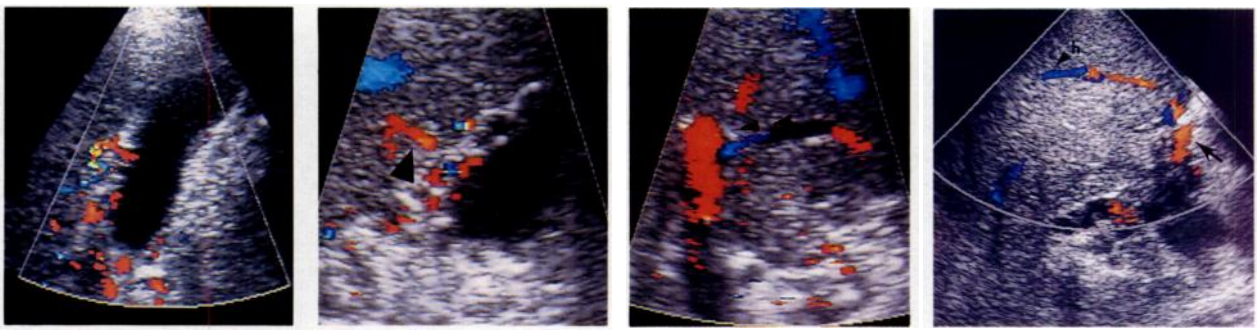


Fig. 3.—Transhepatic shunts in two patients with portal vein cavernoma.

***A–C*, Color Doppler sonograms show that signals from veins around gallbladder (*A*) can be traced to obliquely oriented vessel (*arrowhead* in *B*), which drains into portal vein branch to segment 5 (*arrowheads* in *C*).**

***D*, Color Doppler sonogram shows that flow signals from hilar vein (*arrow*) can be traced to branch of portal vein and to hepatic vein (*arrowhead* labeled h).**

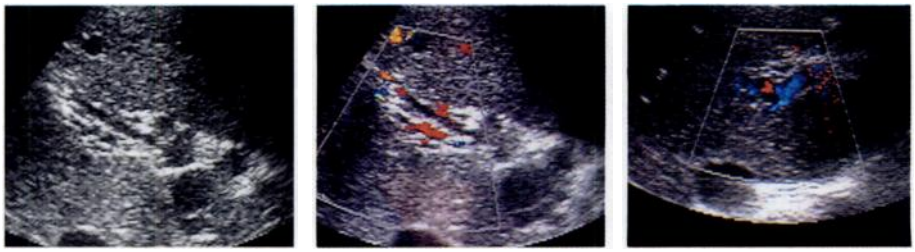


Fig. 4.—Portal vein cavernoma with extension into liver: segment-to-segment shunting occurred.

***A–C*, Oblique gray-scale and Doppler sonograms of right (segment 5, *A* and *B*) and left (umbilical portion, *C*) portal veins. Portal vein branch to segment 5 is obstructed and is replaced by collateral veins with hepatopetal flow signals (cavernoma). Left portal vein is patent and of normal caliber. Flow is reversed and directed toward right lobe, as outlined by blue Doppler signals in *C*. In this way, segment 5 receives portal venous blood from healthy left lobe.**

oped portal vein [1]; (2) a cavernoma is a hemangioma of the portal vein; and (3) a cavernoma is the end product of thrombosis of the portal vein [2]. In 1928, Klemperer [2] reviewed 22 cases in the literature and described a 42-year-old woman who died of pulmonary embolism after 9 years of splenomegaly followed by ascites and gastric hemorrhage. After autopsy, he concluded that the portal vein was present but thrombosed and that the cavernoma represented recanalized thrombus and periportal collateral venous channels. Despite this elegant description and that made by Balfour and Stewart in 1869 [1], the three theories explaining the pathogenesis of CTPV persist in the modern medical literature. In 1977, Odièvre et al. [19] found a greater frequency of congenital malformations (of the heart, kidneys, intestine, ovaries, and skeleton) in children with "idiopathic" extrahepatic portal hypertension than in children who had portal vein thrombosis after umbilical catheterization and concluded that some cavernomas are congenital. In most of the children in our study, the cause of portal vein thrombosis was known. Only one child had a congenital malformation (aortic coarctation), but this child also suffered shock. We were therefore unable to support the theory of Odièvre et al. that some cavernomas are congenital.

In adults, the main causes of portal vein thrombosis are hepatocellular carcinoma, pancreatitis, cirrhosis, liver transplantation, and splenectomy, along with the diseases already mentioned for children [20–22]. Whatever the cause, portal vein thrombosis may be followed by lysis of the thrombus with or without cavernous transformation. In patients with cirrhosis and other liver diseases causing increased resistance to portal inflow, flow velocity in the portal vein tends to decrease [23]. This situation may explain why even if portal vein thrombosis is relatively frequent in these patients (e.g., 7.2% reported by Mori et al. [24] and 15% reported by Kunstlinger et al. [25]), few patients show recanalization of portal vein thrombi and formation of cavernous transformation. (Only five of our patients with cavernous transformation had underlying cirrhosis.) Patients without liver disease seem to show more success in the formation of cavernous transformation, and the majority of our patients had clinically healthy livers.

How does CTPV occur? For nine patients, we witnessed the transition from acute thrombosis of a morphologically normal portal vein to classic cavernous transformation (Figs. 1 and 2), supporting original theories that cavernomas form in response to portal vein thrombosis. This process took 6–20 days, a shorter period than the 1–12 months previously reported [26]. The cavernous transformation formed in or around the main portal vein in all of our patients. The veins constituting the cavernous transformation were believed to be paracholedochal veins [6] as well as recanalized channels within the portal vein thrombus (Figs. 1 and 2).

How does the intrahepatic circulation adapt to thrombosis of the portal vein? The veins constituting the cavernous transformation extended around the intrahepatic portal veins in 57 patients (76%). We were surprised by the large number of our patients (76%) who had extension of the cavernous transformation into the liver, well beyond the bifurcation of the portal vein. This phenomenon has not been emphasized in classic descriptions of cavernous transformation. It is likely that cavernous transformation forms around portal vein thrombi, both at the porta hepatis and around affected lobar or segmental branches, in most patients. We probably detected this intrahepatic extension more frequently in living persons because we used a noninvasive technique without injection of contrast material. Recent improvements in sonographic technology (especially color Doppler imaging) have allowed better detection of intrahepatic vessels, as well as the arterial or venous flow within them. Our Doppler sonographic technique for examining the liver includes a careful search of segmental portal veins [15]. Such a search would be expected to yield greater detection of intrahepatic vessel abnormalities than routine abdominal sonography.

We were able to show that flow in the vessels constituting the cavernous transformation was hepatopetal, and this fact supports the theory that cavernous transformation forms as a bypass route (or portoportal collateral channels) between the splanchnic veins around the obstructed portal vein and intrahepatic portal veins. Flow in segmental portal veins was also hepatopetal, except in nine patients, who had redistribution of blood flow from one segmental portal vein to another, around a nearby obstructed segment of the portal vein. This phenomenon is also seen in certain liver diseases, such as cirrhosis. The mechanism for this intrahepatic redistribution is probably an uneven resistance to blood flow. As resistance increases, blood flow in the severely affected area may be reversed and shunted into the relatively healthier area. This mechanism is also seen in patients with Budd-Chiari syndrome, in which the caudate lobe receives blood flow from other segments, its resistance to blood flow being normal because of the patency of its own hepatic vein(s). When the cavernous transformation extends into the liver, portal blood attempting to enter a region of particularly high resistance may be diverted into another segmental vein in which resistance to inflow is lower. The source of blood in the segmental portal veins with reversed flow is the hepatic artery, through arterioportal shunts at the sinusoid [27].

A second portoportal collateral route involved the veins of Sappey in the wall of the gallbladder. These pericholecystic veins shunt blood into a branch of the right portal vein when patent.

Flow in the periportal and pericystic veins in our patients with cavernous transformation was easily detected (Figs. 1–3). It was steady, or "flat," without the gently undulating heart-modulated pulsations seen in healthy persons (Fig. 2). Weltin et al. [13] and Raby and Meire [14] found similar flow at the porta hepatis in their patients with cavernous transformation. Blood flow through the recanalized thrombus and through collateral vessels may explain the obliteration of normally transmitted cardiac pulsations. Such Doppler shifts may also be shown for other tortuous portosystemic collateral veins, such as the left gastric, paraduodenal, or retroperitoneal veins.

Despite the formation of extrahepatic and intrahepatic shunts, portal hypertension persisted in the majority of our patients, as witnessed by the presence of portosystemic collateral veins in 87%. This finding supports that of Raby and Meire [14], who found the velocity of blood flow in large collateral veins at the porta hepatis of patients with cavernomas to be well below that of flow in the portal vein of control subjects (2–8 cm/sec rather than 8–16 cm/sec). The main portosystemic shunt route was through the left gastric vein, although several other routes were noted. It is interesting that none of our patients had portosystemic collateral channels involving the paraumbilical vein. This route depends on patency of the left portal vein and is usually seen in patients with intrahepatic portal hypertension, especially cirrhosis. As the obstruction in patients with CTPV is prehepatic, the paraumbilical–abdominal vein collateral route would serve no purpose. This route is possible, however, in patients in whom portal hypertension preceded the portal vein thrombus. In such patients, the paraumbilical vein can then serve as an inflow route, bringing blood into the liver from systemic veins of the abdominal wall.

Probably in response to decreased blood flow in the intrahepatic portal veins, blood flow in the hepatic artery increases [28]. The estimation of arterial flow volume by Doppler techniques is difficult, because vessel beam angles and vessel caliber cannot be readily calculated. The diagnosis of increased hepatic arterial Doppler shifts is therefore a subjective one. Dynamic CT studies of patients with CTPV have shown uneven attenuation of hepatic parenchyma, with some areas showing intense accumulation of contrast medium. This phenomenon is thought to be attributable to regionally increased arterial flow [7].

This retrospective study suggests that CTPV is composed of periportal and intrathrombotic venous and arterial channels that form after thrombosis of the portal vein. Cavernous transformation usually occurs in healthy livers, in which it forms a bypass route around the obstructed portal vein into patent intrahepatic portal veins with normal resistance. Cavernous transformation usually extends from the porta hepatis into the liver, around lobar and/or segmental branches of the portal vein. Blood may be shunted from a healthy liver segment into an affected one. The time needed for cavernous transformation to form after acute portal vein thrombosis is shorter than previously described—just a few days after the acute event. Despite extensive intrahepatic and extrahepatic shunts, portal hypertension persists.

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