

Asymptomatic splenic artery occlusion in a child: incidental detection with Doppler ultrasonography

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ABSTRACT

Splenic arterial occlusion is a rare condition usually presenting with signs that are due to splenic infarction. To the best of our knowledge, splenic artery occlusion without splenic infarction has not been reported in the literature. The case presented here was asymptomatic, except for hypertension. The splenic artery occlusion was detected incidentally during the color Doppler examination of renal arteries that was performed to rule out stenosis as a cause. The striking Doppler finding that led to the final diagnosis was peri-pancreatic multiple tubular structures consistent with collateral flow.

Key words: • splenic artery occlusion • splenic infarction • Doppler ultrasonography

Abnormalities of the splenic artery, particularly stenoses and occlusions, are rare types of acquired disorders of splanchnic circulation. Blunt trauma, liver transplantation surgery or pancreatectomy, and torsion of the wandering spleen are some of the etiological factors of stenosis and occlusion of the splenic artery (1-3). A thromboembolic process of cardiac origin is also a reported etiological factor (4).

Herein, an 11-year-old girl with segmental occlusion of the splenic artery is reported. This abnormality was detected with color Doppler ultrasonography during the patient's hypertension work-up. There was no etiological factor for segmental occlusion of the splenic artery in the history, except a minor blunt abdominal trauma that did not require any medical management, which occurred several months earlier. Although a segment of the splenic artery was completely occluded, no infarction of the splenic parenchyma was detected on ultrasonography and computed tomography (CT).

Case report

An 11-year-old girl presented to the department of pediatrics with headache. Her blood pressure was 130/80 mmHg and the physical examination was otherwise unremarkable. Complete blood count, urinalysis, and urine culture revealed no abnormalities. Blood urea nitrogen, creatinine, serum calcium, and uric acid levels were normal. Urinary catecholamine levels were unremarkable. There was no abnormality in the lipid profile. The electrocardiogram and echocardiogram showed no abnormalities.

In order to rule out renovascular hypertension, renal ultrasonography was performed. The kidneys were normal in size and parenchymal thickness, with normal echogenicity. In Doppler examination, renal arteries were normal in both color and spectral mode. Small tubular structures, with lumens that were filled with color, were seen near the body and tail of the pancreas (Figure 1). This appearance was suggestive of collateral blood flow. The celiac trunk and common hepatic artery were patent, whereas the splenic artery could not be visualized, indicating a possible occlusion. A diagnosis of splenic artery occlusion was made. A small patent segment of the distal splenic artery at the splenic hilus was also demonstrated.

The splenic vein was of normal caliber showing normal intraluminal blood flow consistent with patency. The spleen was in normal size and without evidence of ischemic changes. On digital subtraction angiography (DSA), the splenic artery occlusion was confirmed. DSA also showed collateral vessels in the peri-pancreatic region (Figure 2).

Due to the possibility of associated vascular pathologies, a series of laboratory tests were performed, but they revealed no sign of any etiological factors, such as vasculitis.

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Received 21 April 2005; revision requested 25 April 2005; revision received 2 May 2005; accepted 5 May 2005.



Figure 1. Axially oriented color Doppler image at the midline shows numerous small caliber structures consistent with collateral flow at the pancreatic area. White line represents the pancreatic borders (P: pancreas).

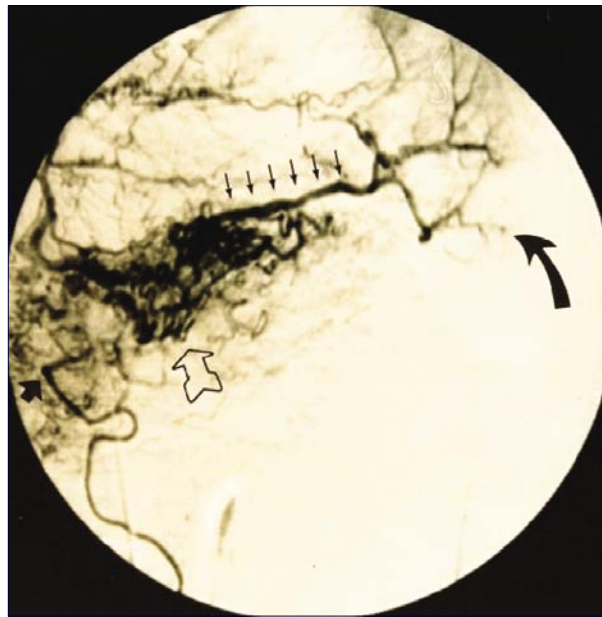


Figure 2. Arteriography performed in the anteroposterior projection with the catheter tip (*black arrow*) at the splenic artery orifice demonstrated occlusion of the proximal splenic arterial segment. The distal half of the splenic artery (*small black arrows*) up to the splenic hilus can be seen, filling via multiple collateral vessels (*big open arrow*). Splenic parenchymal vasculature and splenic size is considered normal (*curved arrow*).

During 5 years of follow-up, the child did not present any signs or symptoms associated with this splenic artery occlusion. Routine ultrasonography and Doppler examinations on a yearly basis demonstrated no evidence of splenic infarction. Hypertension was attributed to chronic glomerulonephritis, which was not related to the splenic vascular occlusion.

Discussion

Non-visualization of the splenic artery may be related to an acquired occlusion, severe stenosis, or the congenital absence of the splenic artery. Major features of congenital absence of the splenic artery are non-visualization of the splenic artery and filling of the intrasplenic branches via collateral channels (5, 6). Existence of a small, patent segment at the splenic hilus excluded the congenital form in the presented case.

The use of color Doppler ultrasonography findings for the incidental diagnosis during a hypertension work-up in the present case was unique and interesting. The presence of a generous peri-pancreatic arterial vasculature raised the question of splenic artery thrombosis, which was subsequently demonstrated with color Doppler ultrasonography, and angiography. The celiac and hepatic arteries were visualized well and patent.

Other diagnostic considerations included atherosclerotic occlusion of the splenic artery, previous surgical intervention of the splenic artery, and splenic artery thrombosis. Atherosclerosis was very unlikely because of the age of the patient and the absence of familial hypercholesterolemia. The patient had no history of a surgical procedure or a cardiac disease.

Acute occlusion of the splenic artery results in infarction of the splenic parenchyma. Patients present with left upper quadrant abdominal pain, fever, chills, nausea, vomiting, pleuritic chest pain, and left shoulder pain (3). Massive gastric bleeding from sub-mucosal gastric collateral vessels, secondary to the splenic artery occlusion, has also been reported (7). None of these clinical symptoms were present in our patient.

Initially, a minor trauma that did not require any medical attention was suspected as the cause of our patient's splenic arterial occlusion. Possible damage to the vessel wall during this minor trauma, leading to a progressive narrowing of the lumen was also considered. The narrowing must have progressed gradually, so that there was time for the development of the collateral vessels, as there was no identifiable injury of the splenic parenchyma.

In conclusion, splenic artery occlusion without associated infarction of the splenic parenchyma is a unique condition. On color Doppler ultrasonography, identification of prominent collateral vasculature in the peri-pancreatic region and splenic hilus may be a clue to its diagnosis.

References

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