Mesenteric Arteritis Complicating Surgical Repair of Coarctation of the Aorta*

Angiographic Findings and Management

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We report a case of mesenteric arteritis complicating repair of coarctation of the aorta in a patient with paradoxical hypertension. The severe vasospastic changes in the mesenteric vessels and the response to treatment with antihypertensive drugs and papaverine perfusion directly into the mesenteric artery were documented by serial angiography. The patient improved rapidly with treatment and recovered completely.

First described by Sealy in 1953, mesenteric arteritis is now a well-recognized complication after surgical repair of coarctation of the aorta. It is reported to occur in 2 to 28 percent of patients.1,2 Also known as the "postcoarctectomy syndrome," it is characterized by acute abdominal pain and distension, ileus, vomiting, and occasionally intestinal bleeding, occurring three to seven days postoperation. Although the precise physiologic mechanism is still not fully understood, this syndrome is related to the paradoxical hypertension frequently observed during the postoperative period.3 This is a serious and potentially lethal condition since it may lead to severe intestinal ischemia, necrosis, and perforation.4 In this report, a case of mesenteric arteritis with complete angiographic documentation of the evolution is presented. In addition to antihypertensive treatment, direct continuous infusion of papaverine into the mesenteric artery was also used and produced rapid recovery. To our knowledge, the angiographic changes in this syndrome and their evolution with treatment have not been reported previously, or the use of papaverine in the treatment of this condition.

Case Report

A 26-year-old man was admitted to the Montreal Heart Institute for surgical repair of coarctation of the aorta diagnosed eight years previously. He had never been treated

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for hypertension and had remained asymptomatic until three days prior to admission, when he presented with an episode of severe systemic hypertension following basket extraction of a left ureteral calculus. At the time of admission, systemic blood pressure was 180/90 mm Hg in both upper extremities and 120/90 mm Hg in both legs. The femoral pulses were markedly decreased and delayed in comparison to the right humeral pulse. A systolic murmur, grade 4/6, and a diastolic murmur, grade 2/6, were heard at the second left intercostal space. The ECG showed signs of left ventricular hypertrophy and the chest x-ray film showed normal heart size with a cardiothoracic ratio of 14.5/30.5 cm. The ascending aorta was slightly dilated. There was no rib-notching. Cardiac catheterization confirmed the diagnosis of coarctation of the isthmus of the aorta, with a gradient of 75 mm Hg across the coarctation. The ductus arteriosus was not patent. The collateral circulation was markedly developed. The aortic valve was bicuspid, with a transvalvular gradient of 10 mm Hg and regurgitation graded 3/4. The left ventricle was normal.

Resection of the coarctation and replacement with an 18 mm Dacron velour graft was performed without complication. The resected aortic segment showed typical coarctation with a residual lumen of 0.3 cm in diameter. During the 40 minutes of aortic cross-clamping, intravenous nitroglycerin perfusion was used to decrease systemic blood pressure. Immediately after surgery, arterial blood pressure was 210/110 mm Hg and decreased to 150/80 mm Hg with intravenous nitroglycerin perfusion between 30 and 50 μg/kg/min. Twenty-four hours later, methyldopa, 250 mg every six hours, was added. For two days, blood pressure remained difficult to control and varied between 130/80 and 180/100 mm Hg. On the third postoperative day, the blood pressure stabilized at 150/80 mm Hg and nitroglycerin was discontinued. The patient was given a normal diet and methyldopa orally. On postoperative day 6, the patient suddenly complained of severe abdominal pain. The abdomen was not distended and there were no signs of peritoneal irritation, but peristaltic sounds were absent. The patient was afebrile with blood pressure of 150/90 mm Hg and normal femoral pulses. The white blood cell count was normal. Gastric drainage was instituted.

Three hours after the clinical onset of symptoms, selective angiography of the superior mesenteric artery was performed using a 7F polyurethane preshaped catheter introduced through the right femoral artery by the Seldinger technique. The main stem of the mesenteric artery was normal. However, there was a complete cut-off of the first two branches and marked beading on two other branches of the mesenteric artery (Fig 1). Distal progression of the contrast medium was delayed with faint opacification of the distal arteriolar network, and no venous return was seen on late films. The findings indicated severe vasoconstriction of the mesenteric vessels. In spite of the dilator effects of the contrast medium, the patient complained of increased abdominal pains during the procedure. A bolus of papaverine, 60 mg diluted in 10 ml of normal saline solution, was therefore injected through the catheter directly into the mesenteric artery, and a second angiogram obtained within 30 minutes. The second injection showed reopening of the two occluded proximal branches of the mesenteric artery, but the beaded aspect of the other branches unchanged (Fig 2). Distal progression of the contrast medium was more rapid compared to the first angiogram indicating partial relief of the vascular spasm. The arterial catheter was left in place and a continuous perfusion of papaverine at 1 mg/min was administered into the mesenteric artery. In addition, a continuous perfusion of nitroprusside at 50 μg/kg/min and methyldopa, 500 mg every six hours, were given intravenously. Within a few hours, the abdominal pains stopped and peristaltic sounds returned. The mesenteric perfusion with papaverine was

Figure 1. Selective angiogram of superior mesenteric artery showing complete cut-off of the first two jejunal branches (upper arrow) and segmental spasms on third and fourth branches (lower arrow).

Figure 2. Angiogram after selective injection of a bolus of 60 mg of papaverine directly into the superior mesenteric artery showing reopening of the first two jejunal arteries (upper arrow) and disappearance of part of the vasospasm on third and fourth jejunal branches (lower arrow). Segmental spasms on distal division branches of second, third, and fourth jejunal arteries are still present.
discontinued after 18 hours when all abdominal signs had completely disappeared. During the following days, blood pressure stabilized at 150/80 mm Hg and was controlled with only methyldopa, 500 mg every six hours orally. Intravenous medication was discontinued and oral nutrition resumed. Fourteen days after the episode of acute abdominal pain, a control angiogram showed a normal superior mesenteric artery and distal bed, with disappearance of all signs of spastic occlusion and beading (Fig 3). The patient was discharged from the hospital 22 days after surgery. Arterial blood pressure was controlled at 150/80 mm Hg with methyldopa 250 mg every six hours, and examination of the abdomen gave normal findings. Fourteen months later, the patient remains well with arterial blood pressure of 150/80 mm Hg taking methyldopa, 500 mg daily.

COMMENT

The clinical findings in this patient were typical of acute mesenteric arteritis complicating coarctation resection. The abdominal syndrome occurred six days after operation and was preceded by paradoxical hypertension. Although not completely understood, the underlying mechanism is probably common to both conditions since the former is always associated with the latter and coincides with the period of maximal plasma renin activity. The angiographic findings in the present case support this hypothesis. The spastic aspect of the mesenteric vessels was partially relieved by administration of papaverine. The beadlike images could have resulted from segmental spasm or inflammatory wall damage. All the changes were completely reversed after 14 days of treatment with vasodilator and antihypertensive drugs. In a recent review, Fox et al have stressed the role of the sympathetic nervous system in the pathogenesis of paradoxical hypertension. The histologic changes in the mesenteric vessels are reported to be similar to those observed in polyarteritis nodosa with intimal rupture and hyperplasia, inflammatory infiltration and necrosis of the elastica and media, and thrombosis. They may represent a more advanced stage of the ischemic process, once irreversible damage and necrosis have occurred.

Because an accurate diagnosis can be established in most patients on the basis of typical clinical findings, angiograms have not been performed in this condition. The more aggressive attitude toward mesenteric ischemia developed in recent years has led to the management of our patient. The angiographic changes were those of severe mesenteric vasoconstriction without thrombotic occlusion, which responded rapidly to vasodilator infusion. The vascular spasm was completely reversible with no permanent damage. The rapid recovery was probably due to early treatment and to the association of antihypertensive drugs and papaverine infusion into the mesenteric artery. The latter has been recommended in the treatment of acute mesenteric ischemia by embolism, and the improvement in survival after embolotomy has been attributed to the relief of the severe vasoconstriction which would persist after removal of the embolus. In the present case, the vascular response to papaverine was shown angiographically, and rapid reversal of mesenteric ischemia and clinical improvement of the patient ensued.

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