Postoperative vasospasm after antegrade mesenteric revascularization: A report of three cases

Bruce L. Gewertz, MD, and Christopher K. Zarins, MD, Chicago, Ill.

Vascular reconstruction for chronic intestinal ischemia can be accomplished by endarterectomy or aortomesenteric bypass. In our practice, antegrade bypasses from the supraceliac aorta to the celiac axis and superior mesenteric artery are currently the most frequently used techniques. Such reconstructions often use multiple or bifurcated large diameter vascular prostheses and have demonstrated excellent long-term patency. Despite these salutory results, we have noted an unusual perioperative response in three of these patients, which is the subject of this report. All three patients underwent uncomplicated elective mesenteric revascularization with grafts (diameter ≥ 6 mm) originating in the supraceliac aorta. Indications for operation included (1) history of postprandial pain, (2) documentation of weight loss, and (3) angiographic evidence of advanced atherosclerotic disease with appropriate collateral development. Episodes of abdominal pain occurred 5 to 20 days after operation when normal food intake was re instituted. In two patients immediate angiograms revealed patent grafts with diffuse mesenteric vasospasm. Treatment with intravenous hyperalimentation and nifedipine for 10 days resulted in complete resolution of symptoms. In the third patient, symptoms were totally relieved by temporary reduction in oral intake and administration of nifedipine. A later angiogram revealed a patent graft. All patients have remained asymptomatic and regained normal weight. This pattern of postrevascularization pain has not been seen in our patients undergoing revascularization with small (i.e., venous) conduits originating in the infrarenal aorta. The cause appears to be a heightened myogenic response of a “protected” vascular bed when suddenly exposed to the high perfusion pressure and blood flow of large caliber antegrade conduits. Prophylaxis with calcium channel blockers and use of smaller diameter grafts (5 mm) may avoid this disturbing syndrome. (J Vasc Surg 1991;14:382-5.)

Vascular reconstruction for chronic intestinal ischemia can be accomplished by endarterectomy or aortomesenteric bypass. In our practice antegrade bypasses from the supraceliac aorta to the celiac axis and superior mesenteric artery are currently the most frequently used techniques. Such reconstructions offer theoretic and practical advantages over grafts originating in the infrarenal aorta. They allow revascularization of both the celiac axis and SMA via multiple or bifurcated large diameter vascular prosthesis and avoid the more advanced atherosclerosis of the distal abdominal aorta. Finally, these grafts have demonstrated excellent long-term patency rates exceeding 90% in some series.

Despite these salutory results, we have noted an unusual perioperative response in some of these patients, which is the subject of this article. The three patients reported herein demonstrated symptoms of continued intestinal ischemia in the postoperative period despite patent reconstructions. Angiograms revealed diffuse mesenteric vasospasm; conservative treatment including bowel rest, vasodilators, and calcium channel blocking agents resulted in complete and permanent remission of symptoms. The possible causes for this syndrome will be discussed.

CASE REPORTS

Case 1. This 41-year-old woman described a 2-year history of postprandial pain, occasionally accompanied by nausea. She reported a weight loss of more than 70 pounds. An extensive diagnostic evaluation including ultrasonography of the biliary tract, upper gastrointestinal endoscopy, and CT scanning was unremarkable. Arteriography demonstrated proximal occlusion of the celiac axis and
superior mesenteric artery with extensive collateralization through the inferior mesenteric artery. She underwent operative revascularization of both the celiac and superior mesenteric artery by means of a Dacron bifurcation graft originating in the supracaeliac aorta. Her immediate postoperative course was notable for persistent tachycardia and the necessity for large volumes of intravenous fluids. The patient also complained of periumbilical pain similar to her ischemic symptoms. She was returned to the operating room immediately and was noted to have edematous but viable intestine. After this procedure she slowly improved. On the seventh postoperative day she underwent angiography that revealed patent grafts. She was discharged on the ninth postoperative day.

Seven days later she was readmitted with crampy, periumbilical pain especially after eating. Angiography was repeated and demonstrated continued patency of her bifurcated grafts, but severe vasospasm was evident in the distribution of the superior mesenteric artery (Fig. 1). Her pain resolved with intraarterial infusion of papaverine. Her treatment included bowel rest and hyperalimentation for 1 week and then slow resumption of oral intake. After 1 week she was discharged from the hospital and has remained asymptomatic to this time.

Case 2. This 45-year-old woman described a 2-year history of abdominal pain. An extensive evaluation including upper gastrointestinal contrast studies and biliary ultrasound examinations revealed no significant gastrointestinal abnormalities. An arteriogram confirmed severe stenosis of the celiac axis with complete obstruction of the superior mesenteric artery and a marked stenosis of the origin of the inferior mesenteric artery. She underwent arterial reconstruction with a bifurcated graft from the supracaeliac aorta to the celiac axis and superior mesenteric artery and patch angioplasty of the origin of the inferior mesenteric artery. Her immediate postoperative course was unremarkable. However, on resuming oral intake 6 days after operation, she complained of severe postprandial pain. An arteriogram was obtained that revealed widely patent anastomoses but multiple areas of vasospasm in the distribution of both the superior mesenteric artery and inferior mesenteric artery (Fig. 2). Her diet was restricted to small feedings, and administration of oral nifedipine (10 mg) 30 minutes before meals was begun. Her symptoms resolved, and the nifedipine was gradually discontinued. She remains asymptomatic 4 years after operation.

Case 3. This 66-year-old woman was admitted with a history of treated peptic ulcer disease and postprandial pain and weight loss. Endoscopy revealed healing of her duodenal ulcer. Arteriography demonstrated a severe stenosis of the origin of the superior mesenteric artery with occlusion of the celiac axis. She underwent bypass of both these vessels by use of a bifurcated graft originating in the supracaeliac aorta. Her immediate postoperative course was unremarkable. However, when feeding was instituted, she complained again of postprandial pain. Her oral intake was restricted, and she was placed on oral nifedipine (10 mg) before meals. Her diet was slowly advanced over 1 week. An arteriogram at that time demonstrated no mesenteric vasospasm with patent grafts.

DISCUSSION

Mesenteric revascularization with prosthetic grafts originating in the supracaeliac aorta is gaining increased acceptance as the preferred technique to bypass obstructive lesions of the celiac axis and superior mesenteric artery. This approach avoids clamping the often diseased infrarenal aorta and
solves the problems of kinking of retrograde grafts.\textsuperscript{4} Hemodynamic results are well documented; flow rates measured at operation often exceed 450 to 500 ml per minute.\textsuperscript{2} It is most important to note that patency of these grafts equals transaortic endarterectomy, which is considered by some a more challenging technique.

Based on these considerations, we use such antegrade grafts in most visceral reconstructions and rely on them especially when mesenteric revascularization is unassociated with replacement of the infrarenal aorta.\textsuperscript{5} The three patients in this report were most unusual because they manifested continued symptoms of mesenteric ischemia in the postoperative period despite patent grafts. In two patients, diffuse mesenteric vasospasm was demonstrated angiographically. With temporary reduction of oral intake, intraarterial papaverine, and calcium channel blockers, these symptoms receded within 1 week. Subsequent angiograms demonstrated complete resolution of vasospasm. In the third patient with an identical presentation, treatment was initiated empirically, and a later angiogram confirmed patent grafts.

All three patients remain asymptomatic at 5, 4, and 3 years of follow-up, respectively.

The origin of this syndrome is unknown, but it may be related to observations of Hollier\textsuperscript{8} and others who have noted occasional patients with pronounced edema of the small bowel after mesenteric revascularization. Indeed, the first patient in this report was noted to have substantial intramural edema but viable bowel when returned to the operating room 12 hours after operation. We postulate that these findings reflect an inability of the intestinal microcirculation to prevent the accumulation of absorbed fluid within the interstitium of the gut.\textsuperscript{7} This may represent failure of capillary "derecruitment" after reinstitution of luxuriant blood flow.\textsuperscript{8} In support of this hypothesis it is well known that the density of perfused capillaries increases in the intestine in response to both feeding (metabolic hyperemia) and local hypoxia (reactive hyperemia).\textsuperscript{9,10} Since one could suppose that the capillary bed distal to a superior mesenteric artery occlusion would be maximally dilated, it is understandable that the sudden restoration of high pressure perfusion would be poorly tolerated. The tendency to local edema could be perioperatively exacerbated by progressive dilution of local plasma oncotic pressure caused by increased absorption of infused crystalloid.

The vasospasm of medium sized vessels could, therefore, be construed as an adaptive response to "protect" the maximally dilated capillary bed. Vasocstriction would be mediated by the myogenic mechanisms intrinsic to vascular smooth muscle. Since these distal vessels would have been chronically exposed to low perfusion pressures, they would be exquisitely sensitive and respond disproportionately to even slight increases in pressure.

A similar response has been well described after surgical repair of coarctation of the aorta.\textsuperscript{11,12} In some series as many as 20% of patients had abdominal pain more than 48 hours after repair. The mechanism may be related to the phenomenon we describe in that the chronically hypoperfused mesenteric circulation is exposed to sudden increases in both perfusion pressure and blood flow. However, several distinct differences are evident. The coarctation syndrome is usually associated with postoperative arterial hypertension and is seen primarily in men.\textsuperscript{13} The precise cause is still unclear. Several investigators have postulated "resetting" abnormalities in baroreceptor function or other changes in the sympathetic nervous system. Increases in circulating catecholamines have been seen in some, but not all, series.\textsuperscript{14}

Such phenomena likely exist to some extent in all
revascularizations of ischemic intestines, although in our experience the response is severe and prolonged in only a small percentage of patients. It is interesting that we have not observed this syndrome in patients with grafts originating on the infrarenal aorta and oriented in a retrograde fashion. Antegrade grafts may elicit a more forceful myogenic response by transmitting a more direct and undamped aortic pressure wave to the arterioles. An alternative explanation is that infrarenal dissections do not disturb the splanchnic innervation, whereas the exposure of the celiac axis undoubtedly injures the celiac neural plexus. Such injury could interfere with postoperative autoregulation of the mesenteric vasculature.

In an effort to avoid this disquieting syndrome, it is now our practice to institute feeding more gradually with smaller amounts of food. Theoretically, this limits mucosal vasodilation during “resetting” of local regulatory mechanisms. Although we have used calcium channel blocking agents to decrease vasospasm of medium sized vessels, the importance of this therapy is unclear. Finally, we currently use smaller graft limbs (5 mm) as suggested by Stoney et al.15 since excessive graft size could contribute to post-revascularization edema.

REFERENCES