

Superficial femoral artery transposition repair for isolated superior mesenteric artery dissection

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Isolated dissection of the superior mesenteric artery is an uncommon event, but many new cases have been reported recently, reflecting the progress of imaging and suggesting that this pathology is not as rare as previously thought. Here we report a case of superior mesenteric artery dissection where we performed, after failure of conservative medical management, an original surgical technique for mesenteric revascularization using a superficial femoral artery transposition. To the best of our knowledge, this is the first report of the use of this technique for complex mesenteric revascularization. (J Vasc Surg 2005;42:788-91.)

Dissection of the superior mesenteric artery (SMA) is an uncommon event but is now reported more often, reflecting the increased use of imaging modalities such as computed tomography angiography (CTA) and magnetic resonance angiography in diagnosing abdominal pathology.¹⁻³ Two general therapeutic strategies can be indicated: conservative or medical management, or surgical revascularization.^{1,4} Major improvements have been made in surgical outcomes, with decreased mortality. Thus, decisions regarding medical management vs surgical intervention are complex, and there is insufficient experience to prove the superiority of one approach over the other. Failure of surgical management carries a high risk of death, thus emphasizing that mesenteric revascularization can be a hazardous procedure. We report an original technique of revascularization of the SMA by using a superficial femoral artery (SFA) transposition.

CASE REPORT

In March 2004, a 53-year-old woman presented in the emergency department with acute abdominal pain suggestive of mesenteric ischemia. She did not have any cardiovascular risk factors but had had recurrent abdominal pain for a few weeks. A scout abdominal film showed multiple air-fluid levels. A CT scan demonstrated Balthazar stage B pancreatitis, confirmed by increased pancreatic enzymes (amylase, 544 U/L; lipase, 512 U/L). Contrast CT suggested a false lumen of the SMA and abnormal thickness of its wall (Fig 1). Limited infarctions of the left kidney were also seen. Others studies

excluded biliary diseases, viruses, tumors, or alcohol as specific causes of the pancreatitis.

Selective angiography using the Seldinger technique with 4F pigtail catheters was performed. This investigation found a long stenosis of the SMA, absence of the inferior duodeno-pancreatic arcade, small bowel malperfusion (Fig 2, A), complete obstruction of a left inferior polar renal artery, a right renal artery stenosis with poststenotic dilatation, and a dissection beginning at the poststenotic dilatation of the right renal artery (Fig 2, B). Lack of strong clinical and angiographic evidence of major mesenteric ischemia led to conservative medical management as the initial approach, including close observation in the intensive care unit, analgesic treatment, heparin anticoagulation, and parenteral nutrition. Calcium inhibitor treatment was begun after hypertension developed during the patient's hospital stay.

Three weeks later, because of increasing abdominal pain and pancreatic enzymes levels (amylase, 1911 U/L; lipase, 4188 U/L), we proceeded with surgical mesen-

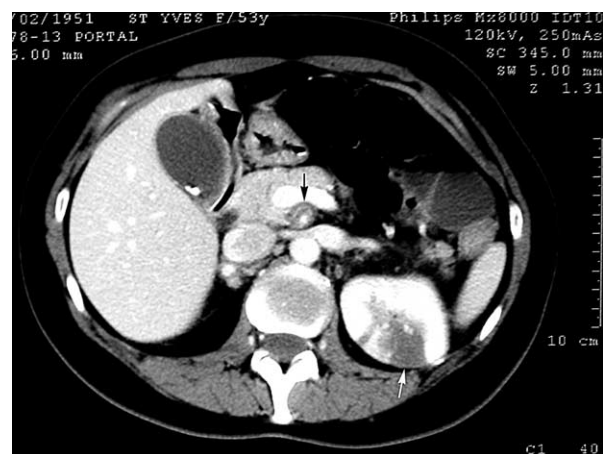


Fig 1. Contrast computed tomography (CT) suggests the diagnosis of isolated superior mesenteric artery (SMA) dissection and shows the enlarged SMA wall (black arrow). Partial infarction of the left kidney is also seen (white arrow).

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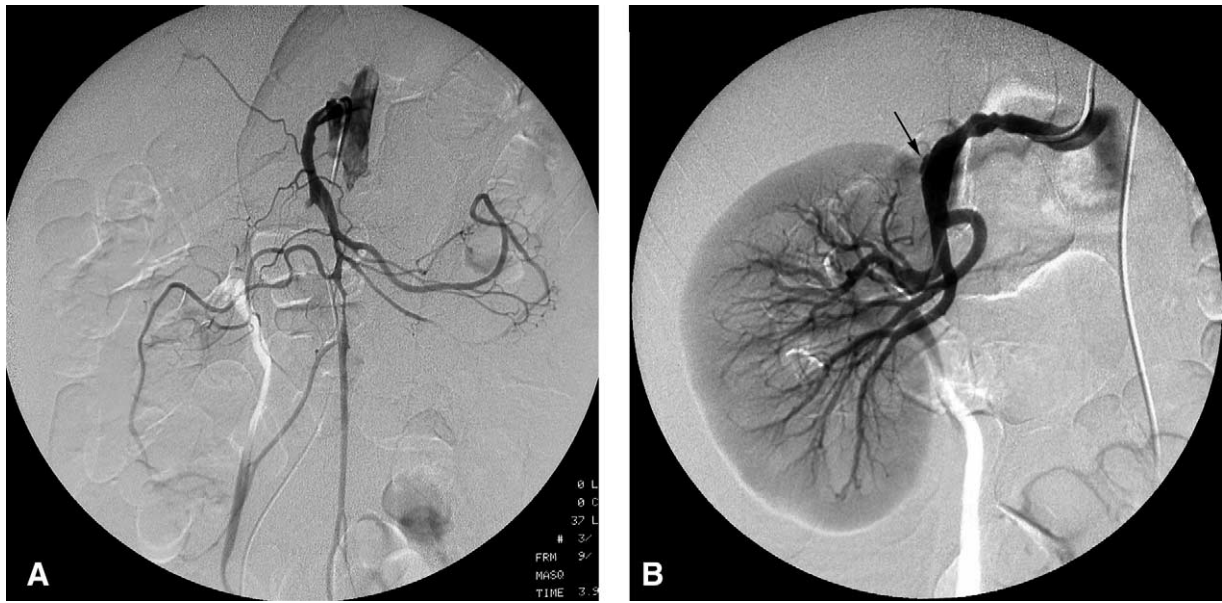


Fig 2. A. A preoperative selective angiogram of the superior mesenteric artery (SMA) shows a long stenosis due to the isolated dissection of the SMA, absence of the inferior duodeno-pancreatic arcade, and small bowel malperfusion. **B.** Preoperative selective renal angiogram shows a right renal artery stenosis with poststenotic dilatation and a dissection beginning at the poststenotic dilatation of the right renal artery (*arrow*).



Fig 3. Three-month postoperative angiogram demonstrates patency of the anatomic reconstruction of the superior mesenteric artery (SMA) by using the superficial femoral artery. *Long arrows* show the proximal and distal end-to-end anastomoses of the arterial graft. *Short arrows* show good distal perfusion of the small bowel. The significant tapering of the mid portion of the graft is probably a spasm or an artifact.

teric revascularization. Through a median laparotomy, intramesenteric exposure of the SMA allowed visualization of the diseased portion of the artery. No saphenous vein was

available because of previous bilateral stripping 20 years ago, and upper limb veins did not appear usable after a preoperative ultrasound evaluation. Thus, after heparin was given, the right SFA was dissected free and replaced by a polytetrafluoroethylene (PTFE) tube graft. The SMA was clamped at its first centimeter and transversely cut. All jejunal and ileal branches previously controlled were carefully clamped and cut. The trunk of the SMA was completely removed and sent for pathologic examination. An intimal flap separating the true and false lumen confirmed the diagnosis of dissection.

End-to-end anastomoses were performed between (1) the SMA ostium and the proximal part of the SFA, and (2) the last SMA bifurcation and the distal part of the SFA (giving the ileo-caeco-appendicular artery). Four other side-to-end anastomoses were performed under binocular loupes between the jejunal or ileal arteries and the SFA. After declamping, the small intestine appeared well perfused. The pathologic segment of the right renal artery was also resected and replaced by the most distal segment of the SFA. We completed the procedure with a cholecystectomy and percutaneous jejunostomy.

The patient was discharged 5 weeks later on antiplatelet therapy. A 3-month postoperative selective angiography demonstrated patency of the SFA transposed in the SMA position, occlusion of two ileal anastomoses, and good general vascularization of the bowel (Fig 3). Six months postoperatively, the patient was well and remained asymptomatic. Pathologic examination confirmed dissection along an 11-cm length of the SMA (Fig 4) and also revealed medial fibrodysplasia.

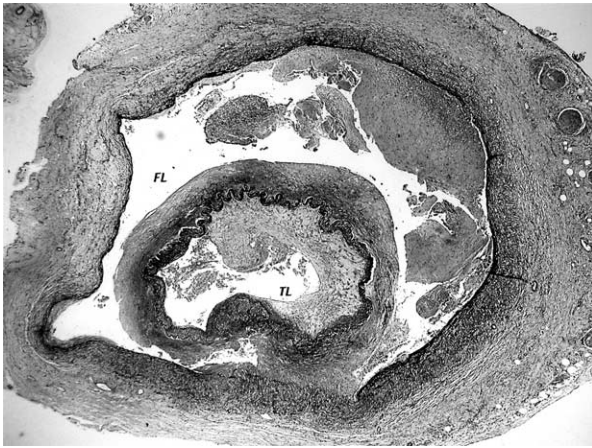


Fig 4. Pathology image (Gordon-Sweet's silver stain, $\times 20$ original magnification) demonstrates the fully dissected superior mesenteric artery between the media and the adventitia. TL, True lumen; FL, false lumen.

DISCUSSION

As a result of the advancements in imaging modalities, especially in CTA, 15 isolated SMA dissections have been reported over the last 2 years, demonstrating that isolated SMA dissection is probably not as rare as it was once believed.¹⁻⁷ Diagnosis remains difficult but must be considered when there is acute abdominal pain with or without vomiting and diarrhea, and imaging shows an enlarged SMA diameter, abnormal SMA wall thickness, and SMA intimal flap with a false lumen.¹ Epigastric pain is typically caused by the dissection itself, the mesenteric infarction, or both. Here, the pancreatitis added another unusual associated cause of pain. This may have partially been caused by the occlusion of the duodeno-pancreatic arcade, which to the best of our knowledge, has not been reported in such a circumstance.

In recent years, a CT scan has been the main investigation to confirm the diagnosis.^{8,9} We emphasize the major importance of preoperative computed selective angiography, however, because this shows the length of the dissection and the jejunal/ileal arteries free of dissection that are usable for elective revascularization.

Conservative treatment with close surveillance and anticoagulation has been successfully used in several reported cases with good outcomes. Surgical revascularization was associated with a poor prognosis years ago,^{10,11} but improved mortality rates have been observed during the last two decades. Revascularization should be considered in the case of increasing size of the aneurysmal dilatation, thrombosis of the true lumen of the SMA, or persistent symptoms despite anticoagulation.¹²

Many conventional surgical techniques such as vein or prosthetic graft bypass,^{4,13} resection of intimal flap, and endovascular stenting have been described to treat SMA dissection.^{5,7,14} We chose to replace the SMA with SFA because of the lack of saphenous vein. We did not

consider the use of a prosthetic graft because of concern over long-term patency.¹⁵ However, the superficial femoral vein could have been considered as an alternative autogenous conduit for this arterial reconstruction.

To the best of our knowledge, this is the first report of SFA transposition in this indication. The SFA appeared to have the optimal diameter for the proximal end-to-end anastomosis at the SMA ostium. In isolated SMA dissection, the ostium and the first few centimeters are usually normal, allowing creation of the proximal anastomosis under optimal conditions. The hypothesis is that the dissection appears at the transition between the fixed and mobile part of the artery on the inferior edge of the pancreas.¹⁶ The arterial graft was also long enough to perform an anatomic reconstruction with multiple side-to-end jejunal and ileal artery anastomoses. The SFA was easily replaced in the lower limb by a tube graft, thus decreasing the risk of possible graft occlusion in this location.

Arteriosclerosis, fibromuscular dysplasia, and diseases of the elastic tissue (Marfan disease, Ehlers-Danlos disease) are possible causes for isolated SMA dissection when a cause is found.¹⁷ Renal arteries are the first sites for arterial fibrodysplasia and can be associated with other affected arteries.¹⁸ Here, the pathologic examination confirmed the association of the SMA and renal artery fibrodysplasia.

This case illustrates a novel treatment of SMA dissection with a well-accepted technique previously described for use in other arterial locations^{19,20}; this is important, as SMA dissection is being detected and diagnosed more frequently with the progression of better imaging techniques.

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