

Open repair of symptomatic superior mesenteric artery dissection

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Keywords: Superior mesenteric artery; Dissection; Visceral dissection; Superior mesenteric artery dissection; Superior mesenteric artery stenosis; Chronic mesenteric ischemia; Superior mesenteric artery endarterectomy; SMA

We present the case of a 55-year-old man with abdominal pain and known, chronic superior mesenteric artery (SMA) dissection. The patient had initially presented 3 years before the present evaluation with acute abdominal pain and was diagnosed with an SMA dissection that was managed conservatively elsewhere, with oral anticoagulation that was eventually transitioned to dual antiplatelet therapy. Subsequently, he had had intermittent episodes of abdominal pain and additional visceral dissections seen radiographically. These were not lifestyle limiting, and no intervention was pursued.

On presentation to our clinic, the patient had another episode of acute, severe epigastric abdominal pain, followed by worsening postprandial pain and a 50-lb weight loss. Extensive evaluation of other gastroenterologic, cardiac, or rheumatologic conditions was performed elsewhere and repeated at our institution with no additional positive findings. Computed tomography angiography of the abdomen and pelvis was performed and revealed celiac axis dissection and subtle aneurysmal degeneration to 16 mm, which was stable. The SMA dissection started in the mid-portion of the SMA, with a partially thrombosed false lumen and high-grade stenosis of the true lumen, especially at the distal end point of the dissection. Duplex ultrasound did not demonstrate velocity elevation consistent with stenosis

in the proximal SMA. However, the distal extent of dissection was poorly visualized. Initially, given the uncertainty of the findings of the diagnostic tests, we monitored the patient clinically for 6 months. However, given the extensive workup and worsening symptoms, the multi-specialty consensus was a diagnosis of chronic mesenteric ischemia, for which he was offered surgical reconstruction.¹ The patient provided written informed consent for the report of his case details and imaging studies.

With the patient under general anesthesia, a midline laparotomy was performed ([Supplementary Video](#), online only). After placement of a self-retaining retractor, the SMA was identified at the base of the mesentery. With the bowel retracted caudally, the mesentery was opened and the SMA dissected free throughout its length. The mesentery surrounding the dissected SMA was inflamed and thickened, requiring cautious dissection. The distal SMA branches were individually identified, dissected, and controlled with silastic loops. After control of the branches, attention was turned to the main trunk of the SMA, which was clamped proximally to the dissection. An arteriotomy was performed in standard fashion with a no. 11 blade and extended with Potts scissors. This revealed the false lumen with several areas of chronic thrombus. The high-grade stenosis in the distal aspect of the dissection was identified, and the SMA was opened beyond this point, demonstrating compression of the true lumen. The true lumen was then evaluated using a 3-mm coronary dilator. The septum was fenestrated and resected with Potts scissors. Given the poor backbleeding from the distal SMA, Fogarty thrombectomy was performed, revealing additional chronic thrombus, resulting in satisfactory backbleeding. The thrombogenic-appearing aneurysmal segment of the SMA wall was resected. Intimal tacking sutures were placed, and a bovine pericardial patch was then fashioned. Anastomosis of the patch was performed with running 5-0 polypropylene suture. After completion of the patch, flow was restored, with strong signals and a palpable pulse throughout the mesentery. Intraoperative completion duplex ultrasound showed no

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Author conflict of interest: none.

Presented at the 2023 Society for Vascular Surgery Vascular Annual Meeting, National Harbor, MD, June 14-17, 2023.

Additional material for this article may be found online at www.jvscit.org.

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The editors and reviewers of this article have no relevant financial relationships to disclose per the Journal policy that requires reviewers to decline review of any manuscript for which they may have a conflict of interest.

J Vasc Surg Cases Innov Tech 2023;9:101255
2468-4287

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<https://doi.org/10.1016/j.jvscit.2023.101255>

technical defects or residual stenosis throughout the distal branches.

The patient did well postoperatively without major adverse events. He was transferred from the intensive care unit on postoperative day 3 and dismissed to home on postoperative day 7, tolerating a general diet without postprandial pain. An early postoperative computed tomography angiogram demonstrated a widely patent SMA without residual dissection or stenosis.

At 4 months of follow-up, the patient continued to do well without signs of mesenteric ischemia, with resolution of his abdominal pain, and tolerance of a normal oral diet. His weight has stabilized. The 4-month computed tomography angiogram demonstrated a

widely patent SMA from the origin to the terminal branches without evidence of stenosis or dissection.

In conclusion, we present a case of symptomatic, chronic mesenteric ischemia resulting from chronic SMA dissection. This was managed by open reconstruction of the SMA with resection of the dissection flap and patch angioplasty with excellent clinical results.

REFERENCES

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Submitted Apr 15, 2023; accepted Jun 1, 2023.