CASE REPORT

Isolated Spontaneous Dissection of the Superior Mesenteric Artery: Successful Primary Therapy with a Nitinol Stent

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ABSTRACT

Isolated spontaneous dissection of the superior mesenteric artery is rare. Conventional treatment involves surgical or conservative management, and is increasingly supported by radiological interventions. We describe herein a patient with this condition, successfully treated by primary percutaneous endovascular nitinol stent placement after an acute isolated spontaneous superior mesenteric artery dissection, who has been followed up for 47 months. We also review the relevant literature and describe the increasing use of endovascular stenting, which is becoming the preferred treatment for this type of condition.

Key Words: Abdominal pain; Aneurysm, dissecting; Mesenteric artery, superior; Stents

INTRODUCTION

Isolated dissection of the superior mesenteric artery (SMA) is rare. Different treatment strategies have been described, among which surgery is often advocated. There is, however, an increasing trend towards less invasive management, including conservative treatment with anticoagulation or endovascular therapy. We describe a patient presenting with abdominal pain due to isolated spontaneous SMA dissection successfully treated by using primary and acute percutaneous endovascular nitinol stenting soon after presentation; this patient has been followed up for 47 months.

CASE REPORT

A 48-year-old Chinese man with a history of mild hypertension and treated with losartan 25 mg daily, presented with acute-onset, severe and progressively increasing epigastric pain for two hours. His vital signs were stable apart from a slightly increased body temperature. Abdominal examination revealed non-specific epigastric tenderness but no guarding or rebound tenderness. Routine laboratory tests (full blood count, liver function, and serum amylase level) were normal. Initially he was suspected to have acute pancreatitis or biliary colic, for which a contrast-enhanced computed
tomography (CECT) was performed. This showed a linear hypodensity in the proximal SMA originating near the ostium with differential luminal enhancement in the distal SMA and several jejunal branches, and raising the possibility of SMA thrombosis or dissection (Figure 1). The rest of the arteries including the aorta were normal and there were no CT features of bowel ischaemia or peritonitis.

An urgent catheter angiogram was performed via the right common femoral artery (CFA), using a 5F Sidewinder catheter (Terumo, Tokyo, Japan) so as to obtain a selective superior mesenteric arteriogram. This demonstrated a linear filling defect within 1 cm from the SMA’s origin, consistent with an intimal dissection flap, giving a double-lumen appearance (Figure 2). The dissection extended to the origins of several jejunal branches. The acute presentation with a slightly increased body temperature leads to concern of impending bowel ischaemia and urgent treatment was felt necessary to prevent progression of the dissection. To avoid open surgery, it was decided to treat the dissection by primary stenting.

The Sidewinder catheter was exchanged for a 7F renal double curve guiding catheter (Cordis, Johnson and

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**Figure 1.** Axial computed tomographic images in the portal venous phase: (a) a linear hypodensity in the proximal superior mesenteric artery (SMA) represents the dissection flap originating within 1 cm of the ostium, and (b) the distal SMA and jejunal branches are enlarged with differential luminal enhancement. The peripheral crescentric hypodensity represents the thrombosed false lumen. There is also fat stranding around the vessels.

**Figure 2.** Selective superior mesenteric arteriogram: (a) a linear filling defect in the proximal superior mesenteric artery (SMA) represents the dissection flap, giving the double-lumen appearance, and (b) with further advancement of the catheter into the true lumen, there is obvious differential enhancement between the true and false lumens.
Johnson, Florida, USA) and a 0.014" guidewire (Cordis, Johnson and Johnson, Florida, USA) was advanced via the true lumen across the length of the dissected SMA. A Precise RX nitinol stent (9-mm diameter x 30-mm length; Cordis, Johnson and Johnson, Florida, USA) was then deployed across the dissected segment with its proximal end just distal to the SMA origin. The post-stenting arteriogram showed satisfactory stent positioning and expansion, and the opacification of several jejunal branches which had not been seen on the previous arteriogram (Figure 3). Balloon angioplasty of the branches was not felt necessary. There was no evidence of a distal embolus. However, the patient subsequently developed a puncture-site pseudoaneurysm of the right CFA, which resolved following a thrombin injection.

The patient’s abdominal pain resolved and a computed tomographic angiogram (CTA) obtained four days later confirmed good SMA patency through the fully expanded stent. The patient was discharged with antiplatelet therapy (aspirin 100 mg daily for 16 months and clopidogrel 75 mg daily that he was still taking) and his anti-hypertensive therapy was optimised. At a follow-up of 47 months after stenting, the patient was symptom-free. Moreover, the CTA obtained at 3 and 9 months (Figure 4), and Doppler ultrasonography performed after 16, 24, 30, 36 and 42 months confirmed persistent patency of the SMA and resolution of the false lumen.

**DISCUSSION**

Dissection of the SMA is rare, especially without concurrent dissection of the abdominal aorta. It was reported in 0.06% of instances in a series of 6,666 autopsies. It was first reported by Bauersfeld in 1947, and since then, more than 100 cases have been described, with the majority in the last decade. Review of the literature reveals that this entity predominantly affects males (80-85%), most of whom were older than 50 years. Postulated pathogeneses include cystic medial necrosis, fibromuscular dysplasia, atherosclerosis, hypertension, abdominal trauma, connective tissue disorder, and those associated with dissecting abdominal aorta. No definite cause is usually found, however.

Anatomically, the SMA can be divided into a proximal fixed retropancreatic portion and a distal mobile portion which pivots with bowel movements at the inferior edge of the pancreas. Solis et al hypothesised that this transition (approximately 2-6 cm from the SMA origin) is prone to shearing forces like at the ligamentum arteriosum in aortic dissection. Yun et al hypothesised that the convex-curved run of the SMA might provide an additional mechanical factor that initiates SMA dissection. There are a few postulated courses of dissection: blind-ending, progression with re-entry into the lumen, or progression with rupture through

![Figure 3. Post-stenting arteriograms in (a) coronal and (b) sagittal views showing satisfactory expansion of the 9 x 30 mm Precise RX Cordis stent within the superior mesenteric artery (SMA) true lumen. There is recanalisation of multiple branches arising from the SMA, not seen in the pre-stenting angiogram.](image-url)
the adventitia. The false lumen may be thrombosed or patent. Patients may be asymptomatic or more typically present with acute abdominal pain caused by the dissection itself or its complications (bowel ischaemia and arterial rupture). Hence early diagnosis is essential in improving survival and requires a high index of suspicion. There is a poor correlation between the duration and severity of clinical symptoms and the extent of mesenteric ischaemia.

SMA dissection is usually diagnosed on CECT during the course of work-up for abdominal pain. Although CT is reliable in evaluating the disease, catheter angiography offers the possibility of endovascular repair. Doppler may provide additional information on the haemodynamics within the SMA, showing the presence or absence of flow within the false lumen.

To date, there is no consensus on the best mode of treatment which may include surgery, radiological interventions, or a conservative approach with or without anti-coagulant therapy. Anti-coagulation is considered valid for this disease due to the risk of thrombosis in the SMA because of its small calibre and relatively slower blood flow as compared to the aorta. Currently, however, there is no established theory on the drugs to use or treatment duration. Some authors proposed a conservative approach when there were no clinical or imaging signs to suggest bowel ischaemia or SMA rupture. A conservative approach, however, does not prevent disease progression; one-third of such patients will require surgery or stenting subsequently.

Endovascular percutaneous stent placement is a safe and minimally invasive alternative to aggressive surgery or uncertain conservative expectant treatment of isolated SMA dissection. The basic approach is to place the stent over the entry site of the dissection to obliterate the false lumen by pressure and promoting flow through the true lumen. If this fails and the false lumen persists, additional treatment of the re-entry site or involved distal branches may become necessary. Wu et al performed additional balloon angioplasty for involved side branches, so as to improve distal flow. This was not necessary in our case as there was improved and adequate distal run-off upon stenting the entry site.

Leung et al reported the first case of stenting in 2000, and since then, there have been 17 cases reported in the English literature. Among these, only six patients were treated primarily (acutely) on the same
Table. Reported cases of spontaneous isolated superior mesenteric artery (SMA) dissection treated by percutaneous endovascular stent placement.

<table>
<thead>
<tr>
<th>Authors, year (reference)</th>
<th>Age (years)/sex</th>
<th>Primary treatment</th>
<th>Time interval to stenting; stent type</th>
<th>Results / follow-up</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 Leung et al, 2000 (12)</td>
<td>67 / M</td>
<td>Fluids and analgesia</td>
<td>Few days later; self-expandable 8 x 68 mm (Wallstent, Schneider Bulach, Switzerland)</td>
<td>Well at 6 months</td>
</tr>
<tr>
<td>2 Yoon et al, 2003 (13)</td>
<td>52 / M</td>
<td>Anti-coagulation</td>
<td>1 Month later; balloon-expandable 6 x 15 mm and 7 x 15 mm (Corinthian, Cordis, Europa N. V The Netherlands)</td>
<td>Well at 12 months</td>
</tr>
<tr>
<td>3 Froment et al, 2004 (7)</td>
<td>58 / M</td>
<td>Stenting</td>
<td>Same day; self-expandable 8 x 20 mm (Wallstent, Boston Scientific, Galway, Ireland) and one balloon-expandable 7 x 28 mm (Jo-WaveMax, Jomed AG, Binningen, Switzerland)</td>
<td>Well at 18 months</td>
</tr>
<tr>
<td>4 Kim et al, 2004 (14)</td>
<td>48 / F</td>
<td>Anti-coagulation</td>
<td>Same day; self-expandable 8 x 70 mm (Wallstent, Boston Scientific, Watertown, MA, USA)</td>
<td>Proximal stent shortening and restenosis of SMA origin at 6 months without symptoms</td>
</tr>
<tr>
<td>5 Kim et al, 2004 (14)</td>
<td>54 / M</td>
<td>Stenting</td>
<td>Same day; self-expandable 8 x 60 mm (Wallstent, Boston Scientific, Watertown, MA, USA)</td>
<td>Progressive dilatation of pseudoaneurysm due to proximal stent shortening at 4 months</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Restenting</td>
<td>4 Months later; self-expandable 10 x 50 mm (SMART, Cordis, Miami, FL, USA)</td>
<td>No symptoms 2 months after restenting</td>
</tr>
<tr>
<td>6 Miyamoto et al, 2005 (3)</td>
<td>59 / M</td>
<td>Anti-coagulation</td>
<td>24 Hours later; self-expandable 10 x 60 mm and 10 x 40 mm (Luminex, Bard, Tempe, AZ, USA)</td>
<td>Well at 3 months</td>
</tr>
<tr>
<td>7 Tsai et al, 2005 (15)</td>
<td>53 / M</td>
<td>Stenting</td>
<td>Same day; balloon-expandable 5 x 18 mm (Jostent, GraftMaster, Jomed, Rangendingen, Germany) and 4 x 38 mm (Jostent, Peripheral, Jomed, Rangendingen, Germany) as the first stent slipped into the pseudoaneurysm sac. Additional urokinase infusion as complicated by thrombus formation in the SMA branches</td>
<td>Well at 22 months</td>
</tr>
<tr>
<td>8 Ozaki et al, 2006 (16)</td>
<td>64 / M</td>
<td>Observation</td>
<td>27 Months later; self-expandable 8 x 20 mm (Wallstent, Boston Scientific, Watertown, MA, USA) and detachable coils to embolise the false lumen</td>
<td>Well at 9 months</td>
</tr>
<tr>
<td>9 Langner et al, 2007 (11)</td>
<td>42 / M</td>
<td>Anti-coagulation</td>
<td>&gt;24 Hours later; balloon-expandable 7 x 15 mm (Palmaz Genesis, Cordis*)</td>
<td>Well at 10 days</td>
</tr>
<tr>
<td>10 Sakamoto et al, 2007 (6)</td>
<td>51 / M</td>
<td>Anti-coagulation</td>
<td>22 Months later; balloon-expandable (Palmaz*) and coil packing to embolise the pseudo-aneurysm</td>
<td>Well at 38 months</td>
</tr>
<tr>
<td>11 Casella et al, 2008 (8)</td>
<td>51 / M</td>
<td>Fluids and analgesia</td>
<td>4 Days later; self-expandable 8 x 36 mm (Wallstent, Boston Scientific, Natick, MA, USA)</td>
<td>Well at 31 months</td>
</tr>
<tr>
<td>12 Wu et al, 2009 (9)</td>
<td>56 / M</td>
<td>Anti-coagulation</td>
<td>6 Days later; self-expandable 10 x 60 mm and 10 x 40 mm (Aurora, Medtronic, Santa Rosa, CA, USA). Additional balloon angioplasty to treat involved side branches</td>
<td>Well at 9 months</td>
</tr>
<tr>
<td>13 Wu et al, 2009 (9)</td>
<td>66 / M</td>
<td>Anti-coagulation</td>
<td>4 Days later; self-expandable 9 x 60 mm (Aurora, Medtronic, Santa Rosa, CA, USA)</td>
<td>Well at 7 months</td>
</tr>
<tr>
<td>14 Baldi et al, 2009 (17)</td>
<td>50 / M</td>
<td>Stenting</td>
<td>Same day; self-expandable 7 x 40 mm and 8 x 40 mm (Precise Smart, Cordis, Miami, FL, USA)</td>
<td>Well at 12 months</td>
</tr>
<tr>
<td>15 Gobble et al, 2009 (10)</td>
<td>48 / M</td>
<td>Stenting</td>
<td>Same day; self-expandable 7 x 20 mm (Xceed, Abbott Laboratories*)</td>
<td>Well at 12 months</td>
</tr>
<tr>
<td>16 Gobble et al, 2009 (10)</td>
<td>78 / F</td>
<td>Stenting</td>
<td>Same day; balloon-expandable 6 x 18 mm (HercLink*)</td>
<td>Well at 11 months</td>
</tr>
<tr>
<td>17 Yun et al, 2009 (5)</td>
<td>NA*</td>
<td>Stenting</td>
<td>Self-expandable 4 x 40 mm nitinol stent*</td>
<td>Stent occluded at 17 months</td>
</tr>
<tr>
<td>18 Present case</td>
<td>48 / M</td>
<td>Stenting</td>
<td>Same day; self-expandable 9 x 30 mm (Precise RX, Cordis, FL, USA)</td>
<td>Well at 47 months</td>
</tr>
</tbody>
</table>

* Detailed information not provided in the article
Different stents have been used to treat SMA dissections. However, there is no consensus as to which stent is best for this purpose, as experience is limited. To minimize the tension on the already weakened arterial wall, Casella et al.8 preferred a self-expanding stent which has a weaker radial force. Of the 10 reported cases, six were treated using the Wallstent, a self-expandable stent made of elgiloy.7,8,12,14,16 However, the Wallstent foreshortens during deployment and is therefore more difficult to deploy accurately. In two of these six cases, delayed proximal shortening of the Wallstent endoprostheses occurred, one of which required restenting using a nitinol stent.14 In the six other reports where nitinol stents were utilised,3,5,10,12,17 accurate stent deployment was achieved with good short-term outcomes. We used a nitinol stent, which shortens less on expansion than the Wallstent, and is more flexible and therefore easier to deploy precisely. It is also uncovered to ensure patency of the side branches.

We believe early stent implantation provides better outcomes in addition to immediate relief of symptoms, without the risks associated with surgery and general anaesthesia. The long-term results, however, are still unknown and need to be evaluated. In chronic mesenteric ischaemia due to atherosclerotic disease, surgery has been shown to be superior to primary SMA stenting that often required re-intervention to maintain patency.19 For a stented SMA in chronic mesenteric ischaemia, Sarac et al.18 reported a primary patency rate of 65% at 12 months. However, these results cannot be extrapolated to stenting in isolated SMA dissection as the pathophysiology differs.8 Follow-up with CTA or Doppler is necessary for all cases, irrespective of the treatment method for early recognition of disease progression or complications related to stenting.

In conclusion, primary percutaneous endovascular stent placement is an attractive option for treating isolated SMA dissections in the absence of bowel ischaemia or peritonitis. Its indications may be extended to patients previously considered for surgery. For instance, it could be an adjunct to surgery in the presence of bowel ischaemia. Post-stenting continuous surveillance is crucial to ensure disease stability.

REFERENCES