Open aortic septectomy for complicated type B aortic dissection

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ABSTRACT

Malperfusion is a complication of acute aortic dissection associated with substantially increased morbidity and mortality. Although endovascular treatment of the dissection with a stent graft to cover the intimal tear and reexpand the true lumen will often be sufficient to treat distal malperfusion, persistent or delayed malperfusion will necessitate additional interventions. Endovascular strategies to increase true lumen expansion include bare metal dissection stent placement and percutaneous fenestration. However, for patients with anatomy not amenable to an endovascular approach, alternative techniques are required. We describe two cases of complicated acute aortic dissection due to partial false lumen thrombosis treated with open aortic septectomy. Although an uncommon procedure, open septectomy can be useful for patients with malperfusion syndromes without appropriate endovascular options. (J Vasc Surg Cases Innov Tech 2023;9:1-5.)

Keywords: Aorta; Aortic septectomy; Dissection; Humans; Malperfusion; Morbidity; Syndrome; Thrombosis

Acute aortic dissection can be complicated by static or dynamic obstruction of multiple vascular beds, including the brain, spinal cord, abdominal viscera, and lower extremities. Up to 33% of acute aortic dissections are associated with a malperfusion syndrome, which results in increased mortality compared with uncomplicated dissections.[1] A proximal aortic intervention is sufficient to treat most cases of malperfusion syndrome.[2,3] However, delayed symptoms can still occur. Fenestration of the dissection septum may decompress the false lumen and improve flow back into the true lumen.[4] Although often performed percutaneously, open septectomy is an option for patients with anatomy unfavorable for an endovascular approach.[5] In the present report, we describe two patients with delayed malperfusion symptoms resulting from false lumen thrombosis who were treated with open aortic septectomy. Both patients provided written informed consent for the report of their case details and imaging studies.

CASE REPORT

Patient 1

A 28-year-old man was transferred from a referring facility after presenting with acute onset chest pain that occurred during weightlifting. Computed tomography (CT) demonstrated acute DeBakey type I aortic dissection (zone 0-10) with evidence of right lower extremity malperfusion (Fig 1, *A and B*) for which he underwent emergent repair with ascending aorta and transverse arch replacement by the cardiac surgery team. He also underwent left-to-right femoro-femoral bypass and prophylactic four-compartment right calf fasciotomy for persistent right lower extremity malperfusion. Three weeks later, he developed acute worsening bilateral leg pain with loss of previously palpable pedal pulses and reduction of his ankle brachial index (ABI) from 1.0 to 0.4. Repeat CT angiography demonstrated thrombosis and expansion of the false lumen at the aortic bifurcation with near occlusion of the true lumen (Fig 1, *C and D*).

Patient 2

A 62-year-old man with a history of peripheral artery disease and bilateral intermittent leg claudication was transferred from an referring facility after presenting with acute chest and back pain. CT angiography demonstrated acute DeBakey Type IIIb aortic dissection (zone 3-10) with occlusion of the left external iliac artery and reconstitution of the common femoral artery (Fig 2, *A and B*). Despite the iliac occlusion, he did not have worsening of the chronic ischemic leg symptoms. His ABI was 0.6 and 0.5 on the right and left, respectively. He was initially treated medically with anti-impulse control. On hospital day 4, he developed new right leg pain and bilateral lower extremity motor and sensory deficits that were concerning for spinal cord malperfusion. CT angiography demonstrated thrombosis and expansion of the infrarenal aortic false lumen with severe compression and malperfusion of the true lumen (Fig 2, *C and D*).

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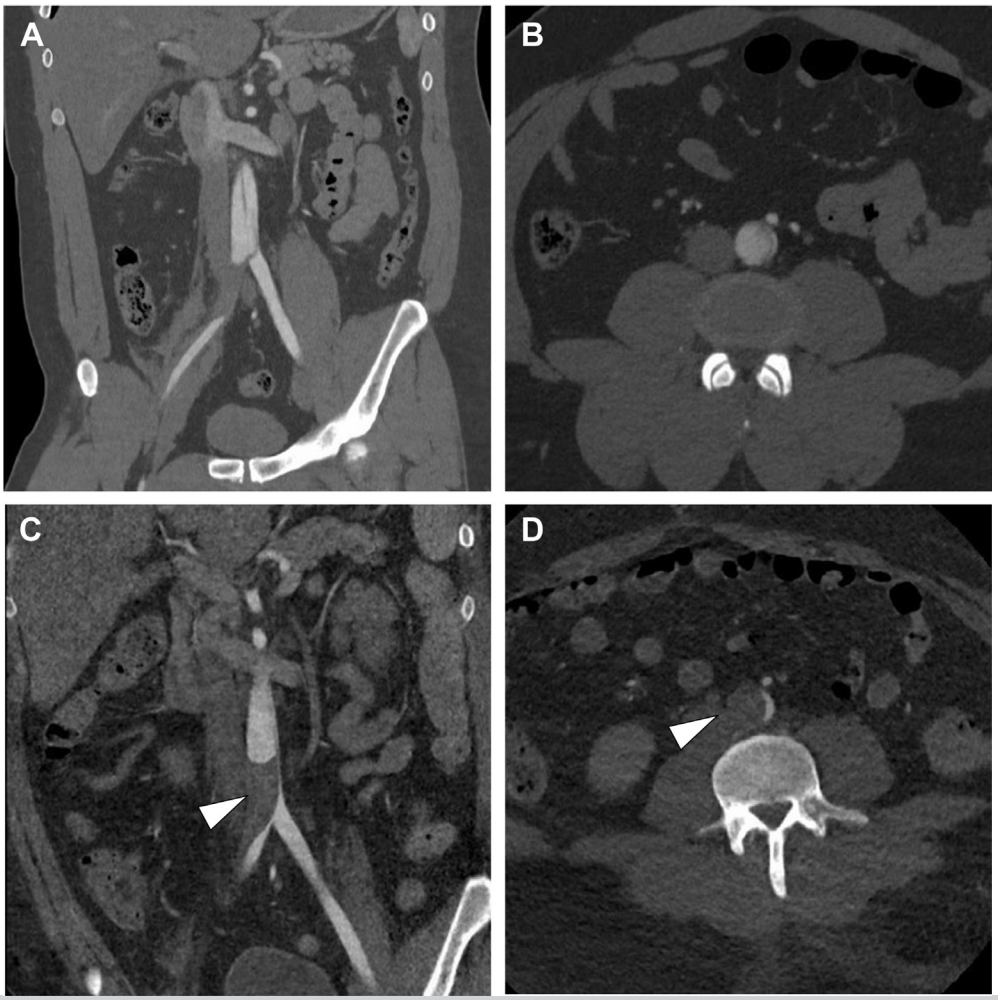

Fig 1. Computed tomography (CT) of abdomen and pelvis of patient 1 in coronal (**A**) and axial (**B**) planes at presentation. Similar locations on the repeat CT scan at the time of open aortic septectomy are shown in the coronal (**C**) and axial (**D**) planes. *White arrowheads* denote acute false lumen thrombosis and true lumen obstruction.

Operative procedure

For both patients, endovascular treatment with either percutaneous fenestration or a bare metal dissection stent was not possible due to false lumen thrombosis. Thus, we offered open aortic septectomy and false lumen thrombectomy to both patients.

Patient 1. The operation was performed through a retroperitoneal aortic exposure with patient 1 in a partial right lateral decubitus position. Proximal control of the infrarenal aorta and distal control of the bilateral common iliac arteries using Fogarty hydragrip clamps were obtained after systemic heparinization. A longitudinal aortotomy was made and extended onto the dissected right common iliac artery. The true lumen was nearly occluded at the level of the aortic bifurcation (Fig 3, *A*). Thrombus was extracted from the false lumen, and septectomy was performed from the proximal clamp down to the proximal right iliac artery. The inferior mesenteric artery was preserved. The residual septum was fenestrated proximally and tacked down with interrupted Prolene sutures distally. The aortotomy was closed using a 10-cm-long bovine pericardial patch to avoid narrowing the aortic diameter. After reperfusion, multiphasic pedal Doppler signals were present, which persisted in the right leg with temporary left iliac occlusion, indicating the right leg perfusion was no longer dependent on the femoro-femoral bypass.

Patient 2. Patient 2 first underwent lumbar spinal drain placement in accordance with our institutional spinal cord ischemia protocol. The operation was performed through a retroperitoneal aortic exposure with the patient in a partial right lateral decubitus position. After systemic heparinization, proximal control was obtained of the suprarenal aorta just distal to the superior mesenteric artery, and distal control was obtained at the distal common iliac arteries. Again, atraumatic occlusion was obtained using Fogarty hydragrip clamps. A longitudinal aortotomy was made from the level of the left renal artery and extended onto the left common iliac artery. The true lumen of the infrarenal aorta was nearly occluded, and extensive thrombus was present throughout the false lumen. The septum

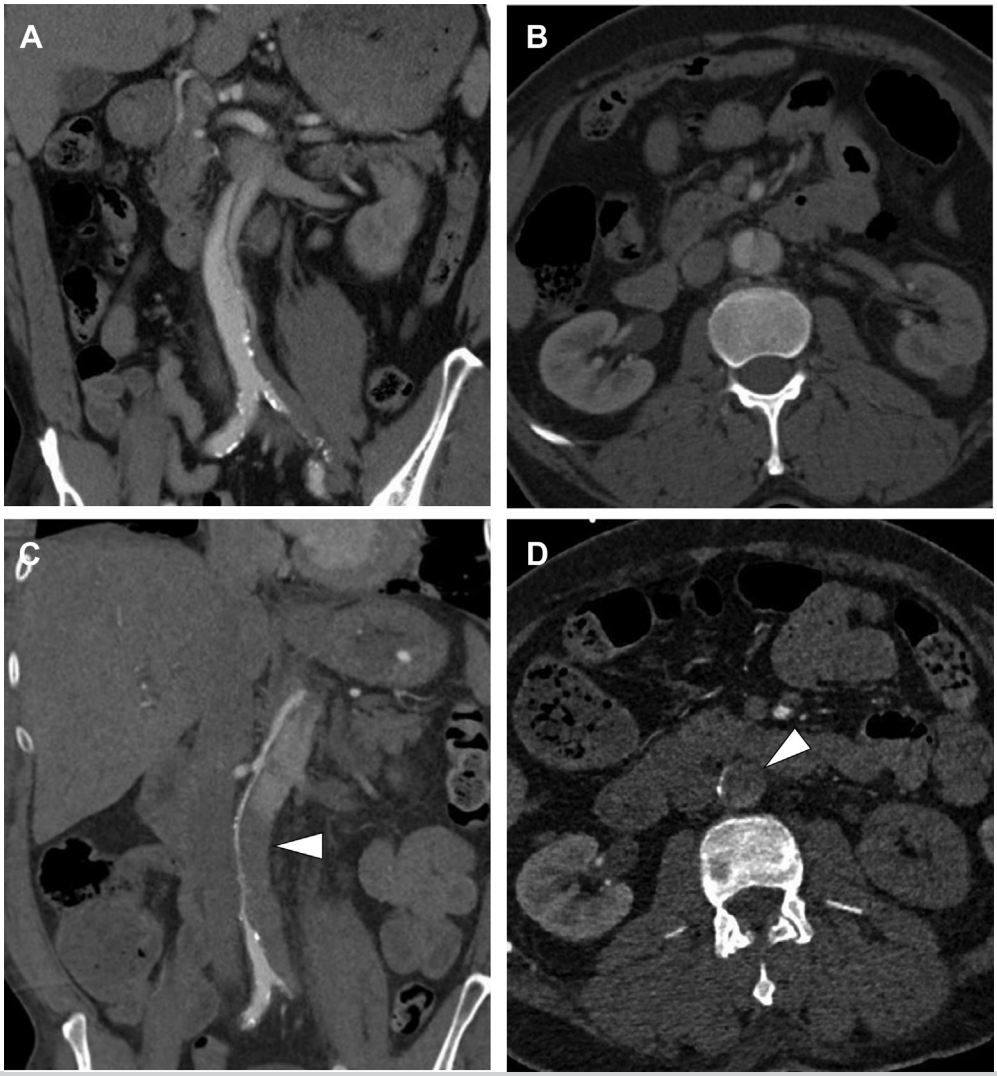

Fig 2. Computed tomography (CT) of abdomen and pelvis of patient 2 in coronal (**A**) and axial (**B**) planes at presentation. False lumen thrombosis and true lumen obstruction (*white arrowheads*) in coronal (**C**) and axial (**D**) planes from the repeat CT after developing ischemic symptoms.

was excised from the level of the left renal artery down to the distal left common iliac artery. The remainder of the aortic wall appeared normal. The residual septum was fenestrated proximally and tacked down distally in the left common iliac artery using interrupted Prolene sutures. The inferior mesenteric artery was identified and preserved. The aortotomy was closed with a 15-cm-long bovine pericardial patch to avoid narrowing the luminal diameter. After reperfusion, the patient had palpable femoral pulses and multiphasic pedal Doppler signals. The ABI was 0.6 bilaterally, unchanged from his admission examination.

Postoperative course

Patient 1. The patient recovered well. A follow-up CT scan 1 week later showed a patent aortic repair (Fig 3, *B*). He later underwent explantation of his femoro-femoral bypass graft because of a groin surgical site infection. He had normal bilateral lower extremity perfusion after graft removal.

Patient 2. The patient regained full motor and sensory function and was discharged home on postoperative day 7 without complications. He had multiphasic pedal Doppler signals and no new lower extremity ischemic symptoms. A follow-up CT scan 2 weeks after his index operation showed a widely patent aortic repair (Fig 3, *C*).

DISCUSSION

Although uncomplicated type B aortic dissection can be managed medically with anti-impulse therapy, most type A and complicated type B dissections will require surgical intervention. For patients with type B dissection, complications such as visceral or extremity malperfusion carry a risk of mortality up to 20% compared with 6.1% for those with an uncomplicated dissection.[1] The goal of surgery is to restore true lumen blood flow, correct branch vessel malperfusion, and promote false lumen thrombosis and favorable aortic remodeling. When

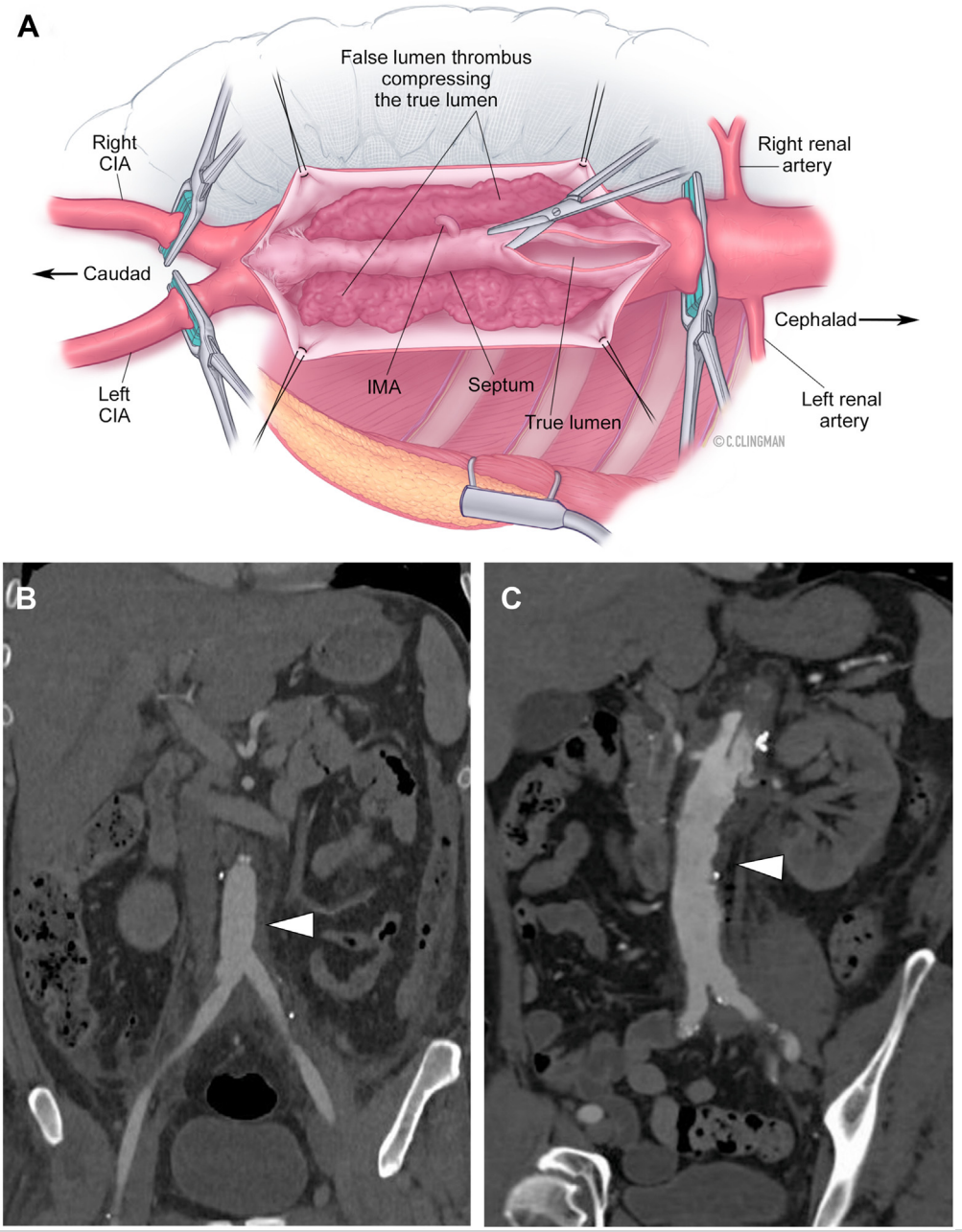

Fig 3. A, Illustration of open aortic septectomy. After lateral aortotomy, thrombus in the false lumen was removed, and the septum was incised to reveal the true lumen. The septum was excised with scissors. A generous fenestration was fashioned proximally up to the aortic clamp. The distal end points were tacked down with interrupted Prolene sutures. Postoperative surveillance computed tomography images of patient 1 (**B**) and patient 2 (**B**) after open aortic septectomy. *White arrows* demonstrate patent aortic true lumen flow. *CIA,* Common iliac artery; *IMA,* inferior mesenteric artery.

proximal aortic repair has failed to correct distal malperfusion, additional intervention will be required.

False lumen flow and pressurization with subsequent compression of the true lumen is the mechanism of peripheral malperfusion and can be either a static or dynamic process.[6] Bare metal stent placement in the true lumen of the aortic visceral segment either alone or in conjunction with proximal thoracic endovascular aortic repair, termed the PETTICOAT technique,[7] has been used to reexpand the true lumen. Alternatively, endovascular fenestration of the septum has become an increasingly popular strategy to equalize the pressure differential and promote true lumen reexpansion.[8] However, both of these procedures will typically be performed in the presence of a patent false lumen. In the present patients, the etiology of their malperfusion was

acute thrombosis of the false lumen, leading to true lumen collapse. In the absence of a patent false lumen, endovascular fenestration will not be technically feasible, and bare metal stent placement would unlikely lead to adequate true lumen reexpansion. Open aortic septectomy and thrombectomy allowed for full restoration of perfusion without aortic replacement. The decision of where to place the clamp proximally can be made on a case-by-case basis. In patient 1, the symptoms were restricted to the lower extremities; hence, an infrarenal clamp was used. Patient 2 had also had lower extremity symptoms but had also had severe true lumen compression up to the level of the renal arteries; hence, a suprarenal clamp was used to allow for fenestration up to that level. In neither case was clinical or radiographic evidence of visceral malperfusion present that would have necessitated the use of a supraceliac clamp. In both patients, malperfusion symptoms resolved and follow-up imaging demonstrated patent aortic repairs. We performed patch repair rather than primary repair of both longitudinal aortotomies to avoid narrowing the luminal diameter. For both patients, longitudinal surveillance imaging is planned to monitor for aneurysmal degeneration of the more proximal residual aortic dissection and for patch degeneration.

CONCLUSIONS

Open aortic septectomy and thrombectomy are uncommonly performed but are useful options for patients with complicated aortic dissection associated with malperfusion secondary to false lumen thrombosis.

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