A Case of Intestinal Necrosis after Bilateral Internal Iliac Artery-Preserving Endovascular Repair for Abdominal Aortic Aneurysm

Tomomi Nakajima, MD, Masataka Sato, MD, Akito Imai, MD, and Yasunori Watanabe, MD

A 79-year-old man underwent endovascular repair for abdominal aortic aneurysm (AAA), and both internal iliac arteries (IIAs) were preserved. Postoperatively, loss of appetite developed. On the fifth day, computerized tomography (CT) showed inferior mesenteric artery thrombus formation, necrosis of the descending colon and rectum, and generalized peritonitis. The endovascular devices had not migrated. A colonic resection was performed. Histological analysis confirmed intestinal necrosis associated with mesenteric thrombus. The colon can become necrotic even if both IIAs are patent. Ischemic changes in the colon should be detected if it occurs and subsequent laparotomy should be done if it is necessary.

Keywords: intestinal ischemia, endovascular abdominal aortic aneurysm repair

Introduction

Intestinal ischemia and necrosis is a complication of abdominal aortic surgery. The risk is lower with endovascular repair than open surgery.\(^1\)\(^–\)\(^4\) In addition, it has been proposed that one or both internal iliac arteries (IIAs) should be preserved to avoid intestinal ischemia.\(^1\) We report a case of intestinal necrosis after bilateral IIA-preserving endovascular repair for abdominal aortic aneurysm (AAA).

Case Report

A 79-year-old man presented for follow-up evaluation of an AAA. The AAA had enlarged to 5.0 cm in diameter. It was a candidate for surgery, and the patient desired treatment. He was medically at high risk for open repair because of advanced age, previous open abdominal surgery for benign prostatic hypertrophy, and previous percutaneous coronary intervention and coronary artery bypass grafting for angina.

Computerized tomography (CT) showed infrarenal AAA, and its anatomical form was suitable for endovascular repair. There was low-grade calcification in the left and right common iliac arteries and IIAs. There was intramural thrombus formation in the AAA, but the inferior mesenteric artery (IMA) was patent. SMA was also patent and the origin of SMA was not stenotic (Fig. 1). The patient was offered the option of endovascular repair and gave informed consent for it. The endovascular repair was performed under general anesthesia. We used a Zenith AAA endovascular graft (Cook Inc., Bloomington, Indiana, IN, USA). The devices were delivered with Amplatz super stiff wire (Cook Inc.), from right and left external iliac arteries (EIAs). Before the sheaths were inserted to EIAs, 6000 units of heparin was administered intravenously. The procedure was performed with 3 pieces devices, TFFB2896ZT as main trunk, TFLE1856ZT as contralateral leg, and TFLE2039ZT as ipsilateral leg. Before the deploy of devices, angiography was taken and IMA was patent. They were successfully deployed, and the left and right IIAs were preserved. Aortic extension devices were not used. There were no endoleaks or other problems on intraoperative angiography. (Fig. 2) The procedure lasted 2.5h. There were no events during the procedure such as decreased blood pressure.

Postoperatively, the patient had a natural course, and systemic blood pressure was maintained at 100–130 mmHg. However, symptoms such as loss of appetite appeared on the first postoperative day. A sense of abdominal fullness was exhibited on the second postoperative day, which was worse on the fourth postoperative day. Despite a movement of the bowel, radiography showed ileus, and there were signs of an increased inflammatory reaction (C-reactive protein [CRP] level was increased to 23.57 mg/dL). On the fifth postoperative day, body temperature had increased to
39 degrees centigrade. CT showed no change in the location of the endovascular devices and that the left and right IIAs and superior mesenteric artery (SMA) were patent. However, the IMA was occluded at the origin, but left colic artery was enhanced via collateral circulation. The periphery of the IMA was enhanced in a patchy fashion. The descending colon, sigmoid colon and rectum were also not enhanced (Fig. 2). The diagnosis was IMA thrombus formation, necrosis of the descending colon and rectum, and generalized peritonitis. The peak creatine kinase [CK] value was 397 IU/L on second postoperative day. There was no significant acidemia. They were normal postoperative course.
A colonic resection with formation of Hartmann’s pouch and colostomy was performed. There was necrosis of the intestinal canal in the area between the left colic flexure and rectum above the peritoneal reflection (Fig. 3). There was moderate contamination ascites. The histological analysis confirmed intestinal necrosis associated with mesenteric thrombus formation. There is full thickness necrosis of intestinal mucosa, and there is no perforation of the intestinal wall.

After the colonic resection, the patient required treatment with a ventilator for four days. However, he recovered without further complications and was ambulatory and able to be discharged 35 days after the colonic surgery.

Discussion

Colonic ischemia following open AAA repair occurs in 0.8%–11% of cases, but this incidence decreases to 1%–2.9% with endovascular repair. However, considering the very high rate of overall mortality of 25%–88% with colonic ischemia, this remains a very severe complication.

Colonic ischemia is primarily caused by perioperative hypoperfusion and reperfusion injury. It is associated with SMA thrombus formation, non-occlusive mesenteric ischemia (NOMI), cholesterol crystal embolization (CCE), and IIA thrombus formation.

In our case, the SMA, IMA, and left and right IIAs were patent before endovascular repair, and only the IMA became occluded after procedure. There were no events such as decreased blood pressure or reduction in colonic blood flow in the peri-procedural period. Nevertheless, CT showed inadequate blood flow to the IMA-perfused area, and pathological examination revealed that the lesion was present continuously, with no obstructing material. This conformed to the findings in artery thrombus. Considering the pathological findings of this case, NOMI and CCE were unlikely causes. Therefore, we diagnosed colon ischemia caused by IMA thrombus formation associated with endovascular aortic repair.

Although the IMA is usually occluded by the endograft in endovascular aortic repair, it is uncommon for this to cause colonic ischemia. A previous publication states that preoperative patent IMA may be a risk factor for colonic ischemia. In addition, it has been proposed that SMA disease, dissection or retractor injury, prior colon resection, or exclusion of IIA perfusion impair collateral circulation in patients with IMA obstruction. In our case, a retractor was not used, and preoperative and postoperative CT showed no dissection injury or SMA disease. Therefore, we considered that it was impossible to evaluate preoperatively the risk of impaired collateral circulation. Even if the left and right IIAs and SMA are patent, IMA obstruction may independently cause necrosis of the descending colon and rectum. This is very rare, and we could find only one case similar to ours.

At present, there are no well-defined predictive factors for colonic ischemia. However, it has been proposed that patients with symptomatic visceral ischemia, ruptured AAA, a patent IMA, or a meandering marginal artery of the colon are at high risk for colonic ischemia. A meandering artery from the SMA and the left and right IIAs perfuse the vascularization area of the IMA as collateral blood circulation. In open surgery, criteria for IMA reconstruction have been proposed. Specific methods include measurement of IMA stump pressure and blood flow measurement of the mesentery with Doppler blood flow measurement intraoperatively. In addition, the color and condition of the colon can be assessed directly in open repair. Although, in endovascular repair, these examinations are not possible, important clinical signs of colon ischemia are not masked by the after effects of a laparotomy. Hence, it is easier to detect colon ischemia and make a decision about whether to perform a laparotomy at an early stage.

If CT shows few collateral arteries from the SMA or IIAs to the IMA perfusion area before the operation, it is necessary to review the operative method. Preoperative angiography of the IMA and SMA may confirm the presence of an arterial arcade. However, this may be difficult to evaluate in the peripheral area of the IMA.

It is necessary to keep in mind that colonic ischemia may occur even if the left and right IIAs are preserved. Patients should be followed up closely, and prompt CT
or laparotomy should be undertaken if symptoms of suspected colonic ischemia appear. This knowledge may help to reduce mortality in these cases.

**Conclusion**

We had a very rare experience of intestinal necrosis after bilateral IIA-preserving endovascular repair for AAA. We have to keep in mind that the colon may become necrotic even if the left and right IIAs are patent. Ischemic changes in the colon should be detected if it occurs and subsequent laparotomy should be done if it is necessary.

**Disclosure Statement**

All authors have no conflict of interest.

**Author Contributions**

Report conception: TN
Data collection: TN
Writing: TN
Critical review and revision: all authors
Final approval of the article: all authors
Accountability for all aspects of the work: all authors

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