Successful Endovascular Repair for Abdominal Aortic Aneurysm Presenting as Aortoduodenal Syndrome

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Aortoduodenal syndrome is a rare duodenal obstruction caused by an abdominal aortic aneurysm. Current treatment involves open aneurysmal repair according to the theory that this procedure releases the duodenum from mechanical compression. However, the mechanism of duodenal blockage remains unclear and reports of endovascular aneurysm repair (EVAR) for aortoduodenal syndrome are quite rare. We report our successful case of EVAR for aortoduodenal syndrome without aneurysmal shrinkage and discuss the mechanism of duodenal obstruction.

Keywords: aortoduodenal syndrome, endovascular repair, duodenal obstruction

Introduction

Aortoduodenal syndrome is an exceedingly rare condition characterized by an obstruction of the duodenum because of an abdominal aortic aneurysm (AAA). Since William Osler described the aortoduodenal syndrome in 1905,1) less than 50 cases have been reported. Historically, treatment has transitioned from gastric bypass surgery to open aneurysm resection following advancements in vascular surgery, and the survival rate has improved.2) Currently, open aneurysm repair is considered the standard treatment for aortoduodenal syndrome, supported by the hypothesis that the aneurysmal resection releases the duodenum from mechanical compression by the AAA.3) However, the exact mechanism of duodenal obstruction has not yet been clarified. There have been few reports of successful endovascular management for aortoduodenal syndrome without aneurysmal shrinkage.4)

We report a case of successful endovascular management for aortoduodenal syndrome without aneurysmal shrinkage and discuss the mechanisms of aortoduodenal syndrome and the benefits of endovascular aneurysm repair (EVAR). The patient provided consent for the publication of this case report.

Case Report

An 81-year-old man presented with sudden abdominal pain and several days of nausea and abdominal discomfort. He was afebrile and had no diarrhea. Physical examination revealed a pulsatile mass in the central abdomen and a distended upper abdomen. Contrast-enhanced computed tomography (CT) revealed an infrarenal AAA with a diameter of 50 mm and a duodenal obstruction at the third portion between the AAA and the superior mesenteric artery (SMA). CT also displayed distension of the stomach and the first and second portions of the duodenum, accompanied by duodenal wall edema (Fig. 1). He was diagnosed with aortoduodenal syndrome.

Considering the patient’s acute symptoms, the possibility of AAA impending rupture could not be excluded completely; thus, emergency surgery was performed. Preoperative upper gastrointestinal examination with an oral contrast agent was not performed because of the emergent situation. Endovascular treatment was selected on the basis of the patient’s age, frailty (Clinical Frailty Scale 6),5) and anatomical suitability as his proximal landing zone was straight and over 2 cm long. Before the operation, a nasogastric tube was inserted and the intragastric fluid was suctioned completely to prevent vomiting during the procedure. Abdominal pain did not diminish after the aspiration of the gastric fluid.
Fig. 1  Preoperative computed tomography (CT) findings.
(A, B) Preoperative CT showed a dilated stomach (right arrow) and duodenum (left arrow). Duodenum wall was edematous. (C, D) The third portion of the duodenum (left arrow) was compressed by the infrarenal abdominal aortic aneurysm (AAA) (right arrow). The diameter of the AAA was 50 mm.

Fig. 2  Perioperative digital subtraction angiogram (DSA) findings.
(A) DSA after deployment of the main body and contralateral leg showed type IA endoleak (arrow). (B) Final DSA after deployment of aortic cuff showed no endoleak. (C) Postoperative computed tomography showed that stent graft was successfully deployed among infrarenal abdominal aorta and bilateral common iliac arteries.
Endovascular Repair for Aortoduodenal Syndrome

The procedure was performed under local anesthesia and conscious sedation with dexmedetomidine to prevent aspiration pneumonia caused by vomiting on tracheal intubation. The AAA was successfully treated by fitting an endoprosthesis (Gore Excluder; W. L. Gore and Associates Inc., Flagstaff, AZ, USA). After the exposure of the bilateral common femoral artery, the main body and contralateral leg of the endoprosthesis were deployed. A proximal aortic extender was added because the digital subtraction angiography revealed a type IA endoleak. On the final angiogram, there was no endoleak (Fig. 2). The operative time was 96 min, and surgery was completed without complications. The patient’s abdominal pain was diminished after the operation, and a physical examination revealed a diminishment in aneurysmal pulsation.

The patient initially received parenteral nutrition and then began oral intake on the postoperative day (POD) 2 because of decreased nasogastric output. However, his oral intake was suspended and parenteral nutrition via a nasogastric tube was restarted on POD3 because of vomiting. CT indicated that the duodenal wall has remained edematous and the stomach and duodenum remained distended. His oral intake was restarted on POD8 after it was confirmed via CT that the edema of the duodenal wall had diminished, and distension of stomach and duodenum had improved on CT. His oral intake volume was gradually increased, and his meal form was gradually converted from liquid to solid from POD8 to POD30, and he continued oral intake without problems. A final CT showed the diminishment of the edema of the duodenal wall and no signs of duodenal obstruction, although the aneurysm sac remained unchanged in size (Fig. 3). The patient was discharged from the hospital without complications on POD32. He has been free from obstructive symptoms for 8 months since the operation, without aneurysmal shrinkage.

Discussion

This paper describes a case of duodenal obstruction, which was successfully managed via EVAR. EVAR under local anesthesia was selected to minimize the invasiveness of approach considering the patient’s frailty and to prevent aspiration pneumonia. The obstructive symptoms gradually improved after the operation without aneurysmal pulsation.

Although several hypotheses for this mechanism have been proposed, including mechanical pinching between the SMA and AAA, inhibition of peristalsis by aneurysm, and narrowing of the duodenal lumen due to aneurysm expansion causing foreshortening of the posterior duodenal wall, the exact mechanism of duodenal obstruction in aortoduodenal syndrome is still poorly understood.3) Based on the pathophysiology of an ileus4) and the clinical course of our case, a hypothesis could be advocated that postoperative nasogastric fluid gradually decreased in accordance with the improvement in the duodenal wall edema. Aneurysmal pulsation and sac pressure initially
inhibited the passage of enteric contents at the third portion of the duodenum, and this led to hypermotility of the duodenum as seen in the ileus. However, the duodenal contraction later became hypotonic because of enteric fatigue, and this resulted in water and electrolyte accumulation in the duodenal lumen and wall. Together, edematous changes in the duodenal wall, an increase in intraluminal pressure in the duodenum, and a duodenal hypomotility caused the duodenal obstruction.

Within this hypothesis, the duodenal obstruction could have been relieved because of the improvements in the edematous change in the duodenal wall and the duodenal motility, and the intraluminal pressure decreased because of the nasogastric suction and depressurization of the aneurysm sac after EVAR. Dias et al. demonstrated that intra-aneurysm pressure and pulsatility reduce significantly after a successful EVAR without an endoleak. In our case, the physical examination confirmed that the pulsation of the abdominal mass diminished after EVAR, though the intra-aneurysm pressure was not measured.

Moreover, the improvement in the aneurysm wall edema might have also contributed to the improvement of duodenal obstruction. It has been reported that stent grafts block and reduce the blood flow to the aneurysm wall from the intravascular space. In this case, preoperative CT showed a slightly thickened and well-enhanced aneurysm wall, whereas postoperative CT showed the reduction in aneurysm wall enhancement and aneurysm wall thickness compared with the preoperative CT findings. This means that the blood flow to the aneurysm wall reduced, and this might have led to the relief of aneurysm wall inflammation. Thus, the edema of the duodenal wall would have also improved partially.

Because obstructive symptoms occur in cases with various size of aneurysm from 40 to 100 mm, it can also be concluded that mechanical pinching by the AAA was unlikely to be the main cause of duodenal obstruction in this case. Moreover, obstructive symptoms improved after EVAR without aneurysmal sac shrinkage. It could be said that depressurization of the aneurysm sac after EVAR is sufficient to reduce obstructive symptoms in cases of duodenal obstruction and that open sac resection is therefore not always necessary to relieve obstructive symptoms because intra-aneurysm pressure reduces even in cases with unchanged aneurysm diameter. In terms of intra-aneurysm pressure, Gore Excluder endoprosthesis was selected because of low permeability and thus less risk of type IV endoleak.

However, these hypotheses are not sufficiently supported by experimental data. Although our patient underwent emergent EVAR because of impending rupture of aortic aneurysm rupture, obstructive symptoms might perhaps have improved without EVAR when duodenal edema was sufficiently improved with intragastric fluid suction alone. Thus, perioperative intraduodenal pressure measurement in endovascular and open repair could be considered in further investigations to clarify which of the following truly contributes to relief of duodenal obstruction: intragastric fluid suction, improvement of duodenal wall edema, depressurization of aneurysm due to EVAR, or surgical removal of aortic aneurysm.

As a treatment strategy, EVAR under local anesthesia had several benefits in this case. First, EVAR is less invasive than open aneurysm repair and is therefore preferred for a frail octogenarian patient. Second, local anesthesia, conscious sedation with dexmedetomidine and preoperative nasogastric fluid suction reduce the risk of aspiration pneumonia from vomiting on tracheal intubation, compared with general anesthesia. To minimize the risk of aspiration pneumonia, preoperative adequate nasogastric fluid suction to release full stomach state and conscious sedation with drugs, such as dexmedetomidine, which have less risk of respiratory depression, are recommended. In case that the patient is unstable and restless under local anesthesia and adequate sedation, switching to general anesthesia under crush induction should be considered. Third, EVAR has less risk of postoperative ileus than open aneurysm repair. In this case, open aneurysm repair has led potentially to the postoperative ileus resulting in an unimproved obstructive symptoms. With endovascular treatment, clear and rapid improvements in symptoms were seen in this case, suggesting that the endovascular management for cases of AAA presenting with duodenal obstruction is feasible.

Finally, an alternative strategy was arranged in case EVAR failed to improve the duodenal obstruction. If required, open surgery in this situation could be simplified to aneurysm sac plication without the need for an aortic clamp or aortic balloon occlusion, provided that proximal sealing with a stent graft was successful. Even if EVAR could not improve the duodenal obstruction, a hybrid procedure with EVAR and secondary open surgery could reduce the total invasiveness of a primary open aneurysmal resection.

A limitation of this case is that preoperative and postoperative upper gastrointestinal series data were not compared because of the preoperative emergency situation and the lack of postoperative patient consent.

**Conclusion**

A case of AAA presenting as aortoduodenal syndrome was successfully treated via EVAR. In this case, sac decompression due to EVAR, intraluminal pressure reduction by nasogastric tube drainage, and a gradual transition to oral nutritional intake were sufficient for the release of duode-
nal obstruction. EVAR may be considered as a treatment option for cases of AAA presenting with aortoduodenal syndrome.

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Disclosure Statement
The authors declare no conflicts of interest.

Additional Note
The patient provided consent for the publication of this case report.

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Study conception: YK, YO, YS
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Analysis: YK, MT
Investigation: YK, YO, YS
Writing: YK, HK
Critical review and revision: all authors
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Accountability for all aspects of the work: all authors

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