Suprarenal Fixation Resulting in Intestinal Ischemia after Endovascular Aortic Aneurysm Repair

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Endovascular aneurysm repair (EVAR) may be associated with specific stent- and procedure-related complications. Hepatic artery anatomic variability may lead to dramatic consequences when unanticipated. A 64-year-old man presented with a 6-cm abdominal aortic aneurysm, suitable for an EVAR procedure. The EVAR procedure was uneventful and the patient was discharged after 2 days. After 2 weeks, he was readmitted for recurrent upper abdominal pain due to acute cholecystitis. The postoperative EVAR computed tomography scan was revisited and the suprarenal bare-metal stent of the Zenith device overlapped the highly calcified origin of both the superior mesenteric artery (SMA) and the celiac trunk. Moreover, the patient appeared to have a right replaced hepatic artery originating from the SMA. He developed diffuse, patchy ischemia of both the large and the entire small bowel, and quickly became unresponsive to vasopressor drugs. He died shortly thereafter. An EVAR procedure may result in a highly complicated course when hepatic artery anatomic variability is present. Fenestrated EVAR or proximal graft scallops should be considered for cases in which the proximal sealing zone is diseased and flow to visceral vessels is compromised.

Endovascular aortic aneurysm repair (EVAR) has been widely accepted as a less invasive treatment alternative to open surgical repair.1 Although the number of complications may be fewer, EVAR is associated with specific stent- and procedure-related adverse events, such as access artery injury, contrast nephropathy, colonic ischemia, spinal cord ischemia, endoleak, stent-graft infections, and limb ischemia.2,3 Intestinal tract ischemia is among the more serious post-EVAR adverse events, with incidence rates ranging from 1% to 10%.3 Presumed causes are nonocclusive ischemia due to shock and (inferior) mesenteric artery occlusion and/or atheroembolization. Celiac trunk or superior mesenteric artery (SMA) coverage or occlusion after EVAR is very rare and is most commonly reported after thoracic endovascular aortic aneurysm repair (TEVAR).5,6 Celiac trunk or SMA occlusion in the presence of a widely patent collateral circulation may protect the patient against ischemic complications.6 Although celiac trunk coverage can have dramatic consequences, it is only necessary in a small minority of TEVAR cases. However, unexpected complications may occur in the presence of anatomic arterial variations combined with a heavily calcified SMA origin. We describe the first reported case in which a right replaced hepatic artery originating from the SMA.
artery (RRHA) arising from the SMA led to an ischemic gallbladder after EVAR.

CASE REPORT

A 64-year-old patient presenting with back pain was found to have a 6.5-cm abdominal aortic aneurysm (AAA). Computed tomography (CT) scan showed an aneurysm with a diameter of 6.5 cm and a long (>4 cm) infrarenal neck with mild wall irregularities. Ileofemoral access was considered sufficient and, although the visceral vessels were severely calcified, there were no signs of stenosis or thrombus (Fig. 1). Overall, the aneurysm was judged to be suitable for an EVAR procedure. Informed consent was obtained and the procedure was scheduled. The EVAR procedure (Cook Medical, Inc., Bloomington, IN) was uneventful. Postoperative CT scan showed no endoleak, gallbladder/intestinal abnormalities, or compromised visceral vascularization, and the patient was discharged after 2 days (Fig. 2). After 10 days, however, the patient presented to the emergency room due to abdominal pain in the right upper quadrant. Physical examination showed rebound tenderness in the upper abdomen. Laboratory blood tests showed slightly elevated leukocytes (11,000/L) and a high C-reactive protein (CRP) level (19 mg/L). Additional evaluation by CT scan showed no abnormalities associated with the stent graft and as well as no other signs of abdominal pathology. As gastric ulcer was considered most likely in the differential diagnosis, the patient returned home with a prescription for a proton pump inhibitor (esomeprazole). After another 2 days, he returned to the hospital and was readmitted for recurrent upper abdominal pain. A further abdominal ultrasound examination suggested acute cholecystitis. The same day a laparoscopic cholecystectomy was performed of a completely necrotic gallbladder. No additional abnormalities were detected during laparoscopy. Subsequent pathologic examination confirmed the diagnosis of a fully necrotic gallbladder. However, abdominal symptoms persisted and leukocytes and CRP remained elevated. Therefore, a diagnostic laparoscopy was performed 2 days later; however, no abnormalities were found, in particular no infected biloma, abscesses, or any other gastrointestinal tract abnormalities.

In the absence of any clear explanations for the patient’s persisting symptoms the postoperative EVAR CT scan was revisited by our vascular team. On review of the original scan, the proximal bare-metal stents were noted to be impeding flow to the heavily calcified SMA origin and the celiac trunk (Figs. 3 and 4). Moreover, the patient appeared to have an RRHA originating from the SMA, although this was already known at the time the EVAR procedure was conducted (Fig. 5). The cystic artery originated from the RRHA. We concluded that this anatomic variation may very well have caused the ischemic cholecystitis as well as the accompanying persisting abdominal pain due to low flow or ischemia. The next day the radiologist successfully placed a 7 × 18-mm balloon-expandable stent (Scuba®; Invatec Medical, Fig. 1. CTA shows a preoperative axial view of the highly calcified origin of the coeliac trunk (A) and superior mesenteric artery (B).

Fig. 2. CTA shows a sagittal view of the aorta postoperatively. The patency of the coeliac trunk and SMA are clearly visible.
Roncadelle, Italy) in both the SMA and the celiac trunk using an over-the-wire technique. Completion angiography showed no evidence of distal atheroemboli. Unfortunately, the patient’s condition deteriorated the next day and a laparotomy was performed. Diffuse patchy ischemia of both the large and the entire small bowel was found, too extensive for resection. A “wait-and-see” approach was followed and the patient was admitted to the intensive care unit (ICU).

The following day a second-look laparotomy was carried out showing a necrotic jejunum, for which a resection was performed. Three days later the patient deteriorated into septic conditions and, during his third laparotomy, fecal spill was found originating from a previous iatrogenic serosal injury. The perforation was sutured and, after extensive abdominal lavage, he returned to the ICU. Multiple-organ failure progressed in the following days. Another CT scan revealed intraabdominal free fluid and a patent stent in both the SMA and celiac trunk. The next day deterioration progressed rapidly with no responsive to vasopressor drugs in addition to worsening of respiratory conditions. Treatment was discontinued with the consent of the family and the subsequently patient died. Unfortunately, no permission for autopsy was obtained.

DISCUSSION

This is the first reported case in which EVAR led to an ischemic gallbladder, most likely due to an RRHA arising from the SMA. Although the proximal sealing zone had been more than sufficient with regard to endoleak occurrence, severe SMA origin atherosclerosis combined with partial coverage of the SMA origin by the bare-metal struts of the stent graft probably led to gallbladder ischemia in the presence of an RRHA. Eventually, disease progression occurred and intestinal tract ischemia and multiple-organ failure ultimately led to his death. Although the RRHA was recognized prior to the EVAR procedure, the severity of atherosclerosis in both the celiac trunk and SMA was underestimated. Acute acalculous cholecystitis is a condition normally only occurring in critically ill patients and is associated with high morbidity and mortality rates of 100% and 67%, respectively. The pathogenesis is somewhat obscure but has been theorized to be bile stasis, sepsis, or ischemia.\textsuperscript{7,8} The suggested relationship with visceral ischemia seems especially relevant when considering this case. However, most reported cases have established a connection with vascular graft occlusion instead of visceral artery atherosclerosis, as in this case.

The surgical anatomy of the hepatic arterial supply is highly variable.\textsuperscript{9} In general, vessels supplying the right and left liver lobes may arise from different origins, such as the SMA, left gastric artery, aorta, or even other visceral vessels. These anomalous
arteries can be either accessory or replaced branches. Various classifications of hepatic arterial variations have been proposed, but the most commonly used ones are those described by Michels and Hiatt et al., who established a classification consisting of 10 and 6 types, respectively. An RRAH arising from the SMA with otherwise normal anatomy, as in this case, was classified as type VI by Michels and type III by Hiatt and colleagues.

Preoperatively identifying accessory or replaced vessels is essential in anticipating future potentially complicated courses. Retrospectively, revascularization of both the celiac trunk and the SMA by bare-metal stenting prior to the EVAR procedure may have prevented the complications in the case we have described. However, revascularization of the celiac trunk and the SMA are associated with primary patency rates of 65% and concomitant risks of bowel ischemia. Alternatively, a Gore Excluder stent graft (W.L. Gore & Associates, Flagstaff, AZ) could have been used. This device has a fully covered proximal sealing cuff without protruding struts. Combining this with SMA stenting could have been done, while taking into account the aforementioned risks.

Other, yet new and even experimental, alternatives for the preservation of visceral vessels would have been a chimney graft technique, a flow-diverting stent, or a fenestrated stent graft. Although initial experiences with flow-diverting stents in the treatment of visceral and peripheral aneurysms have yielded satisfactory results, the results in aortic aneurysm repair are still uncertain. More experience and success have been achieved using the chimney graft technique. However, a recent review concluded that concerns regarding long-term durability and proximal fixation remain, which may affect further development. In contrast, considerable experience has been accumulated with fenestrated and branched endovascular repair of aortic aneurysms (FEVAR). Gastrointestinal ischemia and spinal cord ischemia result in serious complications. However, target vessel patency and survival rates are currently more than acceptable. Also, in this case report, a Reliant stent-graft balloon catheter (Medtronic Vascular, Inc., Santa Rosa, CA) was used for modeling the proximal part of the stent graft. In theory, this may also have contributed to deterioration of an already diseased and calcified SMA origin or led to thromboembolism. In retrospect, in the absence of a type I endoleak, we should have omitted this step.

It is uncertain whether application of one of these techniques would have prevented the unfortunate outcome in the case we have described. At our hospital, we have extensive experience with fenestrated stent grafts and, in retrospect, this technique should have been our first choice. The severe atherosclerosis, however, would have made stent placement challenging, making endograft repair with proximal graft scallops presumably the best alternative.

In conclusion, heavily calcified origins of the SMA or celiac trunk can result in disastrous (ischemic) complications in combination with a suprarenal bare-metal stent. In cases of any doubt associated with the proximal sealing zone, FEVAR or proximal graft scallops should be considered, or at least an infrarenal stent graft (such as the Gore Excluder).

REFERENCES