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Aortoenteric Fistulae: Present-Day Management

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An aortoenteric fistula (AEF) is a communication between the aorta and an adjacent loop of the bowel. The three most useful diagnostic modalities for detecting AEF are abdominal computed tomography scan with intravenous contrast, esophagogastroduodenoscopy, and arteriography. The treatment of AEFs has improved in recent years, but despite the multiple surgical techniques reported, many of the patients do not survive or are left debilitated after treatment. Endovascular repair is an efficient and safe method to stabilize patients with life-threatening AEFs. The aim of this study is to provide a comprehensive and synthetic review of the latest advantages on the diagnosis and management of primary and secondary AEF.

Key words: Aortoenteric fistula – Gastrointestinal bleeding – Perigraft infection – Open surgical repair – Endovascular repair – Complications

Aortoenteric fistula (AEF) is a rare clinical entity that was first described in 1818 by Sir Ashley Cooper.[1] The first secondary AEF appeared in 1953 in a report by Brock[2] who described a fistula between the proximal anastomosis of an aortic homograft and the duodenum. The disease is divided into two types—primary and secondary AEF.

Primary AEFs are uncommon and occur when a large, previously untreated aneurysm erodes into the adjacent bowel. The most common secondary AEFs occur in 0.3%–1.6%[3] of patients after aortic prosthetic reconstruction. There is little consensus about the optimal management of AEFs. Conventional treatment of an AEF consists of extra-anatomic bypass grafting and aortic ligation. For a secondary AEF, it is subsequent graft removal. This technique is associated with a 25%–90% operative mortality rate,[3] an amputation rate of 5%–25%,[5,6] and an aortic stump rupture rate of 10%–50%. In situ aortic reconstruction has been proposed as a less morbid alternative. However, its average perioperative mortality is reported to be 27%–30%.[3] Several recent case reports describe the endovascular management of AEFs in high-risk patients and suggest lower perioperative morbidity and mortality rates than traditional surgery.[6,7] It is a very promising technique to minimize perioperative trauma, creating a better situation to perform open surgery at a later time.

Primary AEF

When an AEF occurs in the absence of previous aortic aneurysm surgery or trauma, it is called a

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primary AEF—a condition much less frequent than a secondary AEF, which occurs as a result of previous aortic aneurysm grafting. Salmon[8] described the first case report of a primary AEF in 1843. The most common predisposing condition associated with primary AEF is atherosclerotic abdominal aortic aneurysm.[4–11] Less commonly encountered conditions that may lead to primary AEFs include syphilis, tuberculosis, mycotic infection, and collagen vascular disease. When there is communication between the aorta and the adjacent viscera, ectopic gas adjacent to or within the aorta is the predominant computed tomography (CT) finding. The fat plane that normally separates the aorta from the bowel is usually obliterated along the affected segment. An associated hematoma sometimes is seen in the retroperitoneum or within the bowel wall or lumen. Since the article by Salmon[8] about 250 cases have been reported in literature.[9,12–16] The incidence at autopsy is between 0.04% and 0.07%.[13–16] Secondary AEFs occur as complications of aortic reconstructive surgery with or without the placement of an aortic stent graft. The importance of primary AEFs lies mainly in their being a rare and easily missed cause of gastrointestinal bleeding and their association with a very high mortality rate if left untreated. In the absence of treatment, the mortality rate is almost 100%. With surgical intervention, survival ranges from 18%–93%.[15–22] As many as 40% of operated cases develop complications, and the overall postoperative mortality rate is more than 30%.[12–14] Primary AEFs associated with infected aneurysms have a worse prognosis, with a postoperative mortality rate exceeding 50%.[19,23]

Secondary AEF

The incidence of secondary AEF is 0.5%–2.3%.[15] Secondary AEFs occur as complications of aortic reconstructive surgery with or without the placement of an aortic stent graft. An estimated 80% of secondary AEFs affect the duodenum, mostly the third and fourth parts (the horizontal and ascending duodenum).[24] Perigraft soft tissue edema, fluid, and ectopic gas may be normal CT findings immediately after surgery. However, after 3–4 weeks, any ectopic gas is abnormal and should be considered a sign of perigraft infection and possibly fistulization to the bowel.[25] The perigraft soft tissue thickening, fluid, or hematoma should resolve within 2–3 months after surgery.[25] Although with aortic aneurysms treated with endografts, there are cases of AEF that occur with graft migration as well as endoleaks that lead to fistulization. The number of secondary AEFs reported in patients with a history of open aortic repair is larger than that reported among patients with a history of endovascular stent placement.[26–30]

Potential Mimics

Because of their overlapping CT features, AEF and perigraft infections may be difficult or impossible to differentiate. However, the specific CT findings that correlate strongly with the presence of an AEF include ectopic gas, focal bowel wall thickening, breach of the aortic wall, and extravasation of contrast material into the bowel lumen.[25,31,32] In addition, when gastrointestinal bleeding is the primary clinical manifestation, any CT features of perigraft infection should raise the radiologist's concern about the possibility of a secondary AEF.

Mimics of AEFs

Mimics of AEFs include retroperitoneal fibrosis, a fibrosing process that involves the retroperitoneum, the area surrounding the abdominal aorta and common iliac arteries with an cause that has yet to be entirely elucidated, although two-thirds of all cases are deemed to be idiopathic, with no known cause.

Infected aortic aneurysms are estimated to account for 0.7%–2.6% of aortic aneurysms[33,34] and their prompt recognition is crucial for prompt surgical intervention and patient survival. Most patients with an infected aortic aneurysm have no specific symptoms until aneurysmal rupture or late-stage septic shock occurs and clinical evidence of bacteremia, including persistent fever, leukocytosis, or both, is present.

Infectious aortitis is an inflammation of the aorta caused by a bacterial, viral, or fungal infection with *Staphylococcus*, *Streptococcus*, and *Salmonella.* These are common causative pathogens responsible for this kind of aortitis.

Finally, perigraft infection without fistulisation can be a mimic of AEFs. Differentiation is aided by the observation of ectopic gas, loss of the normal fat plane, extravasation of aortic contrast material into the enteric lumen, or leakage of enteric contrast material into the paraprosthetic space. These features are highly suggestive of AEF in a patient with bleeding in the gastrointestinal tract. Extravasation of aortic contrast material into the bowel lumen is extremely rare but is the most specific feature of AEF.[25,32] Leakage of enteric contrast material into

the periaortic space is another rare but direct sign of AEF.[35]

Causes

The exact pathogenesis of the development of AEF has not been fully elucidated, but both mechanical erosion and infection are thought to play a role.[36–39] Furthermore, recently there have been multiple reports of AEFs developing after endovascular aortic repair, despite the theoretical lack of extraluminal disruption.[40,41] Regardless of etiology, the traditional management goals of AEF have been to control hemorrhage and infection, and maintain adequate distal perfusion. These have been achieved through graft excision and extra-anatomic bypass. In recent years, endovascular repair has emerged as another therapeutic option, particularly for the rapid control of bleeding from AEF.

Primary AEFs are thought to result mostly from direct wear and inflammatory destruction of an aortic aneurysm. Such fistulae, arising from an atherosclerotic abdominal aortic aneurysm, comprise 73% of all primary AEFs, whereas 26% are caused by traumatic or mycotic aneurysms.[23,42–44] The most common infectious agents responsible for mycotic aneurysms are *Klebsiella* and *Salmonella*, although *Staphylococcus* and *Streptococcus* have also been implicated.[19] Much rarer causes, such as radiation, infection, tumors, peptic ulcers, inflammatory bowel disease (Crohn's disease), and ingestion of foreign bodies, account for the remaining 1%.[45] Due to anatomic proximity, the third part of the duodenum is most frequently involved. About two-thirds of primary AEFs occur at this site, whereas the fourth part of the duodenum is affected in one-third of cases.[16]

Secondary AEFs occur as a complication of advanced perigraft infection, an origin that is generally confirmed at either surgery or autopsy. The dominant cause is vascular reconstructive surgery. It has been postulated that a combination of chronic low grade infection of the aortic graft and repetitive pressure on the intestine from aortic pulsations leads to the formation of these fistulas.[46,47] As a result, AEF and perigraft infection have many similar imaging features.[25]

Clinical Presentation

The clinical signs of AEF include hematemesis, melena, sepsis, acute abdominal pain and vomiting of fresh blood, low blood pressure, fast heart rate, decrease of hemoglobin level, and mild epigastric tenderness. Dark red blood per rectum means active bleeding.

Primary AEFs

The most common clinical features of primary AEFs are upper gastrointestinal bleeding (64%), abdominal pain (32%), and a pulsatile abdominal mass (25%).[12,48] However, these symptoms and signs are concomitantly present in only 10% of patients.[14,16] Other symptoms that may be present include back pain, melena, fever, sepsis, and shock. An intermittent herald bleed often precedes major hemorrhage and is followed by transient closure of the fistula by a thrombus and contraction of bowel around it. Early recognition of this symptom and its clinical importance is parmount. Occurrence of a herald bleed should critically arouse suspicion of a primary AEF and immediate action.

Secondary AEFs

Patients with a secondary AEF usually present with one or more of the following clinical signs and symptoms: gastrointestinal bleeding (80%), sepsis (44%), abdominal pain (30%), back pain (15%), groin mass (12%), and abdominal pulsatile mass (6%).[49] A massive gastrointestinal hemorrhage that occurs secondary to an AEF is usually preceded by transient and self-limited bleeding episodes (''herald bleeds'') that do not result from a true aortoenteric communication but, rather, from focal necrosis and mucosal ulceration.[50,51] The clinical manifestations are often crucial to the diagnosis of AEF, because no single imaging modality is capable of depicting this condition with high sensitivity and specificity.

Diagnosis

The three most useful diagnostic modalities for detecting AEFs are abdominal CT scan with intravenous contrast, endoscopy (esophagogastroduodenoscopy), and arteriography. Of these, the CT scan is by far superior as it is less invasive, more convenient, and more expedient than either esophagogastroduodenoscopy or arteriography. CT has another advantage in that it poses no risk of dislodging the aortic thrombus. Because of these qualities, and its widespread availability, short acquisition time, and high resolution, CT has become the first-line

modality for imaging evaluation of suspected AEF. However, despite its advantages, CT is of a variable sensitivity and specificity and can miss the presence of an AEF.[43] In very emergent cases when there is no time for preangiographic CT, the diagnosis is made by digital subtraction angiography with direct visualization of extravasation of intra-arterial contrast medium by fistula into the bowel lumen.

Esophagogastroduodenoscopy with a water-soluble contrast medium is an excellent investigation to rule out other causes of upper gastrointestinal bleeding such as ulcers and varices. It is helpful in documenting the presence of an AEF only when there is leakage of oral contrast material from the disrupted bowel wall into the perigraft space and should be performed only on a hemodynamically stable patient. A negative esophagogastroduodenoscopy does not rule out the possibility of an AEF.

Arteriography has a role in planning aortic reconstruction but, with the great improvements that have taken place in CT imaging, it has a very limited place in the acute setting.[52] Angiography with embolization therapy or stent placement also may be used to treat massive gastrointestinal bleeding secondary to an AEF.

Ultrasonography may be useful in unstable patients or those in whom the use of intravenous iodinated contrast material is contraindicated. However, ultrasonography is rarely indicated for the diagnosis of AEF.

Magnetic resonance imaging requires more acquisition time and greater technical expertise. Pulsation artifacts and the potential inability to differentiate perigraft gas from aortic wall calcification make magnetic resonance imaging of AEFs more difficult.

Treatment

AEF requires operative management, otherwise death from exsanguinating hemorrhage or sepsis is certain. In most large series only 10%–20% of patients required emergent surgery,[16] therefore the majority of the patients can be evaluated urgently rather than emergently. However, the survival rate is inversely related to the interval between the onset of bleeding and surgical intervention.[17]

Traditional treatment of AEF has consisted of graft excision and extra-anatomic bypass. Alternatives to this have included *in situ* graft replacement and simple graft excision alone. Since its inception, endovascular repair has offered a less invasive alternative for the management of aortic disease, including limited reports for the treatment of AEFs.

Perioperative management

All patients have to be started on broad-spectrum intravenous antibiotics before surgery once the diagnosis of AEF is made. They should be optimally prepared with fluid resuscitation or blood products guided by invasive monitoring, appropriate blood typing, and cross-matching tests. Preoperative placement of central monitoring lines should include Swan-Ganz catheterization. Broad-spectrum antibiotic therapy should be initiated as soon as the diagnosis is suspected. Antibiotics should include coverage for Gram-positive, Gram-negative, and enteric pathogens. The antibiotic therapy should be continued postoperatively for a minimum of 6 weeks, ultimately being tailored to the specific bacteria cultured during operation.

Open surgical treatment

The main goals of surgical therapy are (1) confirmation of the diagnosis, (2) control of bleeding, (3) repair of bowel defect, (4) eradication of associated infection, and (5) revascularization.

For the patients undergoing open repair there are the following options: (1) axillary–bifemoral bypass before graft excision and fistula repair, (2) axillary–bifemoral bypass after graft excision and fistula repair, and (3) primary aortic repair without extra-anatomic bypass. Open repair includes wide drainage and, when possible, placement of omentum in the aortic graft bed.[53] Intestinal repair consists of debridement and primary closure, resection with anastomosis, or resection with diversion.

For stable patients with minimal comorbidities and significant life expectancies, surgical management of AEF by staged extra-anatomic bypass followed by graft excision is optimal because it provides definitive management, limits lower extremity ischemia, and allows for patient recovery between operations. This has been shown to be relatively safe, but it is still associated with a significant mortality of up to 27%.[37,54] Simultaneous repair of extra-anatomic bypass followed immediately by graft excision is an acceptable alternative that has also been shown to be feasible, with acceptable morbidity and mortality.[36,55]

Patients who have undergone the first aortic repair for peripheral arterial disease have patency rates of the extra-anatomic axillary–bifemoral by-

pass of up to 97% the first year and 81.1% in 3 years,[56] whereas in patients who have undergone the first aortic repair for aneurysmal disease and with no evidence of peripheral arterial disease, the patency rates of the extra-anatomic axillary–bifemoral bypass are expected to be higher.

Additional surgical options include graft excision alone, graft excision with *in situ* replacement, and primary repair, all of which should be reserved for select patients.[57] The use of a venous autograft with *in situ* reconstructions is another option to avoid extra-anatomic bypass, as well as the use of allografts. Kieffer *et al*[55] described their 15-year experience with the use of fresh and cryopreserved aortic allografts. They presented results at least similar to all other methods of treating AEFs. An important conclusion of their experience is that fresh allografts should be avoided because of the elevated risk of rupture and cryopreserved allografts are preferred. In addition, they noticed higher rate of complications when using a descending thoracic aortic allograft, and they concluded that this part of the aorta should be avoided when choosing allografts.

Distinction between a more virulent and less virulent organism should be made in the decision to perform an extra-anatomic bypass versus an *in situ* revascularization. Specific antibiotics should be administered once the culture and sensitivities result is obtained. The management of the intestinal portion of fistulas has been demonstrated to have acceptable outcomes with simple bowel repair, although resection may be necessary for certain patients.[58] Intraoperative cultures should be taken and empiric antibiotic therapy initiated.

The Role of Endovascular Therapy

In recent years endovascular repair of primary AEFs has been reported as a successful alternative to open surgery.[40,41,59] This approach is very useful where open repair is not feasible because of anatomic reasons, for example, in patients who have undergone previous radiation therapy, resulting in extensive retroperitoneal fibrosis, or in unstable patients who are poor candidates for major surgery. Endovascular aortic repair offers these patients a less invasive alternative to seal the fistula and control bleeding.[60,61] However, unlike open repair with graft excision, the likely infected graft and/or aorta remains *in situ*, and this places the newly implanted stent graft at risk for infection.

Although these techniques may help, endovascular repair is limited because it does not include intestinal repair. In select patients, AEFs may heal after endovascular repair coupled with antibiotic therapy, particularly those patients with secondary infrarenal AEFs who present without evidence of sepsis. For patients who present with overt sepsis and have prohibitive comorbidities to immediate open repair, endovascular repair, when coupled with percutaneous or even open drainage, may serve as a palliative measure. In addition, for patients who may be medically optimized, endovascular repair may serve as a bridging therapy to open repair, particular if they have ongoing sepsis or other infectious complications. All patients undergoing endovascular repair should receive broad-spectrum antibiotics preoperatively and postoperatively and the question still remains whether the patient should be given lifelong prophylactic antibiotics.

In a comparison of open and endovascular treatment of AEF, endovascular management has been shown to achieve satisfactory short-term results.[62] Kakkos *et al*[63] reported on 2 patients with AEF treated by endovascular means. However, both patients needed open conversion, one in ninth postoperative month, and the other in the 16th postoperative month. Kakkos *et al*[63] concluded that ''due to the high rate of recurrent bleeding and sepsis, endovascular therapy should be used as a temporary measure and a bridge to open repair, whenever this is feasible.''

Comparison of Procedures

There are many studies[13,15,27,62] comparing standard open surgical repair with endovascular repair of AEFs. Patients who undergo open repair have a higher rate of perioperative complications including bowel obstruction, mediastinal abscess, and acute renal failure. Patients in the endovascular group have fewer complications, most of which are related to persistent sepsis. In addition, the mean hospital stay for the open group is significantly longer. However, the patients in the endovascular group are more likely to acquire late infection-related complications, which will lead to open conversion. The open repair group has a longer median survival time after hospital discharge.

Complications

Perioperative complications occur in about 50% of all patients. For those undergoing open surgical

repair, complications consist of acute renal failure necessitating hemodialysis, bowel obstruction necessitating exploratory laparotomy and lysis of adhesions, mediastinal abscess necessitating multiple operative drainages, multisystem organ failure, and pancreatitis. Complications in the endovascular group are related to persistent sepsis (infection due to enteric contamination), and this leads to multisystem organ failure. There are no procedure-related major complications. Rebleeding, when it is present, appears after a free interval of 2 weeks or longer and is successfully treated with either endovascular or open surgery. Overall postoperative complications, apart from rebleeding, are limited to deep femoral vein thrombosis.

The overall endovascular group, as well as those who underwent endovascular repair of secondary infrarenal AEFs, had a lower incidence of perioperative complications and a shorter length of hospital stay compared with those who underwent open repair. Furthermore, nearly all of the endovascular patients could be discharged home, whereas most patients who underwent open repair required placement in skilled nursing facilities. Endovascular aneurysm repair has been described to be technically feasible and suggested to be the preferred first-line treatment for AEFs with no sign of sepsis. On the other hand, some case reports describe recurrence of infection or fistula after successful endovascular aneurysm repair-treated acute bleeding from AEFs, necessitating open surgical treatment.[63,64]

Postoperative Management Follow-up

Patients, who were clinically well without evidence of ongoing blood loss and without evidence of sepsis, were started on oral feeding on postoperative day 1. Select patients with decreasing hematocrits undergo postoperative endoscopic evaluation to confirm cessation of AEF bleeding. All patients need to be placed on lifelong suppressive oral antibiotics after their endovascular repairs; these were organism specific in the setting of positive perioperative cultures. Follow-up CT scans are obtained at 1, 6, and 12 months and annually thereafter.

It is interesting to note that the median overall survival after discharge was longer in the open surgery group, but this is most likely related to the number and severity of the comorbidities in the endovascular group, in addition to their significantly older age. Given these data, endovascular repair as a palliative procedure for select patients is valuable by allowing for relatively short hospital stays and discharge to home.

Conclusion

AEF is a life-threatening entity, not only because it is associated with high morbidity and mortality but also because it often poses a diagnostic dilemma. Knowing that primary AEFs are a rare complication of aortic aneurysms and a rare cause of gastrointestinal bleeding, a delay in diagnosis and treatment carries a very high mortality rate. When there is a high index of clinical suspicion for AEF, any patient in whom a readily identifiable source of gastrointestinal hemorrhage has not been found should be considered to have a primary AEF unless proven otherwise. Early surgical intervention improves the chances of successfully managing this rare, lethal, clinical conundrum. The treatment of AEFs has improved in recent years, but despite the multiple surgical techniques reported, many of the patients suffering from AEFs do not survive or are left debilitated after treatment.

Endovascular repair is an efficient and safe method to stabilize patients with life-threatening AEFs. This minimally invasive surgical technique when combined with broad spectrum antibiotics and drainage (or diversion when necessary) has obvious advantages over open surgery in a field of ongoing massive bleeding that may be complicated by previous surgery or radiation therapy. At-risk patients for open surgery are good candidates for endovascular repair. However, in this group of patients with severe comorbidities the risk of rebleeding is high and further interventions must be considered. According to the opinion of many vascular surgeons, endograft repair should be seen as a temporary measure of treating patients with AEFs. It can be considered a bridge to a more definitive operation at a later time or as a palliative therapy for a patient who may not tolerate surgery.

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