

Abdominal Vascular Catastrophes



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KEYWORDS

- Mesenteric ischemia • Ruptured abdominal aortic aneurysm • Aorto-enteric fistula
- Gastrointestinal bleeding

KEY POINTS

- Mesenteric ischemia (MI) has a variety of causes, each with its own historical clues to assist in diagnosis.
- Early CT angiography without waiting for administration of oral contrast should be pursued in suspected cases of MI.
- Unexplained hypotension, syncope, or ecchymosis should prompt consideration of ruptured abdominal aortic aneurysm (AAA).
- Any amount of gastrointestinal (GI) bleeding in a patient with a history of AAA or AAA repair is an aortoenteric fistula (AEF) until proved otherwise.

INTRODUCTION

Abdominal vascular catastrophes are uncommon yet frequently fatal processes that are of great interest to emergency physicians because rapid recognition and initiation of definitive treatment are essential to prevent long-term morbidity and mortality. The list of abdominal vascular catastrophes is broad, but the focus of this article is on MI, AAA, and AEF.

MESENTERIC ISCHEMIA

Introduction

Acute MI continues to remain an elusive disease to diagnose despite clinicians being taught in medical school and residency about the classic pain out of proportion with examination presentation. Although a rare case of abdominal pain, with an annual incidence of 0.09% to 0.2% per year and approximately 1% of acute abdomen

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hospitalizations,^{1,2} this is offset with a 60% to 80% mortality within the first 24 hours.³ It is imperative that there is no delay in diagnosis because delays in diagnosis lead to increased mortality and morbidity in terms of amount of bowel requiring resection. The presentation of patients with MI is usually nonspecific with a falsely reassuring objective abdominal examination, which can lead to a false sense of security because the late findings of this disease process (absent bowel sounds, positive fecal occult blood test, focal/generalized peritonitis from visceral ischemia, elevated lactate, hypotension, fever, and so forth) have not evolved. In general, a high degree of clinical suspicion should be based on a combination of history, examination, laboratory results, and imaging studies to arrive at the diagnosis of acute mesenteric ischemia.

Anatomy

The abdominal aorta gives off 3 major branches to the intestines (foregut, midgut, and hindgut), which are the celiac artery (CA), superior mesenteric artery (SMA), and inferior mesenteric artery (IMA).⁴ The CA perfuses the foregut (distal esophagus to second portion of duodenum). Acute MI of the foregut is rare because the CA is a short, wide artery with good collateral flow. The SMA perfuses the midgut (duodenum to distal transverse colon), which encompasses nearly the entire small bowel and two-thirds of the large bowel. This is the most common embolic site of MI due to favorable takeoff angle (approximately 45°) from the aorta. The IMA perfuses the hindgut (transverse colon to rectum) and is rarely the sole vessel involved in MI. Collateral circulation from the CA or IMA generally allows sufficient perfusion in reduced SMA occlusion states.

Pathophysiology

In addition to the abdominal aortic anatomy, it is important to understand how the bowel layers are affected by MI, starting from the innermost to outermost layers (mucosa, submucosa, muscularis, and serosa). Early in the course of MI, the furthest layer from the blood supply (mucosa) is the first to become ischemic and is the reason for extreme pain, which is visceral in origin. Because the outer structures (muscle and serosa) have not become ischemic, however, there is minimal irritation of the parietal peritoneum when the examiner indents down against the serosa and the external layers of the bowel. Hence, there is pain out of proportion with the examination early on in the disease process. Over a period of hours, the muscularis and serosal layers become ischemic and infarct, leading to peritoneal irritation and guarding with rigidity. At this point, the pain is in proportion with the examination. It is also important to consider that between the early and late presentations (discussed previously), there is a deceptive pain-free interval of approximately 3 to 6 hours caused by a decline in intramural pain receptors from hypoperfusion.⁵

Etiology

MI can be classified as acute versus chronic or as occlusive versus nonocclusive. The following are the major 4 causes of acute MI⁵:

- Acute arterial emboli – the most frequent cause of MI, accounting for 40% to 50% of cases; the embolus usually lodges in the SMA.³ The proximal branches of the SMA (jejunal and middle colic arteries) are usually preserved because the embolus lodges 3 cm to 10 cm distally from the SMA takeoff, where the artery tapers off and is just after the first major branch of the SMA (the middle colic artery). As a result, the proximal small and large bowels are usually spared.⁶ Due to poorly developed collateral circulation, the onset of symptoms in cases of emboli is usually severe and dramatic pain.⁴ When the bowel becomes ischemic, it has a

propensity to empty itself, leading to vomiting or diarrhea, so-called gut emptying. This is one of the reasons that MI is often misdiagnosed as gastroenteritis. Common predisposing factors include atrial fibrillation, cardiomyopathy, recent angiography, and valvular disorders, such as rheumatic valve disease.⁵ One-third of patients have had a previous embolic event, such as an embolic renal infarct, embolic stroke, or peripheral arterial embolus.

- Acute arterial thrombosis – patients with long-standing atherosclerosis may develop plaque build-up at the origin of the SMA, a site of turbulent blood flow. This subsequent stenosis may lead to long-standing postprandial pain (intestinal angina) and food fear with resultant weight loss. These symptoms of chronic MI can be seen in up to 80% of patients who develop arterial thrombosis. If the plaque acutely ruptures or the stenosis reaches a critical level, patients may present with acute pain, similar to those with arterial emboli.^{3,6,7}
- Mesenteric venous thrombosis (MVT) – generally found in patients with an underlying hypercoagulable state; MVT accounts for 10% to 15% of cases. Patients typically present with less severe and more insidious pain than those with arterial occlusion.⁵ A majority of patients present after more than 24 hours of symptoms. In 1 study, the mean symptom duration was 5 to 14 days, with many patients experiencing pain for 1 month prior to diagnosis.³ Predisposing risk factors include malignancy, sepsis, liver disease or portal hypertension, sickle cell disease, and pancreatitis.^{3,7} Many patients have heritable hematologic disorders, including protein C and protein S deficiency, antithrombin III deficiency, and factor V Leiden mutation. One-half of patients with MVT have a personal or family history of venous thromboembolism.
- Nonocclusive MI (NOMI) – this type of MI occurs in 20% of patients due to failure of autoregulation in low-flow states, such as hypovolemia, potent vasopressor use, heart failure, or sepsis.^{3,6} The underlying ischemia from splanchnic vasoconstriction can further lead to hypotension from endogenous substances, perpetuating a vicious cycle.^{4,5} This accounts for the extremely high mortality rate, usually due to the poor health of the affected population, with multiple comorbidities, combined with the difficulty in treating the primary cause of diminished intestinal blood flow. Patients who present with abdominal pain postdialysis may have NOMI secondary to intradialytic hypotension, leading to vasospasm.⁸

Clinical Findings

The presentation of MI is typically acute severe abdominal pain with a paucity of physical examination findings. There is a widely variable range of performance characteristics of the history and physical examination, which underlines the diagnostic challenge.⁷ History and physical examination findings, such as acute abdominal pain, pain out of proportion, peritoneal signs, guaiac-positive stool, acute abdominal pain, heart failure, and atrial fibrillation, have a wide range of sensitivities and are frequently absent.⁹ Therefore, clinicians should be vigilant in considering MI in the differential diagnosis of abdominal pain of unclear etiology. Assessing a patient's pretest probability for disease, actively searching for known risk factors, and adding in clues based on a patient's history and physical examination findings are an important process for wary clinicians. Early and aggressive imaging based on this process has been demonstrated to decrease overall mortality from MI.¹⁰

Laboratory Studies

Numerous laboratory abnormalities have been described in MI, including elevated amylase, lactate dehydrogenase, large base deficit, and metabolic acidosis. None

of these findings is sensitive or specific for MI. Troponin I levels are often elevated. This finding is not specific for MI and has been shown to lead to delays in definitive care of these patients and inappropriate cardiology consultations.^{11,12} Common laboratory abnormalities, such as hemoconcentration, leukocytosis, and high anion-gap metabolic acidosis with elevated lactate (specifically D-lactate), are neither sensitive nor specific enough to be diagnostic and usually late findings.

Diagnostic biomarkers are a tool that should bear a high sensitivity and specificity, especially in MI, where early symptoms are nonspecific and mortality rises with delayed or missed diagnosis. Many laboratory tests have been studied in AMI, including D-lactate, intestinal fatty acid-binding protein, glutathione S-transferase, ischemia-modified albumin, and D-dimer. Although some have shown promising early results, none is sufficiently well established to either make or exclude the diagnosis of AMI. The serum marker that practicing clinicians are most familiar with is lactate. Although MI mortality is associated with high lactate serum values, a normal serum lactate value does not exclude AMI.¹³ Early in the disease process, lactate is generally normal as it travels through the portal venous system to the liver, where it is converted into glucose via the Cori cycle. As the ischemia load increases and the liver is not able to keep up with the demand, lactate spills over into the systemic circulation, where it eventually increases in late stages.

Imaging Studies

Various imaging methods have been studied and used in the diagnosis of MI, including lower GI endoscopy, radionuclide imaging, peritoneal fluid analysis, MRI, and peritoneoscopy. Imaging that is insensitive or low yield should be avoided, and any imaging that is performed should be pursued in as expeditious a manner as possible, given the time-sensitive nature of the disease. It has been shown that a multidisciplinary approach to suspected cases of AMI with streamlined protocols and early involvement of consultants can have an impact on overall mortality.¹⁴ The following are common diagnostic modalities often described:

- Plain radiographs: the findings on a plain abdominal radiograph are usually nonspecific (ie, small bowel distention with air-fluid levels or ileus), and 25% of patients may have normal findings.³ Patients with normal plain radiographs have a lower mortality rate, presumably because the findings that are visible on plain radiographs are late findings seen in more advanced disease. Characteristic findings, such as thumb printing or thickening of bowel loops, occur in less than 40% of patients.³ Later findings, such as air in the bowel wall (pneumatosis intestinalis) and portal venous system, are ominous signs portending a poor prognosis.
- Ultrasound: the use of ultrasound to detect significant stenosis (>50%) in mesenteric vessels has been shown and has a role in chronic MI, but the role of ultrasound in making the diagnosis of acute ischemia is less well established. This likely is due to limited operator experience in AMI and the abnormality in patient bowel gas patterns that often accompanies AMI, which make visualization of the mesenteric vessels more difficult.
- CT scan: CT is the most commonly used diagnostic tool in suspected MI; the initial sensitivity was 64% but has now improved to 93% with the use of dynamic contrast-enhanced CT.^{15,16} The addition of multidetector row CT (MDRCT) technology has further improved results (**Fig. 1**).^{17,18} The use of MDRCT angiography does not require oral contrast,¹⁶ which has been shown to increase time to image acquisition to 2 to 3 hours,¹⁹ a potentially lethal delay in cases of suspected MI.

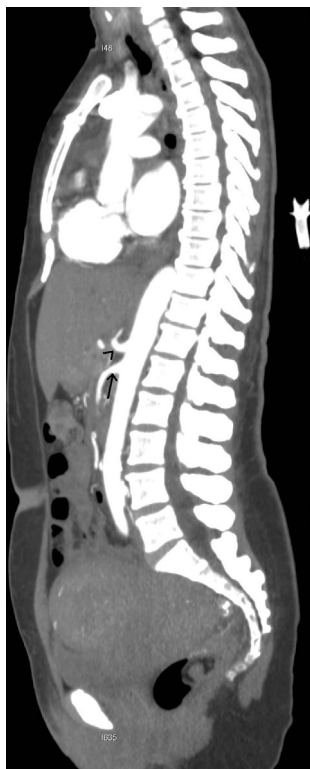


Fig. 1. Sagittal reconstruction of MDRCT angiography of normal celiac artery (*arrowhead*) and normal SMA (*arrow*).

- **Angiography:** angiography was once the diagnostic gold standard in work-up due to its high accuracy and therapeutic role; today it is used primarily as a confirmatory tool when noninvasive radiological studies do not produce conclusive results.^{20,21} Catheter-based therapy and vasodilation still play a large role in management, especially in those patients who are deemed too risky for open surgical techniques. In addition, patients who undergo successful revascularization procedures may still require intra-arterial vasodilators to treat associated vasospasm.²²
- **Laparoscopy:** depending on the institution, the availability of experienced radiologists to interpret CT angiograms and endovascular specialists to perform diagnostic and therapeutic angiography may be limited. In addition, acute renal failure from MI or those with known contrast allergy may prohibit obtaining a contrast study.^{23,24} Furthermore, a CT scan may not show vascular/intestinal pathologies in patients with a high pretest probability of MI.^{25,26} As a result, a diagnostic laparoscopy can fill this diagnostic gap. Studies have shown that the mean time between admission and diagnostic laparoscopy (10.2 h) was significantly shorter in patients who underwent successful revascularization and in those who survived with or without developing short bowel syndrome.²⁵

Treatment

Treatment of AMI should be initiated while the diagnostic evaluation is commencing. Treatment often requires a multidisciplinary approach involving general

and vascular surgeons as well as interventional radiologists. Aggressive fluid resuscitation should be started to correct fluid deficit and metabolic derangements. Broad-spectrum antibiotics are generally given as well. Early surgical consultation is warranted, even before definitive testing is performed, especially in cases of high pretest probability. The presence of peritoneal signs is usually an indicator of late stages of the disease requiring emergency laparotomy and may obviate any confirmatory imaging.

Once a diagnosis is established, surgical treatment of the underlying cause should be performed (ie, embolectomy, thrombectomy, endarterectomy, or bypass). Anticoagulation should be started, in consultation with the treating surgeon. An important part of the postsurgical care involves reducing the profound vasospasm that accompanies AMI. This is typically accomplished through intra-arterial papaverine infusion via an indwelling catheter in the SMA. A growing area of research involves minimizing ischemia-reperfusion injury.

Summary

MI is a vascular emergency, which all emergency physicians must consider early in their abdominal pain differential. It continues to remain a diagnostic challenge, and any delay in diagnosis can contribute to the increases in the already high mortality rate. Clues to the diagnosis should be sought for in the patient history (Table 1). Although the underlying cause varies, early diagnosis and prompt effective treatment can lead to improved clinical outcome. Time is bowel, so if there is a high clinical suspicion for MI, surgical and interventional radiology consultants should be involved early and in parallel with an expeditious diagnostic evaluation.

ABDOMINAL AORTIC ANEURYSM

Introduction

An AAA in itself is a hallmark emergency medicine presentation, where it is a ticking time bomb if not recognized because patients are asymptomatic until it becomes painful as it expands until it ruptures. Once ruptured, overall mortality is as high as 90%; even those with treatment have a 40% to 50% survival.²⁷ The classic presentation consists of abdominal/flank pain, hypotension, and a pulsatile abdominal mass, but this is only present in 50% of all cases at best.

Anatomy/Pathophysiology

A true aneurysm is a dilatation of all 3 arterial layers (intima, media, and adventitia) through a degenerative process that remains unclear but involves the degradation of the media, where elastin is normally found. As a result, the aortic wall becomes more susceptible to influences of high blood pressure. The aorta varies in size by

Table 1 Historical clues in suspected causes of mesenteric ischemia	
Etiology	Historical Clue
SMA embolus	One-third have prior embolic event
SMA thrombosis	80% Have history of intestinal angina
MVT	One-half have personal or family history of deep vein thrombosis/pulmonary embolism
NOMI	More commonly seen in dialysis patients

age, gender, and body habitus, with the average diameter less than 2.0 cm, but, in general, anything above 3.0 cm is considered an aneurysm.²⁸ The risk of rupture increases with the size and rate of expansion.²⁹

Most AAAs originate in the infrarenal aorta, below the takeoff of the renal vessels. In this area, the diameter of the aorta is decreasing and contains a lesser proportion of elastin. Although this is the most common location of AAAs, they may also occur in the suprarenal, pararenal, and juxtarenal areas. These anatomic variations are important when discussing patient candidates for endovascular aortic repair (EVAR), discussed later.

Causes/Risk Factors

Although the degenerative process remains unclear, the following are well-defined risk factors³⁰ that contribute to AAAs:

- Smoking
- Hypertension
- Male gender
- Connective tissue disorder (ie, Marfan syndrome and Ehlers-Danlos syndrome)
- Atherosclerosis
- Infection/arteritis

Clinical Findings

The clinical manifestation of an AAA varies considerably, depending on the location of rupture (**Box 1**). Intraperitoneal rupture typically manifests as sudden death and rarely survives to reach medical attention. Retroperitoneal bleeds may tamponade off temporarily, allowing a patient to present for medical care. In 75% of cases, acute severe pain is the most common presentation of rupture, where the location of pain varies based on the site of rupture.³¹ Those close to renal vessels have flank pain leading to possible renal colic mimicry, whereas those anterior cause abdominal pain and posterior cause back pain. Once a rupture stabilizes, the pain may subside, leading to a false sense of security for both patient and physician. Other uncommon presentations of AAA rupture include radicular femoral/sciatic pain due to nerve compression from the hematoma, acute inguinal hernia from sudden increases in intraperitoneal pressure, and acute high-output heart failure or massive leg swelling from rupture into the inferior vena cava (aortocaval fistula). Unexplained hypotension or syncope, even transient, in a patient with risk factors for ruptured AAA should prompt consideration of the condition. This is evident because it is considered part of the differential of a hypotensive patient when performing the Rapid Ultrasound for Shock and Hypotension examination. Even without rupturing, AAAs can cause subacute flank,

Box 1

Location of rupture in abdominal aortic aneurysm (in decreasing frequency)

- Retroperitoneal
- Intraperitoneal
- Vena cava (aortocaval fistula)
- GI tract (AEF)

abdominal, or back pain due to rapid enlargement and compression of the surrounding structures.

The physical examination on these patients may be unrevealing but can offer clues to the diagnosis. Although insensitive, 50% of patients with an AAA have a pulsatile mass palpable in the epigastrium.³² Besides an abdominal examination, a full vascular examination palpating major pulses (radial, carotid, femoral, and popliteal) should be done to look unequal pulses that can hint to acute aortic syndromes. Although ecchymotic signs, such as Grey Turner (flank), Cullen (periumbilical), Fox (inguinal ligament), and Bryant (scrotal) signs, are neither sensitive nor specific for an AAA, unexplained ecchymosis should always prompt consideration of this vascular emergency.

Imaging Studies

Bedside ultrasound has emerged as the test of choice when screening patients for AAA (Fig. 2). Ultrasound has a 98% sensitivity in fasted patients undergoing screening, and although bowel gas and body habitus can hinder the examination, this is less of an issue in imaging the larger aneurysms that are likely to present ruptured. If a patient with a known history of AAA arrives unstable with symptoms consistent with rupture, however, no confirmatory diagnostic tests are necessary and the patient should be transferred to an operating room expeditiously.

Abdominal CT imaging is generally advised for hemodynamically stable patients, although ultrasound should be performed immediately in patients with high clinical suspicion to aid in triage and to speed disposition. In addition to assessing for alternative conditions, CT provides important anatomic information about the AAA that may be important in surgical planning for open and closed (ie, endovascular) surgical approaches (Fig. 3). Although contrast is not required, its administration is helpful to obtain more aortic detail for preoperative planning and to ascertain whether the patient is a candidate for EVAR. Signs of rupture on CT include retroperitoneal hematoma, free intraperitoneal blood, an indistinct aortic wall, and loss of the normal fat plane around the aorta. Signs of impending rupture or an unstable aneurysm also may be seen and include layering of hematoma within the aorta (crescent sign), breaks in the calcification of the wall, and blebs or other irregularity within the wall.

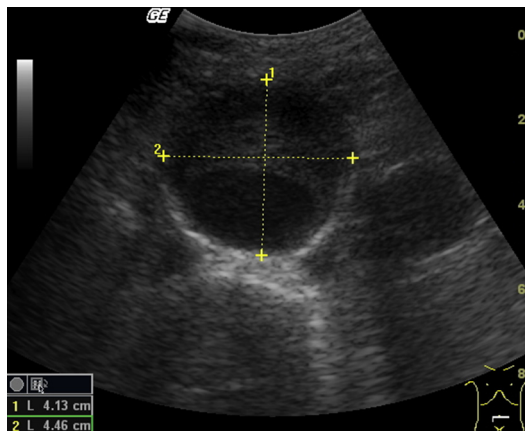


Fig. 2. Bedside ultrasound showing 4.1-cm × 4.5-cm AAA.

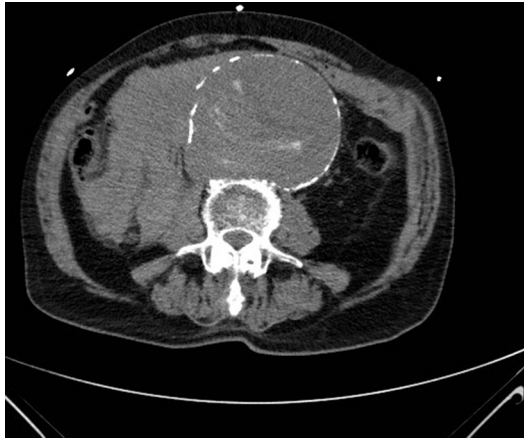


Fig. 3. CT of large AAA – axial slice of abdominal CT scan showing large, heavily calcified, and thrombosed AAA. The calcification exhibits significant discontinuity in the right posterolateral area and signs of retroperitoneal hematoma, suggestive of rupture.

Treatment

Those presenting in clinical shock require tandem resuscitation with bedside diagnosis if there is no known history of AAA. Initial misdiagnosis is common, occurring in approximately 40% of cases, where the most common incorrect diagnoses are renal colic, myocardial infarction, and diverticulitis.

Obtaining 2 large-bore intravenous (IV) lines with uncrossmatched type O blood immediately made available is key. Central access with a sheath introducer or multi-lumen access catheter should be weighed against possible delay in transport to an operating room. In anticipation for the operating room, 6 to 10 units of packed red blood cells, type and crossmatched, should be requested as well as fresh frozen plasma and platelets that may be required during resuscitation. Depending on the institution, implementing a massive transfusion protocol, as well as activating the patient as a trauma, may speed along the process of obtaining an OR room with a surgeon who is ready to go. Resuscitation effort should be focused on controlled volume resuscitation targeting a systolic blood pressure of 80 mm Hg to 90 mm Hg, analogous to patients with penetrating torso trauma.³³

EVAR has become the mainstay for elective repair of AAAs, with 2 multicenter randomized controlled trials showing a 3-fold reduction in mortality.^{19,34} Although randomized trials have not shown improved mortality rates for EVAR in the setting of acute rupture, it seems that perioperative morbidity is decreased and thus EVAR is favored for anatomically suitable patients.³⁵ The main criterion for EVAR is an adequately long aneurysm neck to allow seating of the graft without occlusion of the renal arteries, which is met in 70% of AAAs.³⁶ Although infrarenal aneurysms are preferred (especially in the emergent use), suprarenal and pararenal aneurysms are amenable to EVAR with custom grafts.

Summary

Ruptured or symptomatic AAA is a fatal and time-sensitive condition that emergency physicians should be familiar with and ready for, where timely diagnosis and appropriate resuscitation with operative team management can mean the difference between life and death.

AORTOENTERIC FISTULAS

Introduction

Development of an AEF is a life-threatening and devastating cause of upper GI bleed, which can be difficult to diagnose and treat. Although rare, it is most commonly seen as a delayed complication of aortic reconstruction.

Pathophysiology

The disease is divided into 2 types – primary AEF and secondary AEF. Although uncommon, primary AEF occurs when a large, previously untreated aneurysm erodes de novo into the adjacent bowel. This is often diagnosed unexpectedly during exploratory laparotomy. The third portion of the duodenum, fixed retroperitoneally and in proximity to the descending aorta, is the bowel segment most vulnerable to this. The nidus for this process starts with ischemia and subsequent necrosis of the intestinal wall as a consequence of repetitive traumatic pulsations of an adjacent aortic aneurysm. Subsequent rupture of an expanding aneurysm or perforation of the aorta as a result of contamination with GI contents results in the formation of a communication with the bowel and the potential for rapid exsanguination. Less commonly encountered conditions that may lead to primary AEFs include syphilis, tuberculosis, mycotic infection, and collagen vascular disease, where the chronic inflammation leads to aortitis, erosion, and formation of the fistula. In the absence of treatment, the mortality rate is almost 100%. With surgical intervention, survival ranges from 18% to 93%.

In contrast, secondary AEF occurs as a complication of aortic reconstructive surgery. An estimated 80% of secondary AEFs affect the duodenum, mostly the third and fourth parts (the horizontal and ascending duodenum). As a result of advanced perigraft infection from chronic low-grade infection and the repetitive pressure on the intestine from aortic pulsations, fistulas are formed.

Clinical Findings

The typical symptoms of AEFs include acute abdominal pain, GI hemorrhage (melena, hematemesis, and dark blood per rectum), and sepsis. The most common clinical features of primary AEFs are upper GI bleeding (64%), abdominal pain (32%), and a pulsatile abdominal mass (25%), where these 3 features are concomitantly present in only 10% to 23% of patients. Patients with a secondary AEF usually present with 1 or more of the following clinical signs and symptoms: GI bleeding (80%), sepsis (44%), abdominal pain (30%), back pain (15%), groin mass (12%), and abdominal pulsatile mass (6%). With both primary and secondary AEFs, transient, self-limited, intermittent bleeding episodes (herald bleeds) often precede a major hemorrhagic episode by hours, days, or weeks. This is a result of a small fistula tamponaded by thrombus formation and bowel contraction around it.

Imaging Studies

Three modalities are available to assist in diagnosis: abdominal CT scan with IV contrast, esophagogastroduodenoscopy (EGD), and arteriography. Of the 3, CT scan offers superiority because it is less invasive, more readily available, and more expedient than the latter 2. In addition, it offers the advantage of being unlikely to dislodge the aortic thrombus. The CT may show abnormal communication between the aorta and the bowel, may disclose loss of continuity of the aneurysmal wall, and may demonstrate air bubbles in the aneurysm wall that are pathognomonic for the existence of a fistula.

An EGD with a water-soluble contrast material is usually considered second line but should be performed only on a hemodynamically stable patient. An AEF is usually present when there is leakage of oral contrast material from the disrupted bowel wall into the perigraft space. In addition to evaluating the presence or absence of an AEF, an EGD assists in ruling out other causes of upper GI bleeding, including varices, bleeding masses, and ulcers. A normal EGD or the finding of other pathology without stigmata of recent bleeding does not exclude an AEF, especially if there is a high index of suspicion.

Arteriography is of some value but rarely used in critically ill patients for diagnosis. Its true value lies in embolization therapy and stent placement.

Treatment

AEF requires definitive and emergent operative management, where various surgical modalities exist (graft excision and extra-anatomic bypass, in situ graft replacement, and simple graft excision or endovascular repair). The role of emergency physicians, in the perioperative phase, includes optimally resuscitating the patient with fluids and blood products while initiating broad-spectrum IV antibiotics to cover gram-positive, gram-negative, and enteric pathogens as part of sepsis management.

Summary

AEF is a life-threatening entity that is challenging to diagnose and carries high morbidity and mortality. Any patient who presents with any degree of GI bleeding and has a prior history of aortic aneurysm repair should be considered as having an AEF until proved otherwise.

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