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 MI[5]:

- 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