Abdominal Aortic Emergencies

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KEYWORDS

• Abdominal aorta • Dissection • Aneurysm • Aortic rupture • Acute aortic syndrome

KEY POINTS

- Acute aortic syndrome is a group of diagnoses, including aortic dissection, intramural hematoma, and penetrating atherosclerotic ulcer. These have similar risk factors, including hypertension and dyslipidemia, as well as comparable presentations.
- Aortic aneurysm can be a precursor to dissection and rupture. Close surveillance and risk factor modification are key to prevention of aneurysm progression.
- Aortic endoleak and aortoenteric fistula can be either primary processes or, more commonly, a postoperative complication after aortic repair. Although some endoleaks can be managed conservatively, aortoenteric fistulas are surgical emergencies.

INTRODUCTION

The abdominal aorta is the continuation of the descending thoracic aorta. It begins at the aortic hiatus of the diaphragm at the level of the twelfth thoracic vertebrae and ends on the body of the fourth lumbar vertebrae where it divides into the 2 common iliac arteries. The abdominal aorta is broadly subdivided into suprarenal and infrarenal segments at the level of the renal arteries. The wall of the aorta is composed of 3 layers (tunicae): the intima, media, and adventitia. Advanced age, as well as degenerative processes (and those factors that accelerate them), such as atherosclerosis, fibrosis, and calcification of the aortic wall, impair its elasticity.[1]

ACUTE AORTIC SYNDROME

Acute aortic syndrome (AAS) encompasses a constellation of conditions that have a similar presentation. These pathologic conditions include aortic dissection (AD), intramural hematoma (IMH), and penetrating atherosclerotic ulcer (PAU).

Each of these diseases is described in detail in the following sections. Common features of these conditions are discussed, including risk factors and classification systems used to categorize them.

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Definition

Abdominal aortic dissection

AD occurs when there is a tear in the aortic intima, leading to separation of the media and the intima. (See aortic dissection at: https://radiopaedia.org/cases/aortic-dissection.) This is often preceded by medial degeneration or cystic medial necrosis. There are 2 theories that describe the pathogenesis of AD. The first theory is that there is a primary tear in the intimal layer and, during a subsequent hypertensive attack, blood enters the media at the location of the intimal tear and dissects into the media. The second theory is that the catalyst to dissection is rupture of the vasa vasorum with the development of an IMH, and that the intimal tear occurs as a result of increased wall stress.[1]

AD can propagate in both an anterograde and retrograde manner. As an AD propagates in an anterograde fashion, it can spread to the iliac bifurcation. This usually occurs along the convexity of the aorta, which preferentially involves the left side of the aorta and can lead to occlusion of branching arteries (renal and brachiocephalic), leading to end-organ ischemia and necrosis.[1]

Dissection can be classified as hyperacute (<24 hours), acute (2–7 days), subacute (8–30 days), and chronic (>30 days). Of the 3 conditions that define AAS, abdominal AD is the most common.[2]

Intramural hematoma

IMH is defined as hemorrhage from the vasa vasorum into the media layer of the aortic wall in the absence of a demonstrable 2-lumen flow and primary intimal tear. IMHs comprise approximately 5% to 15% of AASs.[2] Whereas classic AD more commonly affects the ascending aorta, IMH more commonly involves the descending aorta.[3] Approximately two-thirds of IMHs degenerate into aneurysm or dissection, and IMH is often thought of as a precursor to AD.[2] IMH can lead to AD in 28% to 47% of patients and aortic rupture in 21% to 47% of patients.[1] Approximately one-third of IMHs resolve spontaneously.[2]

Of the conditions of AAS, the risk of aortic rupture is higher in patients with IMH and PAU than in those with AD.[1]

Penetrating atherosclerotic ulcer

PAU is defined as ulceration of an atherosclerotic plaque in the intimal layer of the aorta that extends into the media with rupture of the internal elastic lamina. Alternatively, PAU can be thought of as a localized dissection that is limited by extensive calcification associated with progressive atherothrombosis.[3] PAU makes up approximately 2% to 7% of AAS.[3] PAU is most commonly seen in patients with extensive atherosclerotic disease who are often 70 years or older. Complications of PAU include false aneurysm, aortoenteric fistula, AD, and aortic rupture. Ulcers with an initial diameter greater than 20 mm and a depth of greater than 10 mm are associated with a high risk of ulcer progression.[4]

Of the conditions of AAS, the risk of aortic rupture is higher in IMH and PAU patients than in those with AD.[1]

Causes and Risk Factors

Abdominal aortic dissection

One of the major risk factors for dissection is hypertension (prevalence ~70%), especially in patients with poorly controlled blood pressure despite multidrug therapy.[2] Chronic hypertension leads to intimal thickening, fibrosis, calcification, and extracellular fatty acid deposition, in addition to extracellular matrix degradation, elastolysis, and apoptosis.[1] This leads to intimal disruption and thickening. This cascade leads to necrosis of smooth muscle cells and fibrosis of elastic structures in the aortic wall, which can lead to both aneurysm and dissection.[1]