Thoracic Aortic Syndromes

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KEYWORDS

• Aortic dissection • Aortic intramural hemorrhage • Penetrating aortic ulcer

KEY POINTS

- Aortic dissection is an uncommon disease that often presents with varied and atypical findings suggestive of more frequently encountered conditions; therefore, it poses an exceptional diagnostic challenge to emergency providers.
- Mortality associated with aortic dissection is significant at presentation and advances with every hour the lesion is left untreated.
- Although almost all patients who have symptoms possibly caused by aortic dissection will not have aortic dissection, key features of the disease, including risk factors, pain characteristics, physical examination findings, and routine ancillary studies, allow clinicians to develop a rational approach to diagnostic testing.
- When the diagnosis is sufficiently likely to indicate definitive testing, computed tomography angiography is the advanced imaging test of choice in most centers, but transesophageal echocardiography and MRI may be appropriate alternatives in certain circumstances.
- Patients with diagnosed or strongly suspected aortic dissection require expeditious surgical evaluation, aggressive analgesia and anxiolysis, and treatment with rapid-acting, titratable agents to first lower heart rate, and then blood pressure, to specific targets.

INTRODUCTION

Aortic dissection (AD) is among the most immediately lethal diseases in medicine, with a mortality of 1% per hour,[1] and has effective temporizing medical therapies and a surgical cure. AD is, therefore, among the disorders of greatest interest to emergency physicians, yet is not diagnosed on its initial presentation in up to half of cases.[2–4] In fact, 1 expert asserts that "difficulty in diagnosis, delayed diagnosis or failure to diagnose are so common as to approach the norm for this disease, even in the best hands…"[5] This article explains why AD poses a diagnostic dilemma, proposes a strategy for its rational evaluation, and describes the principles of treatment.

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PATHOPHYSIOLOGY

AD occurs when the innermost layer of the aortic vessel wall is torn, creating a false lumen that transmits a longitudinal column of blood. It is sometimes referred to as a dissecting aortic aneurysm; however, this term is discouraged because it is both inaccurate and conflates AD with aortic aneurysm, a distinct clinical entity. AD is thought to result from the hydrostatic pressure accumulated as blood is pumped through the aorta, as well as movement of the aorta itself, with every cardiac cycle. Histologically, AD is associated with characteristic changes in the vessel wall known as medial degeneration. The former term, cystic medial necrosis, has fallen out of favor because the observed lesion demonstrates neither cysts nor necrosis.

Conditions that increase the pressure exerted by blood on the vessel wall predispose patients to AD. These include hypertension, pregnancy, stimulant use (eg, cocaine), weight-lifting, and pheochromocytoma. AD is more likely in conditions that weaken the vessel and accelerate medial degeneration, such as large-vessel vasculitides and congenital connective tissue disorders, including Marfan, Loeys-Dietz, Ehlers-Danlos, and Turner syndromes. Finally, AD may be caused by lesions of the aortic valve itself, such as bicuspid aortic valve, aortic valve instrumentation or aortic surgery, and syphilitic aortitis.

The Stanford classification designates type A dissections as lesions involving the ascending aorta, whereas type B dissections are confined to the descending aorta. Type A dissections are more common and much more dangerous, which drives differences in the therapeutic approach. Variants of AD include aortic intramural hemorrhage, which is a hematoma completely contained within the vessel wall, and penetrating aortic ulcer, which is a disruption in the vessel wall that usually leads not to dissection but to aneurysm. These lesions are both treated similarly to AD.

AD causes morbidity and mortality by several mechanisms. Type A dissections can progress proximally to cause pericardial effusion with tamponade, as well as acute aortic valve insufficiency. Both types of dissections can breach the outer adventitial layer of the vessel, leading to free rupture and exsanguination into the chest or abdomen. Most sequelae of AD, however, result from the false lumen extending across ostia of branch arteries, leading to acute ischemia of potentially any organ in the body.

CLINICAL FEATURES AND EPIDEMIOLOGY

AD is an uncommon disease, with prevalence estimates ranging from 3.5 to 6.0 per 100,000 patient-years in the general population.[6] Untreated, AD carries a devastating mortality of 40% on presentation and an additional 1% rate of death per hour, to a 1-year mortality of 90%.[1] In a center where postmortem CT is routinely performed on patients with out-of-hospital cardiac arrest of uncertain cause, AD was determined to be the cause in 7% of cases.[7]

Approximately 1 in 10,000 emergency department (ED) patients will have AD, a number so small that emergency providers may only see several cases in their career. Only one-quarter of patients with AD present with a combination of classic features (pain of sudden onset or ripping or tearing quality, blood pressure differential, and widened mediastinum on chest xray [CXR]); 1 in 25 patients diagnosed with AD has none of the classic features.[1] Furthermore, AD can cause myriad symptoms localizing to any organ system or body part, and each of these symptoms can be explained by more common conditions, often by more common dangerous conditions that quite reasonably establish the focus of care but ultimately turn out to be distractors.[8]

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