Acute aortic syndromes

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Abstract

Acute aortic syndrome includes a wide spectrum of aortic conditions such as classic acute aortic dissection, intramural haematoma, symptomatic penetrating aortic ulcers and traumatic aortic lesions. These result primarily from disruption of the inner wall layer, and involve thinning of the aortic wall, increased wall stress, progressive dilatation, evolution of intramural haemorrhage and possibly dissection and rupture. Chronic hypertension and connective tissue disorders are often implicated. Echocardiography, contrast-enhanced computed tomography and dynamic magnetic resonance imaging are used to establish the diagnosis. Aortic dissection is primarily classified according to anatomical characteristics: presence or absence of ascending aortic involvement is distinguished for prognostic and therapeutic reasons. In general, open surgery is indicated for dissection involving the ascending aorta, whereas medical management and/or endovascular stent-graft implantation is considered where the ascending aorta is spared. Pathology involving the aortic arch can be treated using a hybrid approach combining debranching with stent-graft implantation or branched or fenestrated stent-grafts. Stent-graft-induced remodelling seems to have long-term benefits in both complicated and uncomplicated distal dissection. In addition, long-term medical therapy to control hypertension and surveillance are paramount in all patients who have survived aortic dissection to reduce late complications, including recurrent dissection, aneurysm formation and rupture.

Keywords Acute aortic syndrome; aortic dissection; intramural haematoma; MRCP; penetrating aortic ulcer; stent-graft; TEVAR; thoracic endovascular aortic repair

Key points

- The incidence of acute aortic syndromes (AAS) is increasing
- Management of AAS should be based on low-threshold CT imaging
- Endovascular technology is encouraging in various types of aortic dissection
- Overall management of AAS should involve a multispecialty aortic team

Introduction

Acute aortic syndromes (AAS) are defined as emergency conditions with various manifestations of disruption of the intima and media of the aortic wall, leading to intramural haematoma (IMH), penetrating aortic ulcer or dissection of the medial layer and formation of a true lumen and a false lumen. Despite technological improvements and better patient care, aortic dissection remains a life-threatening condition and requires special treatment.[1]

The estimated incidence is 2.6–3.5 cases per 100,000 people per year. This may, however, underestimate the true incidence as hospital-based reports do not account for pre-admission deaths. Indeed, a prospective analysis of 30,412 middle-aged men and women with 20 years' follow-up reported 15 cases per 100,000 patient–years at risk of aortic dissection, with a 67.5% male preponderance. In individuals 65–75 years of age, the incidence may even be as high as 35 cases per 100,000 people per year.[2]

Pathology

An AAS occurs when a tear or an ulcer allows blood to penetrate from the aortic lumen into the media, or when a rupture of the vasa vasorum causes a bleed within the media. The inflammatory response to blood in the media may lead to aortic dilatation and rupture. Aortic dissection is defined as disruption of the medial layer provoked by intramural bleeding, resulting in separation of the aortic wall layers and subsequent formation of a true and a false lumen with or without communication.

Aortic IMH is considered to be a precursor of dissection, and originates from ruptured vasa vasorum within the medial wall layers. It results in aortic wall apoplexy that may provoke a secondary tear and classic aortic dissection. IMH is usually located in the descending aorta, is typically associated with hypertension, and can extend, progress or resorb. Deep penetrating aortic atherosclerotic plaques can lead to IMH, aortic dissection or perforation. Non-invasive imaging has recently elucidated this disease process, which often further complicates IMH.

Classification

The prime classification is based on the anatomical location of the intimal tear and degree of propagation of the false lumen. Aortic dissections are classified according to anatomical location by the Stanford and DeBakey classification.

DeBakey's nomenclature is based on the anatomical site of the intimal tear, and the extent of the resulting dissection. In a type I

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dissection, the intimal tear originates in the ascending aorta, and the dissecting haematoma extends past the origin of the left subclavian artery. Type II dissections are confined to the ascending aorta. Type III dissections begin after the origin of the left subclavian artery and extend distally. The fundamental distinction described by the Stanford classification is whether the dissection is proximal (type A) or distal (type B) to the origin of left subclavian artery. The Stanford classification is conceptually founded on prognostic grounds, and type B dissections often imply better prognosis than type A dissections (Figure 1).

Aortic dissection may also be classified according to the timing of diagnosis relative to the onset of symptoms: acute within 2 weeks, subacute within 2–8 weeks, and chronic beyond 8 weeks. Type B aortic dissection is traditionally seen as acute and chronic in relation to symptom onset; acute is within 2 weeks and chronic is >2 weeks. This classification has recently been revised to acute (<15 days), subacute (15–92 days) and chronic (>93 days).

Aetiology and pathogenesis

Any disease process undermining the integrity of the elastic or muscular components of the media predisposes the aorta to dissection, and degeneration of any aortic layer is a major predisposing factor in most non-traumatic aortic dissection (Table 1). Degeneration of the media from enhanced apoptosis is a feature of several hereditary connective tissue diseases, notably Marfan's and Ehlers–Danlos syndromes. In the absence of Marfan's syndrome, medial degeneration is usually minor in most cases of aortic dissection; it is nevertheless qualitatively

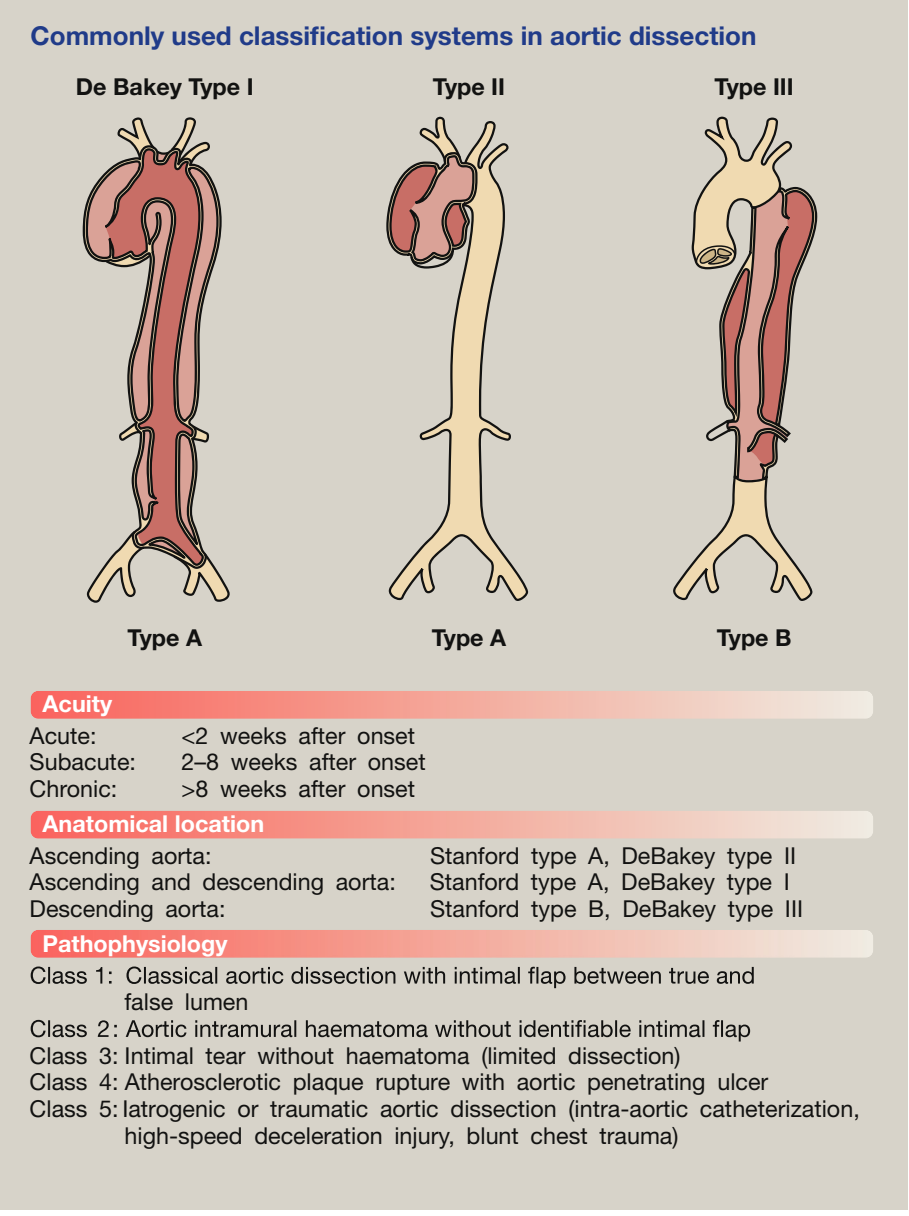

Figure 1

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