Acute Aortic Dissection

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60.1 Clinical History

A 55-year-old male was brought to the casualty with acute retro-sternal excruciating chest pain (radiating to medial aspect of left arm and back) and acute shortness of breath since 1 h. He was a chronic smoker and alcoholic. There was no history of hypertension or diabetes mellitus. The patient was diaphoretic with a pulse rate of 110 per minute, blood pressure of 108/90 mmHg, and respiratory rate of 24 per minute; he was hemodynamically stable. Since the ECG was suggestive of acute inferior wall myocardial infarction, it was decided to take up the patient for primary angioplasty. The baseline investigations were normal. He was given a loading dose of aspirin and clopidogrel and was shifted to the cardiac catheterization laboratory. About 20 min later, he developed excruciating pain in the right lower limb. Stenting of the left coronary was performed; however, his condition deteriorated and could be revived despite inotropic support.

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60.2 Autopsy Findings

The moderately enlarged heart was kept with the entire aorta. The ascending aorta was larger than the pulmonary artery. Adventitial hemorrhage and a "crackling" consistency were present over the ascending aorta. The hemorrhage also extended into the adventitia of the pulmonary trunk and the anterior right and left atrioventricular grooves. These changes on the external aspect were produced by an acute aortic dissection (AAD), involving the entire aorta (Stanford type I), affecting the greater curvature of the descending aorta. There was a transverse intimal tear (Fig. 60.1a), 4 cm in length in the ascending segment, 3.2 cm above the aortic annulus. The aortic valve was devoid of any congeni-

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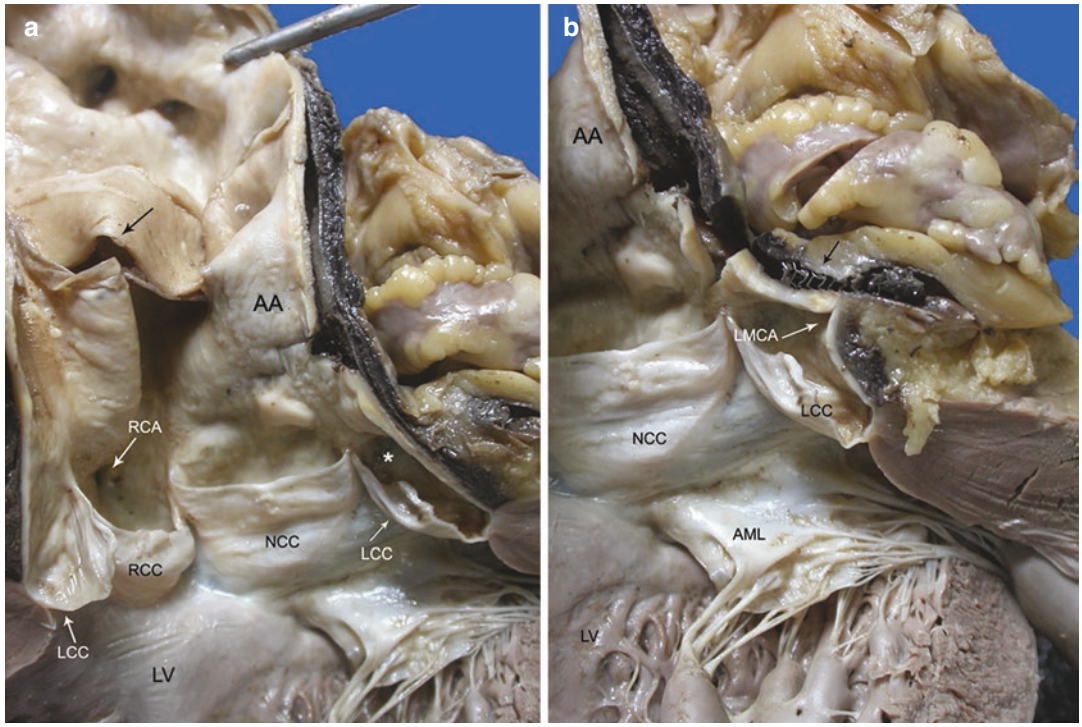

Fig. 60.1 (**a**) A transverse tear (white arrow) is seen in the intima of the ascending aorta AA, well above the sinutubular junction with presence of hemorrhage in the wall. * represents the ostium of the left main coronary artery LMCA, not visible in the image; (**b**) An additional cut reveals the presence of a stent (black arrow) in the false lumen of the dissected LMCA (*AML* anterior mitral leaflet, *LCC* left coronary cusp, *LV* left ventricle, *RCA* right coronary artery, *RCC* right coronary cusp, *NCC* coronary cusp)

tal anomalies. The ostium of the left coronary artery was narrowed and a stent was seen to be deployed into the false channel of the artery (Figs. 60.1b and 60.2a–c). The right coronary artery was surrounded by hemorrhagic epicardial fat (Fig. 60.1a); there was no dissection. There was extension of the dissection into the right brachiocephalic artery (0.8 cm), left common carotid artery (1.6 cm, Fig. 60.1a), left subclavian artery (1 cm), and also into the common iliac arteries. Features of acute myocardial infarction and aortopathy were not seen. There were pulmonary edema and smoking-related respiratory bronchiolitis.

Cause of Death: Acute myocardial ischemia following AAD.

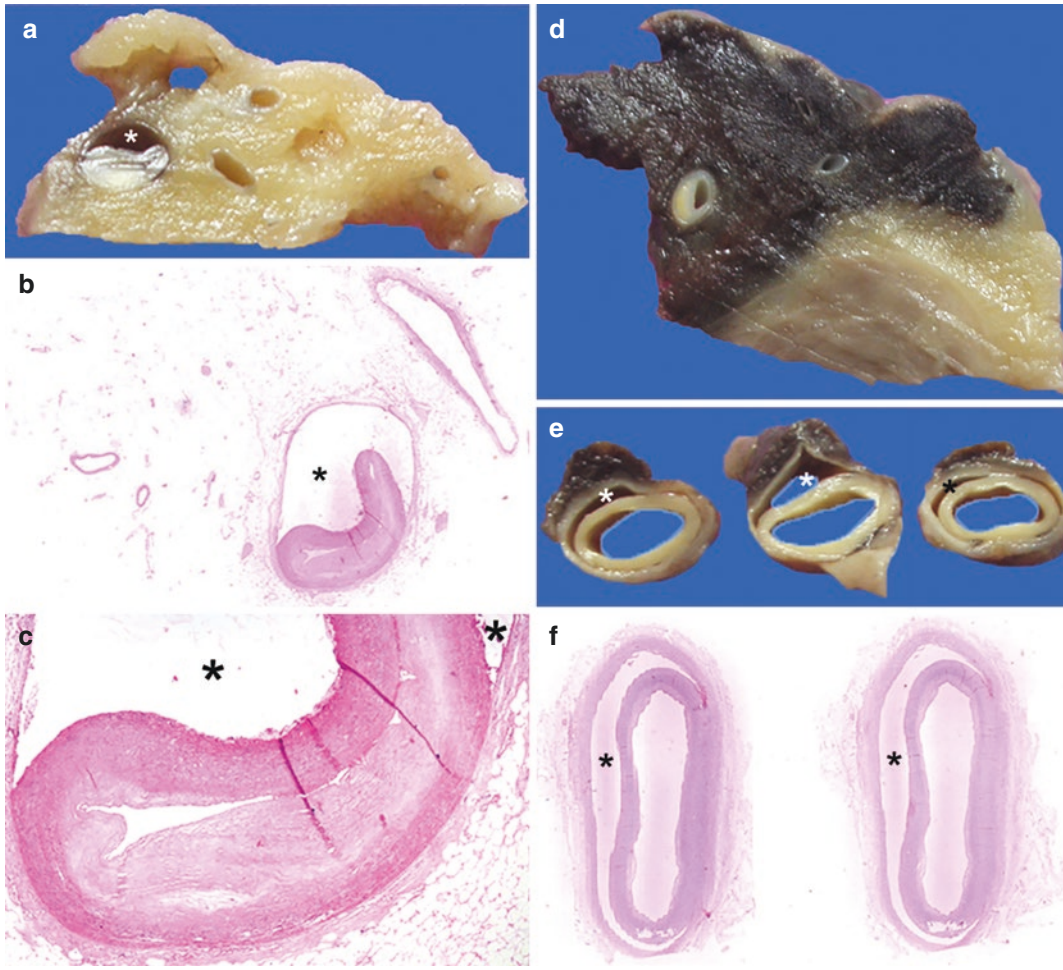

Fig. 60.2 Dissection seen in (**a**) the cut surface of the left anterior descending artery; (**b**) scanned H&E stained section, and (**c**) low power (H&E × 100) of the artery. Note eccentric non-critical plaque atheromatous plaque and slit-like true lumen; (**d**) the right coronary artery is surrounded by hemorrhagic epicardial fat; dissection of the left common carotid artery as seen in (**e**) serial slices and (**f**) scanned H&E stained section. * is in the false channel

60.3 Discussion

The differential diagnoses of acute severe chest pain in adults must include acute aortic syndrome (AAS) in addition to the more common acute coronary syndromes (see Chaps. 24–27) and pulmonary thrombo-embolism (see Chap. 68). AAS is a well-established term that includes 3 uncommon (3.5–6.0 per 100,000 patients/year), but devastating aortic disorders of acute aortic dissection (AAD), penetrating atherosclerotic ulcer (see Chap. 58), and intra-mural hematoma. The common binding factor for these lesions is the clinical presentation of aortic pain (with or without radiation), pulse deficits, blood pressure variations, and/or end-organ ischemia; the pain is caused by disruption of the media that develops often due to long-standing alterations in the aortic wall. In this instance, AAS was initiated by a transverse almost circumferential intimal tear in

the ascending aorta, which allowed the "high pressure" blood to enter and split the aortic media at the junction of its inner two-thirds and outer one-third. This is the defining feature of classic aortic dissection (AD) that is responsible for 85–95% of cases of AASs. It results in the formation of double-barreled aorta with its true lumen and a false channel within the wall; the latter may propagate within the wall for a variable distance taking an anterograde (most commonly) or a retrograde course. The propagation may re-enter the true lumen through another tear (intimal re-entry or exit tear) or may result in aortic rupture. With reference to the duration of symptoms, the ADs are hyper-acute (less than 24 h, as in the present case), acute (within 1 week), sub-acute (8–30 days), and chronic (for more than a month).

The estimated worldwide incidence of AAD is around 4.8 per 100,000 individuals per year, which may increase up to 30 in persons above the age of 65 years. However, the true incidence remains unknown as some cases would have had sudden cardiac death and may not have been necessarily autopsied. It occurs more often in males and has a chrono-biologic rhythm pattern with its more frequent occurrence during winter, in the early hours of the morning, and on Mondays! Conditions that increase the wall stress or induce abnormalities of the aortic media are the main causes of non-traumatic and non-iatrogenic AAD. Hypertension (often poorly controlled) is the major risk factor, seen in almost two-thirds of the patients with AAD; other factors include syndromic/non-syndromic genetic abnormalities, congenital aortic valvular abnormalities (see Chaps. 13 and 14), inflammatory diseases, smoking, and drugs like cocaine or amphetamines. In the non-inflammatory conditions, there are very often moderate to severe alterations in the aortic media (aortopathy, see Chap. 61) that permits the catastrophic disruption.

Aortic dissection is classified according to the extent of involvement. In the Stanford system, presence and absence of dissection of the ascending aorta constitute Type A and Type B dissections, respectively. Type A AAD is more frequent (60–70%, incidence rate of 1–3 per 100,000 individuals per year) and affects younger individuals, while Type B AAD has a rate of occurrence of 1.6 per 100,000 individuals per year, affecting the elderly hypertensive patients. The other classification was devised by DeBakey Type I where there is dissection of the ascending aorta with variable extension into the remaining segments; Type II with dissection limited to the ascending aorta; and Type III where there is involvement of the descending aorta distal to origin of the left subclavian artery with dissection in the thoracic (Type IIIa) or thoraco-abdominal (Type IIIb) segments. In the case presented, there was dissection of the entire aorta (Stanford Type A or DeBakey Type I). Our patient did not have hypertension. The only risk factor was smoking, and despite such extensive involvement, there were hardly any medial changes seen on histology. There was large intimal tear in the ascending aorta, which is seen in 65% cases of AAD. Such tears are located in the right lateral wall within 3 cm of the sinutubular junction. Tears in the descending aorta are present in close proximity to the ligamentum arteriosum (20% of the cases). Both areas are sites of greatest hydraulic stress. Arch (10%) and distal portions of the descending aorta (5%) are the less common sites for such tears.

Acute chest pain, which has been variously described as instantaneous, severe, sharp, stabbing, tearing, ripping, or lancinating (aortic pain) is the most common mode of presentation seen in almost 80–90% of the patients. Radiation of the pain to the neck indicates involvement of the ascending aorta, while radiation to the back or abdomen is suggestive of dissection in the descending aorta. But sometimes, the pain may be absent. Since the blood pressure in the false channel is high and constant, there is dynamic or static compression of the true lumen leading to malperfusion of the aortic branches, resulting in end-organ ischemia or infarction. This results in a variety of non-cardiac symptoms and signs. The cardiac complications include aortic insufficiency, myocardial dysfunction, and hemopericardium due to rupture within the pericardial cavity. There can also be an extravasation of blood onto the pulmonary arterial adventitia (pulmonary sheath hematoma) or within the wall (intra-mural hematoma) or even fistulous connections with features of right ventricular failure.

Our patient was an elderly male smoker at risk for atherosclerosis and had presented with typical angina and normal blood pressure. ECG also showed features of inferior wall myocardial infarction. The ischemic myocardial dysfunction is seen in 1–2% of patients with AAD and is caused by an extension of the dissection into the coronary artery or an ostial occlusion by the dissection flap, often affecting the right artery. Differentiating Stanford A AAD with coronary malperfusion from true ACS can be challenging and the misdiagnosis occurs to an extent of 40% in the emergency room. It is important to note that AAD is the most catastrophic event associated with a high early mortality rate of 1–2% per hour within the first few hours. Besides, misdiagnosis may lead to inappropriate administration of anticoagulant/thrombolytic therapy or even interventions, as seen in this patient. The only clue of AAD was the excruciating pain in the lower limb that manifested during per-cutaneous coronary intervention suggesting passage of catheter into the false lumen. This case demonstrates that AAD should always be considered as a differential diagnosis in patients presenting with acute chest pain. It is imperative to carefully evaluate clinical history (especially history of hypertension), chest pain characteristics, and risk factors in patients presenting with ST segment elevation. It must be a matter of habit to check the arterial pulses and blood pressure in all four limbs for possible difference. D-dimer level over 1600 ng/mL within the first 6 h of presentation favors aortic dissection and may be a useful tool in differentiating it from ACSs. Rapid identification and categorization of AAD are aided by imaging as the therapy and outcome relies on multi-disciplinary approach for the use of medical therapy and surgical and/or endo-vascular interventions.

Further Reading

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