Acute aortic syndromes in emergency departments

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Abstract

Introduction: Acute aortic syndrome (AAS) is a modern term to describe interrelated emergency aortic conditions with similar clinical characteristics and challenges. AAS includes aortic dissection, aortic intramural hematoma, and penetrating ulcer of the aorta. They are an important differential diagnosis of acute coronary syndrome.

Objective: The purpose of this article is to review the relevant variants of AAS presentations and diagnoses through three case reports.

Conclusion: AAS refers to a heterogeneous group of conditions that cause a common set of signs and symptoms. Prognosis is related to undelayed diagnosis and appropriate surgical repair in the case of proximal involvement of the aorta; affection of distal segments of the aorta may call for individualized therapeutic approaches favoring endovascular in the presence of low-perfusion or imminent rupture, or medical management.

Keywords: Acute aortic syndrome; Intramural hematoma; Penetrating aortic ulcer; Aortic dissection; Emergency.

INTRODUCTION

In 1760, Dr. Nicholls was the first physician to describe in autopsy acute aortic dissections (AD) [1]. Over two centuries, there was increasing interest and awareness of chronic and acute aortic syndromes (AAS). The term AAS comprises heterogeneous and life-threatening aortic diseases with similar symptoms but different demographic, clinical, pathological, and survival characteristics [2,3]. AAS includes: penetrating atherosclerotic aortic ulcer (PAU), intramural aortic hematoma (IMH), and the classic aortic dissection (AD).

In emergency medicine, the importance of early diagnosis and management of AD is well considered. It is well known as a survival predictor. Although the chest pain of AD is widely suspected and recognized, less consideration has been given to the presentation associated with other aortic pathologies.

In this article, we report three observations of AAS showing the similarities and the differences between AD, IMH, and PAU of the ascending aorta.

OBSERVATION 1

A 52-year-old man with a history of diabetes and untreated hypertension presented to the emergency department (ED) with tingling chest pain and paresthesia of the lower right limb. Examination at admission found: tachycardia at 108 beats per minute (bpm), polypnea at 20 cycles per minute (CPM), and symmetrical 170/100mmHg blood pressure (BP) in both

upper limbs. Cardiac and pulmonary auscultation was without abnormality. Peripheral pulses were present and symmetrical to both upper limbs. Examination of the right lower limb showed paleness and coldness of the right foot with an abolition of the right pedal and back-tibial pulses compared to those of the left lower limb. The electrocardiogram (EKG) showed sinus tachycardia with an under-shift of ST-segment of 1 mm below. The diagnosis of acute ischemia of the right lower limb was retained. But given the associated chest pain, the diagnosis of AD was suspected. Antihypertensive treatment was administered with analgesic treatments.

Aortic CT exploration showed a type A dissection beginning upstream of the left subclavian artery (Figure 1) and extending to the abdominal aorta, superior mesenteric artery, and the right common iliac artery (Figure 2).

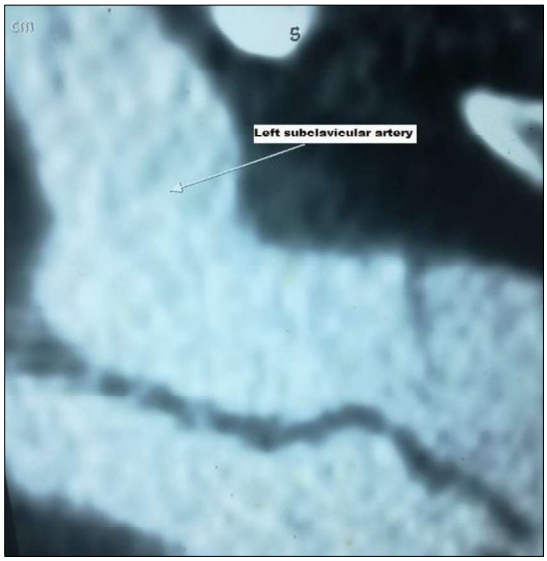

Figure 1: Aortic CT exploration showed a type A dissection beginning upstream of the left subclavian artery

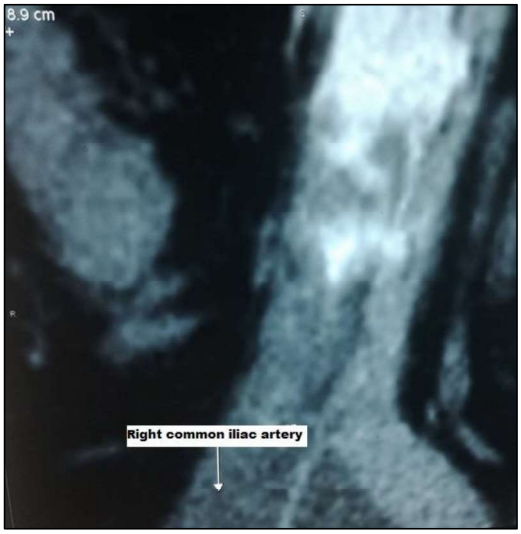

Figure 2: Extending of the dissection to the abdominal aorta, superior mesenteric artery, and the right common iliac artery

The patient was transferred to the cardiothoracic surgery department. Conservative treatment was decided with a good clinical course at three months.

OBSERVATION 2

A 60-year-old man with a history of hypertension and type B aortic dissection for which he underwent surgery (he had a prosthesis in the thoracic descending aorta) presented to the ED for severe thoracic pain for three days followed by paresthesia of the right lower limb since 1 day. Admission examination found: blood pressure of 130/60 mmHg, a pulse at 70 beats/min, polypnea at 25 CPM. The blood pressure was symmetrical in the two upper limbs and the peripheral pulses were present. But, the right lower limb had weaker pulses compared to those of the left lower limb. The EKG was normal with no repolarization or conduction disturbances. Exploration by aortic angiography showed a permeable prosthesis of the descending thoracic aorta and a type B aortic dissection of the

terminal thoracic and high abdominal aorta upstream of the emergence of the celiac artery which is of normal caliber. The patient received intravenous antihypertensive therapy until strict control of the BP. He was referred to his surgeon with a favorable outcome up to three months of follow-up.

OBSERVATION 3

A 70-year-old man with a history of hypertension and type B aortic dissection (AD) presented to the ED with tingling chest pain for 24 hours. Examination at admission found: HR at 70 bpm, polypnea at 20 CPM, and asymmetrical blood pressure (110/60 mmHg) in both upper limbs. Cardiac and pulmonary auscultation was normal. Blood pressure was symmetrical in the two upper limbs and the peripheral pulses were present and symmetrical. The EKG showed tall T waves in anterior leads. Given the normality of cardiac markers, no change in repeated EKGs, and the previous AD, the diagnosis of recurrence of AD was suspected and an analgesic treatment was initiated.

Aortic CT exploration showed an aneurysmal thoracic descending aorta containing an intra-mural hematoma extended to the supra-renal aorta. The patient was referred to a thoracic surgery department.

DISCUSSION

The term AAS was first reported by Vilacosta et al. [1, 3] in 1998. It refers to a heterogeneous group of conditions that cause a common set of signs and symptoms, the foremost of which is aortic pain.

In a review of 464 patients from the International Registry of Acute AD (IRAD), two-thirds were male with a mean age of 63 years. Women were less frequently affected by acute aortic dissection and significantly older than men with a mean age of 67 years [3].

The main risk conditions for AAS are hypertension, atherosclerosis, and a history of cardiac surgery as described in observation 2.

The pathophysiology of AAS is well described: all mechanisms weakening the aortic media layers lead to higher wall stress. This wall stress can induce aortic dilatation and aneurysm formation, eventually resulting in intramural hemorrhage, aortic dissection, or rupture (Figure 3).

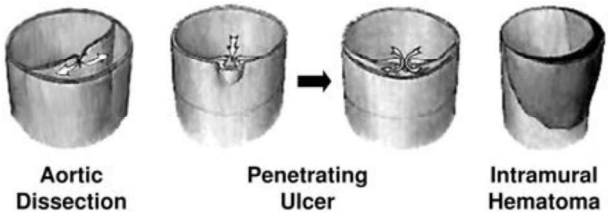

Figure 3: Schematic of aortic dissection (left), penetrating ulcer (middle), and IMH (right).

When classifying the AAS, the most confusing element is the similarity of presentations: acute onset of severe chest or back pain. The pain may be described as tearing, ripping, migrating, or pulsating.

Various diseases may also cause this striking presentation, including trauma, pseudo-aneurysm, and ruptured atherosclerotic aneurysm.

Acute Aortic Dissection (AAD)

AD is the most common aortic emergency (70% of AAS) [3] and occurs when a tear develops in

the aortic intima (inner aortic wall layer) allowing blood to pass between the aortic wall layers creating a false tract between the intima and the media (middle layer) or adventitia (outer layer). The dissection can progress in a retrograde or an antegrade fashion and cause complications including aortic rupture, pericardial tamponade, aortic valve regurgitation, acute coronary syndrome, acute neurological syndrome, renal failure, and bowel or limb ischemia [4-6]. AD is classified according to the location and extent of involvement of the aorta. In the Stanford classification of aortic AD, type A involves the ascending aorta while type B does not involve the ascending aorta. In De Bakey classification, type I involves the ascending aorta, aortic arch, and descending aorta. De Bakey type II AD involves only the ascending aorta while De Bakey type III is confined to the descending aorta.

Intramural Hematoma

IMH originates from a ruptured vasa vasorum in the medial wall layers. The bleeding into the medial layer forms the hematoma. This may lead to infarction of the intima and a tear causing aortic dissection [7]. IMH represents up to 20% of AAS [7, 8]. Miming AD, the IMH can also extend along the length of the aorta either anterogradely or retrogradely. Ascending aortic IMH will present with chest pain while descending IMH may present with back pain or abdominal pain. It is not possible to differentiate aortic IMH from AD clinically as it needs imaging to confirm and exclude dissection.

Penetrating Atherosclerotic Ulcers (PAU)

PAUs are deep ulcerations of atherosclerotic plaques penetrating the intima. These can lead to AD or IMH [9]. These ulcers can deepen and result in aortic perforation with catastrophic results. Penetrating ulcers, like intramural hematoma, need imaging studies for documentation. More and more cases of penetrating ulcers of the aorta are being documented with the widespread use of multi-slice computerized tomographic aortograms. PAUs are often found in the descending aorta.

Diagnostic Strategies

Several imaging techniques have been used in the detection and assessment of patients with AAS. Computed tomography (CT), magnetic resonance (MR), and trans-esophageal echocardiography (TOE) can obtain echotomographic images that help to elucidate the anatomy pathology of these particular syndromes.

Based on the IRAD guidelines [10], contrast-enhanced spiral CT scanning, TEE, and MRI are all extremely accurate in the diagnosis of acute aortic dissection.

Selection of a diagnostic test for suspected aortic dissection requires consideration of both the information required and the access to and experience with the imaging modality at the institution.

In IRAD, CT-scan was used most frequently as the first test in 61% of cases, and TTE or TEE was used as a secondary technique in 56% of the cases. An average of 1.8 methods was used to diagnose acute AD.

CONCLUSION

Much has been learned about the risk factors, clinical characteristics, diagnosis, and management of acute AD over the last decade. Technological advances in imaging techniques and a better understanding of the pathobiology of acute AD have led to the discovery of variants of aortic pathologies now called AAS. Furthermore, several surgical and percutaneous treatment strategies are continuing to improve and evolve. As a result of knowledge and interest in this area, the outcomes of patients treated for AAS have improved.

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