MDCT Evaluation of Acute Aortic Syndrome

Seung Min Yoo, MD, PhD[a], Hwa Yeon Lee, MD, PhD[b], Charles S. White, MD[c,*]

KEYWORDS

- Acute aortic syndrome • Aortic dissection
- Intramural hematoma • Penetrating atherosclerotic ulcer
- Multidetector CT (MDCT)

Acute aortic syndrome (AAS) comprises aortic dissection (AD), intramural hematoma (IMH), penetrating atherosclerotic ulcer (PAU), and unstable aortic aneurysm. Because the highest mortality of AAS, particularly AD, occurs during the first 48 hours after onset of symptoms, prompt diagnosis and immediate initiation of appropriate therapy is essential for a favorable outcome.[1] Unfortunately, several studies have reported that a delay in diagnosis of more than 24 hours after admission occurs in up to 39% of patients with AD.[2,3] This is mainly because of a significant overlap of clinical symptoms between AAS and acute coronary syndrome (ACS) or pulmonary embolism (PE). In addition, the annual incidence of AAS, ACS, and PE has been estimated at 0.5 to 3.0, 440.0, and 69.0 per 100,000 in the United States, respectively.[4,5] This relatively rare occurrence of AAS compared with ACS and PE increases the likelihood of delayed diagnosis or misdiagnosis of AAS as ACS or PE. Although radiologists are not directly involved with history taking or physical examination of patients with suspicious AAS, a precise understanding of both the pretest probability and typical clinical symptoms and signs of AAS is valuable for radiologists to get the broadest perspective of AAS.

Current multidetector CT (MDCT) equipped with state-of-the art tube and detector technology, and optimal temporal and spatial resolution has become widely available globally. With appropriately obtained MDCT data in patients who have findings suspicious for AAS, the diagnostic accuracy of MDCT is nearly 100%.

This article provides a summary of AAS, focusing especially on MDCT technique and findings of AAS, as well as recent concepts regarding the subtypes of AAS, consisting of AD, IMH, PAU, and unstable aortic aneurysm.

AORTIC DISSECTION

Pathogenesis of Aortic Dissection

The exact mechanism of AD still remains unclear.[6] AD is characterized by intimal rupture and subsequent formation of a false lumen parallel to the original aortic lumen. An entry tear is likely to be a primary event for development of most AD. In some cases, intramural hemorrhage in the media followed by intimal rupture may also be an initiating event. Most patients with AD have hypertension. The most common sites of entry tear are the right lateral wall of the ascending aorta and the descending aorta just distal to the left subclavian artery, where the shearing stress against the aortic wall generated by hypertensive blood flow is maximal. Once an entry tear is made, propagation of AD ensues along the aortic lumen, either in an

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[a] Department of Diagnostic Radiology, 351 Yatop-dong Bundang-gu, CHA Medical University Hospital, Bundang 463-712, Korea
[b] Department of Diagnostic Radiology, 65-207 Hangang-ro 3 ga Youngsan-gu, Chung-Ang University College of Medicine, Seoul 140-757, Korea
[c] Department of Diagnostic Radiology, 22 South Greene Street, University of Maryland, Baltimore, MD 21201, USA
* Corresponding author.
E-mail address: cwhite@umm.edu (C.S. White).

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antegrade or retrograde fashion. When an ascending aortic dissection propagates into the aortic arch and descending aorta, the dissection often extends along the greater curvature of the aortic arch, resulting in frequent involvement of aortic arch branches (**Fig. 1**).[7] The term intimal flap is a misnomer. As the dissection flap is composed of intima and the inner two-thirds of media, intimomedial flap is a more appropriate terminology. The thickness of the outer wall of false lumen is only one-third of the intimomedial flap and one-quarter of the original aortic wall. The outer wall of the false lumen is thus vulnerable to aortic rupture.[7,8] Surgeons operating on AD often describe this structure as paper-thin. Most aortic ruptures occur in the vicinity of the entry tear. The presence and location of the high-density hematoma on pre–contrast-enhanced CT suggests the site of aortic rupture. Hemopericardium or right hemothorax indicates rupture of the ascending aorta, whereas hemomediastinum and left hemothorax suggest rupture of the aortic arch and descending aorta, respectively (**Fig. 2**).[7]

Predisposing Factors

Predisposing factors related to AD are hypertension, aortic disease (eg, bicuspid aortic valve, aortic coarctation, and aortic aneurysm),[9] connective tissue diseases of the aorta (eg, Marfan's syndrome and Ehler-Danlos syndrome),[10,11] direct trauma to the aortic wall,[12] cocaine abuse,[13] and pregnancy.[14]

Most AD occurring in young patients is related to Marfan's syndrome.[9] Cocaine use may result in rapid increase of blood pressure, making cocaine users vulnerable to intimal tear. AD occurring in young women is often associated with pregnancy-induced hypertension during the third trimester or labor.[15]

CLINICAL FINDINGS OF AORTIC DISSECTION AND CHOICE OF IMAGING MODALITIES

Typical AD has been associated with severe chest or back pain of sudden onset with a tearing or ripping quality in an older patient (ie, sixth or seventh decade) who has hypertension.[3] However, ripping or tearing chest pain may not be a typical descriptor in patients with AD. According to a study performed by the International Registry of Acute Aortic Dissection (IRAD), the incidence of tearing or ripping pain (51%) was less frequent than that of sharp pain (64%) in patients with AD.[4] The location of pain is related to the site of AD: patients with ascending AD are more likely to have anterior chest pain, whereas those with descending aortic dissection more often complain of posterior chest, back, or abdominal pain. A migratory nature of the pain (16.6%) and radiation of pain (28.3%) to the interscapular region, back, or abdomen are fairly typical of AD.[4]

Focal neurologic signs or symptoms, and a pulse deficit or pressure difference between the two extremities are also characteristic of AD. For example, in a patient with severe chest pain of sudden onset, a nonpalpable unilateral extremity pulse is highly suggestive of AD, regardless of the nature of the pain (**Fig. 3**).

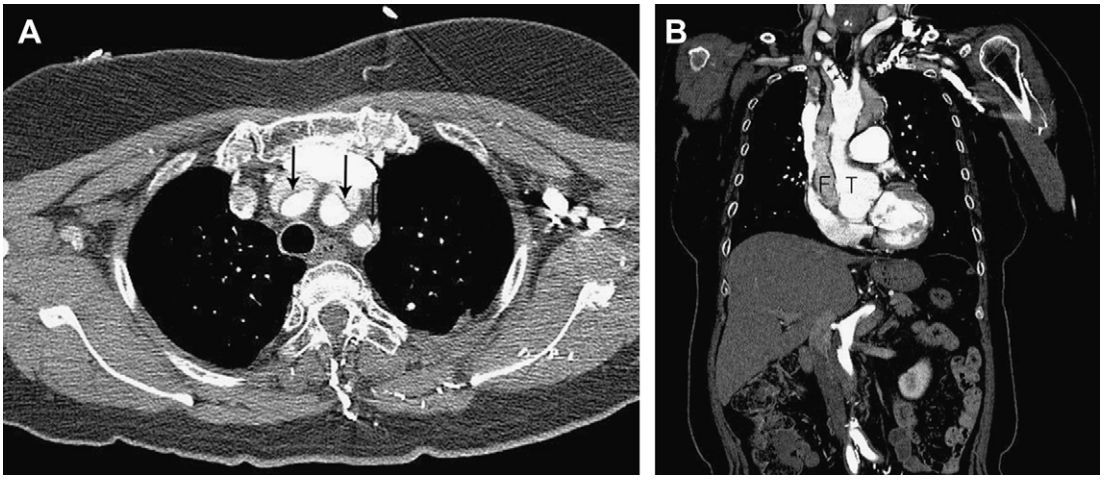

Fig. 1. Extension of intimomedial flap into all of three aortic arch branches in 67-year-old woman with Stanford type A aortic dissection. (*A*) Intimomedial flap (*arrows*) is noted in brachiocephalic, left common carotid, and left subclavian artery on contrast-enhanced axial CT image at the level of left brachiocephalic vein. (*B*) Intimomedial flap (*arrows*) extending into brachiocephalic artery is clearly noted on coronal MPR image. The contrast enhancement of true lumen (T) is higher than that of false lumen (F) owing to slow flow of the false lumen.