Acute Aortic Syndrome: Recent Trends in Imaging Assessment Using Computed Tomography Angiography

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Multidetector computed tomography (CT) has become the best modality for evaluating an aorta in the acute setting, due to its ability to rapidly detect aortic pathology with high spatial resolution. Moreover, using three-dimensional multiplanar reconstruction, CT scans can provide informative images and accurate measurements of aortic size and other anatomical landmarks. This review focuses on the CT imaging findings of aortic normal anatomy, aortic aneurysm, and the imaging spectrum of acute aortic syndrome, which includes penetrating atherosclerotic ulcer, intramural hematoma, and aortic dissection.

Key words Computed Tomography Angiography · Aorta · Aortic disease · Aneurysm · Dissection · Hematoma · Ulcer

INTRODUCTION

Acute aortic syndrome (AAS) denotes a spectrum of life-threatening aortic conditions with characteristic “aortic” pain and similar clinical profiles. The spectrum includes penetrating atherosclerotic ulcer (PAU), intramural hematoma (IMH), dissection, and unstable aneurysm. Rapid diagnosis of and treatment decision regarding these serious disease entities are necessary; however, their clinical presentations are similar, and their differentiation depends on the use of radiologic diagnostic tools. Multidetector computed tomography angiography (CTA) is the commonly used imaging tool for evaluating an aorta in the acute setting. It offers fast acquisition and reliable diagnostic accuracy, thereby enabling accurate treatment planning. This review focuses on the CTA findings of the normal anatomy of the aorta, aortic aneurysm, and differential diagnosis of AAS.

NORMAL AORTA AND AORTIC ANEURYSM

Normal anatomy of the thoracic aorta

The thoracic aorta is grossly divided into four segments: the aortic root, ascending aorta, aortic arch, and descending aorta

(Fig. 1). The aortic root is the first portion of the aorta, beginning just distal of the aortic valve, and consists of the aortic annulus, sinus of Valsalva, and sinotubular junction. The ascending aorta extends from the sinotubular junction to the origin of the right brachiocephalic artery (RBCA). The aortic arch begins at the origin of the RBCA and ends at the origin of the left subclavian artery (LSCA). The aortic arch is subdivided into the proximal (RBCA to left common carotid artery) and distal segments. The distal arch is often narrower than the proximal descending aorta at the attachment site of the ligamentum arteriosum, called the “aortic isthmus.” The descending aorta begins at the ligamentum arteriosum and extends to the diaphragmatic hiatus; its proximal dilated portion has been termed the “aortic spindle.” The aortic wall is histologically composed of three layers: the inner mostly “intima” layer lined with thin endothelium; the “media” composed of smooth muscle cells, which are encased by concentric elastic and collagen fibers on the border zone in both the inner and outer layers; and the “adventitia” layer containing mainly collagen, the vasa vasorum, and lymphatics [1,2].

Measurement of aortic diameter by CTA is generally recommended with three-dimensional (3D) reconstructed images whenever possible; the maximum diameter is measured perpendicular to the centerline of the vessels on the curved multiplanar reconstruction (MPR) images [1,3]. In situations where 3D and MPR imaging are unavailable, the minor axis (smaller diam-

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eter) is generally closer to the true maximum diameter than is the major axis diameter on transaxial images; Use of the minor axis diameter enables more accurate and reproducible measurements, particularly in tortuous aneurysms [4,5] (Fig. 2). No consensus has been reached on whether the aortic wall should be included or excluded in the aortic diameter measurements, even though the difference in diameter can be large, depending on the amount of intraluminal thrombus or atheromas on the arterial wall [1]. Recent prognostic data were derived from measurements that included the wall [6]. Electrocardiography (ECG)-gated acquisition protocols have recently been used to reduce motion artifacts, especially for the aortic root and ascending aorta [7,8]. The dimension of the aortic root varies through the phases of the cardiac cycle. However, whether the measurement of the aortic root should be performed dur-

ing the systole or diastole phase has not yet been accurately assessed; nevertheless, diastolic images have the best reproducibility [1]. However, measurement of the aortic annulus for preprocedural CT evaluation of aortic valve replacement is traditionally performed in the systolic phase, during which it manifests with its intrinsically largest diameter [9]. The normal diameters of the aorta have been defined based on normative measurements performed by several investigators using large subject populations [10-12] and should be considered relative to age, sex, and body surface area [13]. Table 1 provides the generally accepted maximal normal diameters of the aorta and practical size criteria for aortic aneurysms [14].

Aortic aneurysm

An aneurysm is defined as a permanent dilatation of the aorta exceeding the normal measurements by more than 2 standard deviations at a given anatomic level. It is usually caused by atherosclerosis (degeneration of the media) and is associated with inherited conditions including Marfan syndrome, Loeys-Dietz syndrome, Ehlers-Danlos syndrome, and Turner syndrome [14-16]. Aortic aneurysm is divided into two main categories of true aneurysm and pseudoaneurysm, depending on involvement of all three layers of the aortic wall (intima, media, and adventitia). A true aneurysm contains all three layers of the aortic wall, without disruption of any layer, and usually has a fusiform appearance. In a pseudoaneurysm, the intimal-medial layer is disrupted because of trauma (usually seen in the aortic isthmus), penetrating aortic ulcer (usually seen in the descending aorta) in underlying severe atherosclerosis, or inflammation (the so-called "mycotic aneurysm," shows a tendency to involve the ascending aorta in cases of infective endocarditis) [11,17].

In the dilated aorta, an intraluminal thrombus develops along the intimal layer of the aortic wall due to decreased blood flow. The intraluminal thrombus resembles an atherosclerotic plaque; however, it has a smoother margin and a crescentic or concentric shape (Fig. 3). It can be differentiated from IMH according to location (internal to intimal calcifications) and usually shows a low density, whereas the IMH shows a high density in the acute phase with chest pain [7].

The ductus diverticulum and aortic spindle are normal variants that can mimic a thoracic aortic aneurysm [7,18]. The ductus diverticulum is a remnant of closed ductus arteriosus and

Fig. 1. Anatomic segments of the thoracic aorta. The thoracic aorta is grossly divided into four segments: aortic root, ascending aorta, aortic arch, and descending aorta. The aortic root includes the aortic annulus, sinus of Valsalva, and sinotubular junction. The aortic arch begins at the origin of the RBCA and ends at the origin of the LSCA and is subdivided into proximal (RBCA to LCCA) and distal segments. RBCA: right brachiocephalic artery, LSCA: left subclavian artery, LCCA: left common carotid artery.

Table 1. Aortic diameters

Segment	Maximal normal diameter (cm)	Aneurysm size criteria* (cm)	High risk of complication (cm)	Recommended surgical repair (cm)
Ascending	4	5	6	5.0–5.5
Descending thoracic	3	4	7	5.5–6.5
Abdominal	2	3	7	5.5–6.5

* ≥ 1.5 times the expected normal diameter

consists of a convex focal bulge along the anterior undersurface of the isthmic regions of the aortic arch. In contrast with post-traumatic pseudoaneurysm, the ductus diverticulum has smooth margins with symmetric shoulders and forms obtuse angles with the aortic wall. An aortic spindle is a smooth, circumferential bulge below the isthmus in the first portion of the descending aorta [7] (Fig. 4).

When aneurysms reach a diameter of 6 cm in the ascending and 7 cm in the descending aorta, they distinctly increase in risk of rupture. Consequently, treatment (surgical repair or en-

doluminal stent graft) is recommended when the diameters are 5.5 cm and 6.5 cm, respectively [19]. On CTA, acute rupture of an aneurysm appears as an ill-defined, heterogeneous, peri-aortic soft-tissue hematoma, sometimes with a focal contrast blush of active extravasation at the site of rupture. Pleural effusions or hemothorax are also often seen.

ACUTE AORTIC SYNDROME

AAS includes a spectrum of life-threatening aortic conditions

Fig. 2. Measurements of aortic size on transaxial images acquired using CTA. (A) A three-dimensional volume-rendered image (left) and transaxial images (right) acquired using CTA in a 62-year-old woman show diffuse tortuous aneurysm through the aortic arch. The minor axis (arrow) is closer to the true maximum diameter of the aorta because the longitudinal axis of the aortic arch is not perpendicular to that in the transaxial image. (B) Chest radiograph (left) and transaxial CT image (right) of a 63-year-old man show severe chest wall deformity with kyphoscoliosis. Consequently, the descending thoracic aorta lies transversely in the transaxial image. However, the diameter of the aorta is within the normal range with minor axis measurement (arrow). CTA: computed tomography angiography.

Fig. 3. Representative images of an intraluminal thrombus (A) and atherosclerotic plaque (B). (A) The intraluminal thrombus (black arrows) has a circumferential border with a crescentic shape, low density, and is located internal to the intimal calcifications (white arrows). (B) The atherosclerotic plaque (black arrows) is similarly located inside the intimal layer (white arrow) as an intraluminal thrombus; however, it has a more irregular border and tends to develop ulceration.

with characteristic “aortic” pain and similar clinical profiles and includes aortic dissection, IMH, and PAU [20,21]. Sometimes, ruptured aortic aneurysms are included under AAS. The common presentation of AAS is sudden severe thoracic or abdominal pain that can radiate to the back, neck, throat, or jaw. In cases of aortic arch vessel involvement, syncope can be an initial presentation because of hypoperfusion of the brain. The initial presenting signs or symptoms of AAS are clinically indistinguishable in some cases, and they can develop together or be directly related; therefore, imaging surveillance is important in the diagnosis and identification of the culprit lesion. The classification of AAS is based on the location and extension. The Stanford classification system grades AAS based on whether it involves the ascending aorta, and the DeBakey classification system grades AAS based on both extension and site of origin [1]. Depending on the involvement of the ascending aorta, the Stanford classification system categorizes dissections into types

A and B, with type A dissection involving the ascending aorta, and the type B dissection lacking involvement of the ascending aorta. The DeBakey classification system divides dissections into types I, II, and III, with type I originating in the ascending aorta and extending to the aortic arch and descending aorta, type II originating in the ascending aorta and limited to the ascending aorta, and type III originating in the descending aorta (usually just distal to the origin of the LSCA) and mostly extending to the descending aorta. Occasionally, however, type III dissections can extend proximally into the aortic arch and ascending aorta.

Aortic dissection

Aortic dissection is defined as an intimal tear (intimal-medial flap) and medial dissection with longitudinal spread of blood. Aortic dissection is the most common cause of AAS, with an incidence of 0.2–0.8% [14]. According to a recent meta-analy-

Fig. 4. Representative images of the ductus diverticulum (A) and aortic spindle (B), which mimic an aortic aneurysm. (A) Incidentally detected ductus diverticulum in a 42-year-old man who underwent chest computed tomography during a health screening. The ductus diverticulum (arrows) has a convex focal bulge along the anterior undersurface of the isthmus regions of the aortic arch, smooth margins, and forms obtuse angles with the aortic wall. (B) Aortic spindle in a 55-year-old man with chest discomfort shows a smooth, circumferential bulge (arrows) below the isthmus in the first portion of the descending aorta.

sis by Takagi et al. [22], the incidence of aortic dissection was significantly more frequent in the winter (28.2%) than in other seasons. The major risk factors for aortic dissection are hypertension, male sex, advanced age, and underlying aortic aneurysm or IMH. Other predisposing factors are cystic medial necrosis, Marfan syndrome (younger patients), Ehlers-Danlos syndrome, bicuspid aortic valve, coarctation of the aorta, pregnancy, trauma, and arteritis [23]. The common sites of intimal tear are within several centimeters of the sinus of Valsalva (75%) in the ascending aorta and just distal to the LSCA origin in the descending aorta. The complications of aortic dissection are aortic rupture, hemothorax, mediastinal hematoma, hemo-pericardium, acute aortic regurgitation, branch vessel occlusion, and end-organ ischemia. The treatment strategy for aortic dissection generally depends on the presence of ascending aortic involvement and complications; therefore, most guidelines

have been applying the Stanford classification system. Acute type A aortic dissection has a mortality of 50% within the first 48 hours if not surgically repaired. Therefore, immediate surgical management is the treatment of choice [1,24,25]. The treatment of type B aortic dissection depends on the presence or absence of complications. In patients with uncomplicated type B dissection (absence of malperfusion or end-organ ischemia), medical therapy (to control pain and blood pressure) with serial imaging follow-up using CT or magnetic resonance imaging is recommended. Patients with acute complicated type B aortic dissection are treated using thoracic endovascular aortic repair (TEVAR) [26]. However, if patients have contraindications to TEVAR, such as lower extremity arterial disease, tortuosity of the iliac arteries, or/and an inadequate landing zone for the stent graft, open surgical repair should be considered [27].

On non-enhanced CT images, medially displaced intimal

calcification is a suggestive finding of aortic dissection. Non-enhanced CT images are also important for identifying coexisting lesions, such as IMH, hemomediastinum, hemopericardium, and hemothorax [28,29]. On contrast-enhanced images, the entry site of the intimal tear is directly visualized using an intimal flap separating the true lumen from the false lumen. The true lumen is usually smaller than the false lumen, with earlier opacification in the arterial phase. The false lumen has a slower flow and a larger diameter and can contain thrombi [30]. Differentiating between the false and true lumens is sometimes challenging, especially when they show similar opacification and size (Fig. 5). Several specific findings aid in the identification of a false lumen, such as the cobweb sign (residua of incompletely sheared media) or beak sign (acute angle between the dissecting flap and outer wall) [14,21,30,31].

The pulsation artifact is the most common cause of a pseudodissection in the thoracic aorta [32]. It is caused by pulsatile movement of the ascending aorta during the cardiac cycle, and the right posterior and left anterior aspects of the ascending aorta are the most commonly affected sites of the pulsation artifact (Fig. 6). However, in type A aortic dissection, the false lu-

men is usually located along the right anterolateral wall of the ascending aorta and extends distally in a spiral fashion along the left posterolateral wall of the descending aorta [32]. To avoid this

Fig. 6. Stanford type B aortic dissection involving the descending thoracic aorta in a 66-year-old man. The pulsation artifact typically affects the right posterior and left anterior aspects of the ascending aorta (arrows). T: true lumen, F: false lumen.

Fig. 5. Differentiation of the false and true lumens of aortic dissection on CTA. (A) Arterial-phase CTA in a 45-year-old man with type B aortic dissection. The true lumen “T” is easily detected, is smaller than the false lumen “F,” and has higher opacification. (B) Arterial-phase CTA in a 65-year-old woman with type A aortic dissection. The true and false lumens show similar opacification; however, the false lumen “F” is larger than the true lumen “T” with residual sheared media (arrows) and forms an acute angle (arrowheads) between the dissecting flap and the outer adventitial layer. CTA: computed tomography angiography.

artifact, retrospective ECG-gating should be used during CT, especially if a lesion on the aortic root or ascending aorta is suspected [8]. Since the retrospective ECG-gated CT scan technique significantly increases the radiation dose, various techniques for reducing the radiation dose should be considered, such as using a low tube voltage, ECG-based tube current modulation, automatic exposure control, and iterative reconstruction [31].

Intramural hematoma

IMH is defined as an intramural thrombus in the medial layer without a false lumen or a visible defect on the intimal layer. IMH accounts for 10–25% of AAS [1,33,34]. Hypertension is the most common predisposing factor for IMH. In the traditional understanding of the development of IMH, hypertension can provoke aortic medial layer degeneration, resulting in spontaneous rupture of the vasa vasorum and leading to hema-

Fig. 7. A 46-year-old man presenting with acute chest pain. (A) Non-enhanced CT revealed a circumferential, crescentic, high-density thickening along the aortic wall in the ascending aorta and aortic arch, suggesting an acute aortic IMH. (B) On contrast-enhanced CT images, which were acquired based on the findings presented in (A), the IMH presented as a well-circumscribed, crescentic thickening with mild enhancement. Neither pleural effusion nor pericardial effusion was noted. (C) During the follow-up period, small extraluminal contrast outpouchings were found in the aortic arch (arrowhead), suggesting an intimal tear. (D) After 2 weeks, the aortic IMH progressed to type B aortic dissection. CT: computed tomography, IMH: intramural hematoma.

toma formation with medial layer splitting [35,36]. On the other hand, in patients with severe atherosclerotic disease, rupture of a PAU or atherosclerotic plaque can result in intimal-medial injury and bleeding into the media [37]. Thus, the current opinion regarding IMH is that it can be a variant of or a precursor to aortic dissection with a small intimal defect and thrombosed false lumen without a re-entry tear [31,37].

The clinical presentation of IMH is similar to that of aortic dissection; thus, image-guided evaluation is essential for accurate diagnosis. For the diagnosis of IMH, non-enhanced CT is very helpful and enables the evaluation of circumferential, crescentic, and high-density thickening (>7 mm in diameter; 60–70 Hounsfield unit in attenuation) along the aortic wall that extends in a longitudinal (non-spiral) pattern [35]. In the subacute or resolving phase, the density of the IMH decreases to isodense with blood on non-enhanced CT. Similar to aortic dissection, aortic IMH can present with inwardly displaced intimal calcifications. On contrast-enhanced CT images, the IMH presents as an intramural fluid collection resembling a non-enhancing, well-circumscribed, crescentic region in the aortic wall. During the follow-up period, a small extraluminal contrast outpouching is often found and indicates an intimal tear or PAU (Fig. 7).

Indications for endovascular treatment of IMH are basically the same as those for dissection: medical treatment with serial imaging follow-up for type B aortic IMH and immediate surgical treatment for type A aortic IMH [21,38]. During the follow-up period of type B aortic IMH, if a gradual increase in IMH thickness, luminal dilatation, pseudoaneurysm formation (or ulcer-like projection), or progression to aortic dissection occurs, surgical or endovascular treatment is required [38]. An “ulcer-like projection” refers to localized contrast filling with obvious communication with the true lumen, which represents a site of new intimal disruption in IMH. In contrast, there is another type of localized blood pool in IMH, which shows no obvious communication with the true lumen and has a connection with

the branch arteries; this is the so-called “intramural blood pool” (Fig. 8). This blood pool is not associated with a poor prognosis, and most resolve over time or become stable during follow-up (86%) [39].

Penetrating atherosclerotic ulcer

PAU is an ulceration of atherosclerotic lesions that penetrates the intima and extends into the medial layer. The estimated incidence of PAU is 2.3–7.6% in AAS [29]. PAU is usually accompanied by IHM and can lead to aortic dissection, aortic aneurysm, or rupture [40] (Fig. 9). PAU develops in underlying advanced atherosclerosis; therefore, it commonly occurs in the mid-descending aorta or aortic arch and is very rare in the ascending aorta (which is protected from atherosclerosis by rapid blood flow). On non-enhanced CT, PAU is difficult to delineate and can occasionally be found with concomitant IMH or atherosclerosis. Contrast-enhanced CT shows a protrusion of contrast filling through the atheroma outside the normal aortic lumen. The aortic wall can be thickened with IMH or contrast enhancement (Fig. 10). PAU can be differentiated from an atherosclerotic ulcer, which is confined to the intima, by a lack of

Fig. 9. Natural course of an atheromatous ulcer (modified from Hayashi et al. Radiographics 2000;20:995-1005 [40]). PAU: penetrating atherosclerotic ulcer, IMH: intramural hematoma.

Fig. 8. A 75-year-old man with Stanford type B aortic IMH. The intramural blood pool (arrows) appears as a focal contrast medial filling in the IMH without communication with the true lumen and has a connection with the intercostal artery (arrowheads). IMH: intramural hematoma.

Fig. 10. A 75-year-old man with acute chest pain underwent chest CT for evaluation. (A) Contrast-enhanced CT shows irregular atheromas in the aortic arch, with protrusion of contrast filling (white arrows) outside the intimal calcifications (black arrows), suggestive of a PAU. A concomitant aortic IMH is seen in the distal aortic arch and descending aorta. (B) Follow-up chest CT performed after 1 week revealed the development of aortic dissection in the distal aortic arch (previous IMH), and the entry site is the PAU. CT: computed tomography, PAU: penetrating atherosclerotic ulcer, IMH: intramural hematoma.

Table 2. Potential computed tomography protocols in acute aortic syndrome

Suspicious disease	Non-enhanced scan	Arterial phase	Delayed phase
Aneurysm rupture	Optional	Standard	Optional
Aortic dissection	Recommended	Standard	Standard
Intramural hematoma	Recommended	Standard	Optional
Penetrating aortic ulcer	Recommended	Standard	Optional

IMH and no symptoms [41]. Uncomplicated PAU can usually be managed conservatively with close imaging follow-up, similar to type B aortic dissection. Patients with PAU experience persistent or recurrent pain, hemodynamic instability, rapidly expanding aortic diameter, aortic rupture, or distal embolization of the thrombus. The presence of these symptoms is considered an indication for surgical or endovascular repair treatment [40,42]. There is no clear cutoff size for PAU that warrants treatment. In the study by Ganaha et al. [38] a depth >20 mm or a neck >10 mm was associated with higher complication rates.

COMPUTED TOMOGRAPHY PROTOCOLS IN ACUTE AORTIC SYNDROME

CTA has become the best modality for diagnosing aortic disease, especially acute aortic disease, due to its ability to rapidly detect aortic pathology [1,21,43,44]. Most aortic diseases

are easily detected in the arterial phase, and delayed (venous) phases are often included in protocols to evaluate the late-enhancing false lumen and perfusion state of the impacted solid organs, especially in aortic dissection (Table 2) [21]. In addition, the delayed phase is important in postoperative CT imaging followed by stent-graft insertion in order to evaluate for endoleaks [7]. A recent study by Sueyoshi et al. [45] investigated the instability and natural course of type B aortic IMH and found that enhancement of the false lumen was the only independent predictor of IMH-related events. According to their report, delayed-phase CT can be useful for predicting the prognosis of IMH. While non-enhanced CT is not always necessary for diagnosis, it can sometimes be helpful in characterizing the stage (acute, subacute, or chronic) of hematoma, accompanying IMH, or other underlying disease entities (aortic dissection or PAU).

CONCLUSION

We have described the normal anatomy of the aorta, aortic aneurysm, and AAS with respect to CTA findings. CTA provides both schematic and detailed morphologic information on the aorta, thereby enabling exact diagnosis and guiding the management of aortic disease.

Conflicts of Interest

The authors declare that they have no conflict of interest.

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