Acute aortic syndrome: Comprehensive MDCT evaluation

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Learning objectives

1. To describe and illustrate the spectrum of MDCT findings in AAS.

2. To highlight the significance of imaging findings in determining prognosis and management of patients with AAS.

3. To present a specific search pattern for findings in patients with AAS.
Background

The term Acute Aortic Syndrome (AAS) is used to describe three closely related emergency entities of the thoracic aorta which are clinically indistinguishable: classic aortic dissection, intramural hematoma and symptomatic penetrating atherosclerotic ulcer. MDCT is the most accurate imaging modality for the initial diagnosis, differentiation and staging.  

**Aortic dissection** is defined as tear in the aortic intima, disruption of the medial layer of the aortic wall, resulting in separation of the aortic wall layers and subsequent formation of a true lumen and a false lumen, separated by a layer of intimo-medial tissue known as the intimal flap (Fig.1).

**Aortic intramural hematoma (IMH)** is considered a precursor of dissection, and it represents spontaneous bleeding caused by rupture of vasa vasorum in media. In contrast to classic dissection, IMH has no intimal tear, no entry or false lumen flow (Fig.2).

**Penetrating atherosclerotic ulcer (PAU)** is defined as an ulceration of atheromatous plaque that has eroded the inner, elastic layer of the aortic wall, and penetrated into the medial layer, and has produced a variable amount of hematoma within the media (Fig.3).

**Epidemiology of Aortic Dissection**

Studies suggest an incidence of 2.6 to 3.5 cases per 100 000 person-years.

There are many risk factors for aortic dissection. The most common predisposing factor is hypertension.

Patients with aortic dissection at a young age (<40 years old) are less likely to have hypertension and more likely to have a genetic connective tissue disorder like Marfan’s syndrome and Ehlers-Danlos syndrome, a bicuspid aortic valve, or have had prior aortic surgery.

**Clinical presentation**
Patients with acute aortic dissection typically present with the sudden onset of severe chest pain, although this description is not universal.

Neurologic deficits such as syncope and cerebrovascular accident can be a presenting sign in as many as 20% of patients.\(^9\)

Cardiovascular manifestations involve symptoms suggestive of congestive heart failure due to severe acute aortic regurgitation. These include dyspnea and orthopnea.

Other manifestations include the following:

- Severe hypotension as is a poor prognostic indicator. It is usually associated with pericardial tamponade, severe aortic insufficiency, or rupture of the aorta.
- Dysphagia from compression of the esophagus,
- Flank pain if the renal artery is involved,
- Abdominal pain, if the dissection involves the abdominal aorta,
- Paraplegia,
- Lower extremity ischemia,
- Anxiety and premonitions of death\(^{1,9,11,12}\)

**Natural course and prognosis**

An acute Stanford type A aortic dissection is highly lethal with a mortality of 1% to 2% per hour after symptom onset. If left untreated, 24 hour mortality is 25%, and 50% during first 72 hours and rises to 90% in first month.\(^4\)

When treated, freedom from dissection-related events and survival improves. After repair of a type A aortic dissection, survival is in the range of 50% after 10 years. There is, however, a 17% operative mortality.\(^{13}\)

Stanford type B acute aortic dissection affects the descending aorta and is less lethal than a type A dissection. Patients with an uncomplicated type B dissection have a 30-day mortality of 10%. However, in patients who develop ischemic complications such as renal failure, visceral ischemia, or a contained rupture, they often require urgent aortic repair. Repair carries a mortality of 20% by day 2 that increases to 25% by day 30.\(^{11}\)

Complications of the aortic dissection are diverse and numerous:

1. Aortic rupture with eventual hypotension, shock and death from exsanguination;
2. Pericardial tamponade secondary to hemopericardium;
3. Acute aortic regurgitation and pulmonary edema - complication of proximal aortic dissection propagating into a sinus of Valsalva with resultant aortic valve insufficiency;
4. Myocardial ischemia due to left or right coronary ostium involvement;
5. Neurological complications due to carotid artery obstruction -Ischemic CVA, spinal cord ischemia due to aortic branch involvement;
6. Mesenteric and renal ischemia;
7. Claudication from extension of the dissection into the iliac arteries.\textsuperscript{1,9,11,12}

**Aortic IMH** may extend, progress, and proceed to a classic dissection, rupture or aneurysm; it can also regress, or less frequently reabsorb, reportedly in 10\% cases\textsuperscript{1,5}.

IMH of the ascending aorta has a prognosis similar to a type A dissection. Likewise, IMH of the descending aorta has a prognosis similar to type B dissections.\textsuperscript{1}

IMH of the descending aorta, especially when confined to a short segment or with aortic diameters < 50 mm has a better outcome, but does not preclude one from early progression.\textsuperscript{5}

**The penetrating atherosclerotic ulcer (PAU)** can resolve completely or stay stable, but they can also lead to aortic dissection, aortic saccular aneurysms and even spontaneous aortic rupture. There are conflicting reports about the most common course of the penetrating atherosclerotic ulcer\textsuperscript{14}.

As for the implications of initial PAU size, studies shows there is significant correlation between maximum diameter and depth of PAU and with disease progression\textsuperscript{15}.

**Management of AAS**

Management of acute aortic syndrome relies heavily on imaging findings, therefore comprehensive evaluation of AAS is essential for the right treatment. Treatment modalities include medical management, endovascular stents and surgical repair including a combination of a segmental surgical resection of the dissection followed by insertion of a synthetic endovascular stent graft.

Surgery provides definitive treatment for patients with Stanford type A acute aortic syndrome. In general, implantation of a composite graft in the ascending aorta with or without re-implantation of coronary arteries is performed. In addition, restoration of aortic valve competence is paramount in patients who develop aortic insufficiency. This can be achieved by re-suspension of the native aortic valve or by aortic valve replacement and is dependent on the size of the aortic root and the condition of the aortic valve.\textsuperscript{1}

Patients with uncomplicated aortic dissections confined to the descending aorta are at present best treated with medical therapy. The indications for endovascular interventions in type B dissection are accepted for persistent or recurrent pain, aortic expansion,
dissection progression, and end-organ hypoperfusion syndromes. Endovascular stent grafts have been used successfully as a less invasive procedure for patients with surgical indications for chronic type B aortic dissections. ¹

Similar to type A and B aortic dissections, surgery is advocated in patients with type A IMH and an initial trial of medical therapy in patients with type B IMH. ¹

When the IMH is associated with a PAU, urgent surgical aortic graft replacement should be considered not only for the patient with a type A IMH, but also for those with type B IMH, particularly if the patient has persistent pain and/or if there is an interval increase of a pleural effusion.¹⁵

Symptomatic ulcers with signs of deep erosion are more prone to rupture than others. It is reported that the initial PAU size, maximum diameter and depth all correlate significantly with disease progression. Patients with a PAU that initially measured 20 mm or more in maximum diameter or 10 mm or greater in maximum depth have a high risk of disease progression and thus should be considered candidates for early surgical or endovascular repair.¹⁵
Fig. 1: Aortic dissection starts from an intimal tear within the aorta which allows blood to enter and infiltrate into the media. A false channel or lumen is created separating the intima from the rest of the aortic wall.

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Fig. 2: Aortic intramural haematoma (IMH). Intramural haematoma begins with rupture of the vasa vasorum, which results in bleeding and haematoma formation within the medial layer of the aortic wall.

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Fig. 3: Penetrating atherosclerotic ulcer (PAU). A penetrating atherosclerotic ulcer forms when atheromatous plaques erode. The ulcerating atherosclerotic lesion penetrates the intima and progresses into the media.

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Findings and procedure details

Comprehensive evaluation of acute aortic syndrome should include:

- Recognizing and differentiating aortic dissections, intramural hematomas and penetrating atherosclerotic ulcers.
- Applying Stanford type A or Stanford type B classifications, accordingly.
- Documenting size of PAU and IMH (maximum diameter and maximum depth of PAU; maximum thickness and extension of IMH);
- Noting the maximum diameter of any aortic aneurysm, as measured perpendicular to the vessel wall, also its length, luminal diameter and involvement of all branch arteries, such as the renal arteries.
- Extension of dissection, localization of intimal tears - place of entry and reentry.
- Differentiation of the true lumen from the false lumen
- Diameters of true and false lumen, comments on signs of significant compression of a true lumen by the false lumen.
- Diameter of dissected aorta
- Any side branch and iliac artery involvement.
- Organ hypoperfusion.
- Presence of fluid in the pericardium, mediastinum or pleural cavity, suggestive of rupture of the dissection.
- Iliac vessel tortuosity, calcifications, stenoses and minimum diameters to evaluate their suitability for passage of a catheter if endovascular stent grafting of the aorta is contemplated.

**Recognizing and differentiating aortic dissection, intramural hematoma and penetrating atherosclerotic ulcer.**

**Aortic dissection**

Non-contrast CT may demonstrate only subtle findings such as displacement of atherosclerotic calcification into the lumen that is a commonly identified finding. (Fig 4a)

MDCT angiography gives excellent detail of specific findings for aortic dissection: 1. Intimal flap; 2. double lumen (Fig 4b).

**Intramural haematoma**

In acute IMH, an unenhanced CT provides invaluable clues for diagnosis. Acute intramural haematomas (IMH) appear as a focal eccentric or circular high-attenuating (60-70 HU) thickening of the aortic wall (hyperdense crescent sign); in addition, inward
displacement of intimal calcifications may be seen. These two key findings are best seen in the non-contrasted CT. Examples of the hyperdense crescent sign (Fig. 5a, Fig. 5b, Fig. 6, Fig. 8) and the displacement of intimal calcifications (Fig. 5a, Fig. 5b, Fig. 8) associated with IMH's are clearly seen with this technique.

IMH exhibit low attenuation in relation to the aortic lumen on CT angiography (Fig. 5c, Fig. 5d, Fig. 9), and the finding can be quite subtle. Unlike aortic dissection, no intimal flap is present on the CTA.

Without a non-contrast scan, IMH can be easily overlooked and sometimes misdiagnosed as parietal thrombus, hence a non-contrast phase before CTA is strongly recommended in the technique protocol for acute aortic syndrome.

Unfortunately, IMH can be difficult to distinguish from a thrombosed aortic dissection (Fig. 7). This latter entity is of little clinical significance since it does not affect therapeutic decision-making.

**Penetrating atherosclerotic ulcer**

Typical finding of PAU is a focal contrast-filled, pouch-like protrusion of the aorta, extending beyond the expected aortic wall boundaries (so called mushroom-like appearance). (Fig. 9) As in aortic dissection and IMH, focally displaced and separated intimal calcifications may occasionally be seen.

PAU are usually located in arch and descending aorta, and, rarely, in the ascending aorta.

PAU are often associated with IMH (Fig. 9).

Penetrating atherosclerotic ulcer should not be confused with aortic pseudoaneurysm and aortic ductus diverticulum (Fig. 28, Fig. 29).

**Stanford classification of acute aortic syndrome**

- **Stanford type A** - involves the ascending aorta and/or aortic arch, and may or may not extend to the descending aorta (Fig. 11a, Fig. 11b, Fig. 12). The tear can originate in the ascending aorta, the aortic arch, or more rarely, in the descending aorta (Fig. 14).

- **Stanford type B** - involves the descending aorta or the arch (distal to the left subclavian artery), without the involvement of the ascending aorta. (Fig. 11c, Fig. 13).

**Differentiation of the true lumen from the false lumen, signs of significant compression of a true lumen by the false lumen**
False lumen:

- · often larger lumen size due to higher false luminal pressures
- · often of lower contrast density due to slower flow and delayed opacification (Fig.20)
- · Wedges around true lumen (beak-sign)

Beak sign represents acute angle between the dissection flap and the outer wall at the distal end of the false lumen. (Fig.15a, Fig.18b, Fig.26b)

- · Collagenous media-remnants (cobwebs)

Cobwebs-sign is seen as slender linear areas of low attenuation specific to the false lumen due to residual ribbons of media that have incompletely sheared away during the dissection process (Fig.16a).

- · In circumferential dissection when one of the lumina is surrounded by the other, inner lumen is always true lumen (Fig.15b and Fig.18c).
- · may be thrombosed and seen as mural low density (17d).

True lumen:

- · True lumen is the dissected portion of aorta that is continuous with the lumen of the undissected portion of aorta. (Fig.17a). Often, particularly in a type A dissection, continuity is indeterminable with CT.
- · Eccentric flap calcification, if present - is the sign when the side of the dissection flap facing true lumen contains calcifications, whereas the side of the flap facing the false lumen was of soft-tissue attenuation without apparent calcification. (Fig. 17b and Fig. 17c) Explanation: The term intimal flap is actually a misnomer. Because the rupture of an aortic dissection extends along the aortic media and the detached flap is thicker than that of just the intima as it includes a part of the media also. Thus, the side of a flap facing the false lumen is only composed of soft tissue from the media, but the side of flap facing the true lumen shows calcifications belonging to the intima.
- · The true lumen is usually smaller than false lumen.

Differentiation of true lumen from false lumen is crucial in assessing luminal origin of branch vessels, and it is of a great significance in planning endovascular intervention.

Filiform (extremely narrow) true lumen is a sign of significant compression of the true lumen due to high pressure in false lumen, which can have ischemic complications (Fig.15c, Fig.15d, Fig.19b and Fig.19c).

Complications of dissection
Complications of aortic dissections include aortic rupture, pericardial tamponade, major branch vessel occlusion (static, dynamic occlusion or thrombosis) and organ ischemia.

Even the small amount of fluid in pericardium, mediastinum or pleural cavity must be noted since it suggests of rupture of the dissection (Fig. 22, Fig. 23).

Finally, Beware of the Pitfalls! (Fig. 30)
Fig. 4: 60 year old female with an aortic dissection. a) Displacement of intimal calcifications shown on unenhanced CT (arrows) ; b) Intimal flap (arrow) and two lumens (stars) - findings specific for aortic dissection on CT angiography.

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Fig. 5: 50 y.o. female with an acute IMH of the ascending aorta in transaxial and coronal reconstructions ; 1. a and b : Unenhanced CT ; the hyperdense crescent sign representing an intramural haematoma ( thick arrow ) and intimal calcifications displacement ( thin arrow ) 2. c and d : A CTA depicting an IMH ( star ).

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Fig. 6: 71 y. old female was admitted to hospital due to a sudden onset of chest pain that improved. She was referred by a cardiologist for a MDCT coronary angiography. Unenhanced CT exam obtained as a part of calcium scoring revealed a dilation of the ascending aorta with a focal hyperdensity (star), a finding compatible with an intramural haematoma .

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Fig. 7: MDCT coronary angiography of a same patient as Fig.6 In addition to a large IMH (star), MDCT coronary angiography revealed small amount of the contrast material in aortic media (thick arrow in b) and c) with visible intimal flap (curved arrow in b) and c). The finding is consistent with a thrombosed aortic dissection that was confirmed at surgery.

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**Fig. 8:** 61 y. old female with atypical chest and back pain who was examined in the ER and scheduled for a CT of the thoracic spine as an outpatient. Thoracic spine CT was unremarkable, but it revealed a descending aorta intimal calcification displacement (thin arrow) and hyperdense crescent sign (thick arrow) suggestive of an intramural haematoma.

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Fig. 9: Same patient as in Fig. 8; CTA of the thoracic aorta was performed the following day, and showed a coexisting PAU (arrow) and an IMH (star) of the descending aorta (Stanford type B).

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**Fig. 10:** Follow-up CTA of the same patient as on Fig.8 and Fig.9, obtained 4 months later; CTA shows regression of both the PAU and the IMH.

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**Fig. 11:** Stanford classification: Both a) and b): Stanford type A (affects ascending aorta and arch); c) Stanford type B (begins beyond left subclavian artery)

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**Fig. 12:** 52 years old male with a Stanford type A aortic dissection CT exam shows aortic dissection that involves ascending aorta, aortic arch and descending aorta.

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**Fig. 13:** 57 y old male with a Stanford type B dissection. CTA shows the entry point distal to left subclavian artery with the dissection involving only the descending aorta.

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Fig. 14: Same patient as Fig.13 An interval CTA obtained 3 days after the initial scan because the patient’s clinical condition worsened. These images all show propagation of the dissection in a retrograde fashion that has evolved into a Stanford type A. Additionally, pericardial fluid is now present - an unfavorable prognostic sign.

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Fig. 15: 50 year old male with a Stanford type A dissection: a) The dissection involves the ascending and descending aorta; a visible beak-sign (arrows) marks the false lumen which is larger than the true lumen. b) Circumferential intimal lap separating inner true lumen (t) from outer false lumen (f). c) Narrow-filiform true lumen as a sign of a compression by the false lumen. d) Consequently, there is a lack of contrast opacification in the left kidney consistent with hypoperfusion and ischemia.

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Fig. 16: 42 y.o. female with a Stanford type A dissection - Cobweb-sign (arrow in a) a specific finding for a false lumen, representing the residual ribbons of media that have incompletely sheared. - Note the dissection extending into the brachiocephalic, left common carotid and left subclavian arteries see b) and c); - spiral configuration of dissection d) and e); - lower contrast density of a false lumen due to slower flow and delayed opacification (a, b, c, d, e)

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**Fig. 17:** 59 years old male with a Stanford type B dissection; a) entry point of dissection is beyond the left subclavian artery. True lumen (t) is continuous with a normal section of the aorta. b and c) Eccentric flap calcification (arrows) is present along the true lumen side of flap. d) False lumen (f) contains thrombus (star) and is larger than the true lumen.

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**Fig. 18:** 40 year old male with Stanford type A dissection involving the aortic root (a) and extending into brachiocephalic trunk (d). Note the circumferential intimal flap with inner true lumen (c) and beak sign (b).

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Fig. 19: Same patient as Fig. 18, Dissection extending to celiac trunk (a) and right iliac artery (d). Note the ischemic configuration of true lumen which is extremely narrowed (b and c), Right kidney demonstrates a lack of perfusion due to right renal artery occlusion (c); Left kidney enhances with left renal artery originates from the false lumen (c).

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**Fig. 20:** 60 years old male with type A dissection: Contrast differences between arterial and venous phase can be helpful in differentiating true (t) and false lumen (f). False lumen is of lower contrast density in arterial phase (a and c) due to slower flow and demonstrates delayed opacification in venous contrast phase (b and d). Note: dissection extends into brachiocephalic trunk and left common carotid artery (c).

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**Fig. 21:** Same patient as fig. 20: a) dissection involves aortic root; b) dissection extends into right common carotid artery and right subclavian artery; c) dissection extends into left common iliac artery; d) dissection extends into left renal artery without signs of left kidney hipoperfusion; e) right renal artery and superior mesenteric artery originate from the true lumen.

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**Fig. 22:** 54 year old man with Stanford type A dissection after aortic valve replacement: • Dissection involves the aortic root (a); • There is a large periaortal haematoma (b and e) and pericardial and pleural fluid (a) consistent with the rupture of dissection. • Dissection involves both iliac arteries, which are aneurysmal (d and e). • Note: The left renal artery originates from the true lumen and right from false lumen (c and e).

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**Fig. 23:** 59 year old female with a ruptured Stanford type A dissection.

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**Fig. 24:** Male, 48 y. o. with Stanford B aortic dissection. a and b) entry point of dissection is beyond the left subclavian artery; c) false lumen is larger, but there is no ischemic configuration of the true lumen; d) both kidneys enhance.

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Fig. 25: Same patient as on fig.24; An interval CTA obtained three days after the initial scan shows narrow true lumen as a sign of a compression by the false lumen (a,b,c,d). Also visible signs of hypoperfusion of the lower pole of the left kidney (d).

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**Fig. 26:** 52 y.o. male with Stanford A dissection (same patient as on fig.12) Dissection involves the aortic root (a), and extends into brachiocephalic trunk and left subclavian artery (c). Note: beak sign (b)

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**Fig. 27:** 61 y.o. male with Stanford A dissection: Dissection involves the aortic root (a), and extends into brachiocephalic trunk, left common carotid artery and left subclavian artery (c); True lumen is narrowed, and the left kidney demonstrates a segmental hypoperfusion (d).

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Fig. 28: 44 y.o. female with traumatic aortic pseudoaneurysm: CTA, multiplanar and VRT reconstructions. Pseudoaneurysm characteristically occur along the undersurface of the aortic isthmus at or near the site of the ductus arteriosus and should not be confused with PAU. Also an important differential diagnosis include ductus diverticulum.

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**Fig. 29:** Ascending aorta pseudoaneurysm (blue arrows) in 61 y.o. man who had undergone coronary artery bypass grafting four years ago. There is also aneurysmal dilatation of ascending aorta and periaortal haematoma consistent with rupture. Note: bovine aortic arch (red arrow in d) - incidental finding; patent coronary bypass graft (white arrow in c).

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Fig. 30: 63 y.o. female with sudden onset of chest pain a) Patient was initially misdiagnosed with an aortic dissection because of aortic motion artifacts that simulated intimal flap (arrows), b) An interval ECG-Gated CTA obtained subsequently showed unremarkable findings.

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Conclusion

Given the distinctive findings on these MDCT angiographies we recommend that radiologists ask (and answer) critical questions, specifically; 1. Is there an aortic dissection, PAU, IMH? And if so, differentiate, categorize and describe the type of aortic dissection, IMH and PAU, etc.; 2. Evaluate the extent of the dissection, locate the intimal tears -place of entry and reentry; 3. Differentiate the true lumen from the false lumen, recognize signs of significant compression of a true lumen by the false lumen; 4. Assess branching arteries and iliac arteries for involvement and look for signs of end-organ hypoperfusion e.g. renal perfusion; 5. Recognize aortic rupture.

With comprehensive MDCT evaluation of AAS, a radiologist should provide answers to critical questions with pathoanatomic details. Collectively, the report is crucial in determining prognosis and providing guidance for clinicians to select correct clinical management strategies.
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References


