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REVIEW ARTICLE

The role of multidetector-row CT in the diagnosis, classification and management of acute aortic syndrome

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ABSTRACT

The term “acute aortic syndrome” (AAS) encompasses several non-traumatic life-threatening pathologies of the thoracic aorta presenting in patients with a similar clinical profile. These include aortic dissection, intramural haematoma and penetrating atherosclerotic ulcers. These different pathological entities can be indistinguishable on clinical grounds alone and may be confused with other causes of chest pain, including myocardial infarction. Multidetector-row CT (MDCT) is the current modality of choice for imaging AAS with a sensitivity and specificity approaching 100%. Early diagnosis and accurate radiological classification is associated with improved clinical outcomes in AAS. We review the characteristic radiological features of the different pathologies that encompass AAS and highlight the vital role of MDCT in determining the management of these life-threatening conditions.

The term “acute aortic syndrome” (AAS) encompasses a heterogeneous group of patients with a similar clinical profile presenting with one of several non-traumatic life-threatening pathologies of the thoracic aorta.¹ These include aortic dissection, intramural haematoma (IMH) and penetrating atherosclerotic ulcers (PAUs).^{2,3} These different pathological entities can be difficult to distinguish clinically and may be confused with other causes of chest pain, including myocardial infarction, oesophageal perforation or pulmonary embolism.^{2–4} Aortitis and traumatic aortic injury are not considered as part of the clinical spectrum of AAS, as they both demonstrate distinct pathophysiological and clinical features.^{2,3}

We provide a literature review of the role of multidetector-row CT (MDCT) in the acute assessment of patients with suspected AAS and highlight the importance of early diagnosis and accurate classification in the management of AAS.

ACUTE AORTIC SYNDROME RISK FACTORS AND CLINICAL PRESENTATION

There are many risk factors that predispose to the development of AAS, either through weakening of the aortic media or by exposing the wall of the aorta to increased hydraulic stress (Table 1).^{3,5} The most common predisposing condition is hypertension, which is reported in 72%

of patients with AAS and is often uncontrolled at the time of presentation.^{2,3,5} Younger patients with AAS (less than 40 years of age) are less likely to have a history of hypertension but are more likely to have an alternative predisposing condition such as a bicuspid aortic valve, Marfan’s syndrome, homocystinuria, Ehlers–Danlos syndrome (Type 4), Loeys–Dietz syndrome or previous aortic surgery.^{3,6–9}

AAS has a high associated mortality and morbidity rate, which mandates early diagnosis and management.^{2,10} Early diagnosis and accurate radiological classification has been shown to have a significant impact on outcome in patients with AAS.^{2,3} However, the clinical presentation of AAS may mimic other more common conditions and a high index of suspicion should be maintained particularly when risk factors are present.^{2,3,11}

AAS typically presents acutely with severe chest pain of a tearing nature in 84–90% of reported cases.^{3,11,12} Pain of maximal intensity at the time of onset favours a diagnosis of AAS as opposed to the gradual increase in intensity that typically occurs with acute coronary syndromes.^{3,11,12} Chest pain that radiates to the neck, jaw or throat may indicate involvement of the ascending aorta, whereas pain referred to the back or abdomen suggests that the descending aorta is involved.^{12–14} The presence of pulse

Table 1. Risk factors predisposing to acute aortic syndrome

Hypertension
Genetic disease
Marfan's syndrome
Loeys–Dietz syndrome
Vascular Ehlers–Danlos syndrome (Type 4)
Turner syndrome
Coarctation of the aorta
Bicuspid aortic valve
Familial thoracic aortic aneurysm and dissection syndrome
Inflammatory vascular disease
Syphilis
Behcet's disease
Takayasu arteritis
Giant cell arteritis
Aortic wall infection (bacteraemia or extension of adjacent infection)
Iatrogenic factors
Aortic catheterization
Cardiac valve or aortic surgery
Other
Crack cocaine or other stimulant abuse
Chronic corticosteroid or immunosuppressant therapy
Phaeochromocytoma
Polycystic kidney disease
Pregnancy

deficits and a regurgitant aortic murmur are other clinical findings that favour a diagnosis of AAS over other causes of chest pain.^{3,13} Syncope occurs in 13% of patients with acute aortic dissection and often heralds the development of complications associated with a high mortality, including cardiac tamponade, severe aortic regurgitation, aortic rupture or cerebral vessel occlusion.^{9,11,12,15}

Typical signs and symptoms of AAS are less common among the elderly, and an age of more than 70 years in patients has been identified as an independent predictor of hospital mortality.¹⁴ In a review of 977 patients with acute aortic dissection, 6.4% presented atypically without pain.¹⁶ These patients were typically older and presented with clinical features of heart failure, stroke or syncope rather than chest pain.¹⁶ Patients on steroid medication and those with Marfan's syndrome are also more likely to present atypically without chest pain.¹⁷

The International Registry of Acute Aortic Dissections (IRAD) is a conglomerate of international cardiothoracic surgical research centres that have combined to collate data on the presentation, management and outcomes of patients presenting with acute

aortic dissection.¹⁸ Since its inception in 1996, anonymized clinical data from patients with acute aortic dissection have been submitted to IRAD for analysis from >30 institutions across 11 countries. The information provided by IRAD has provided significant insight into the clinical presentation, management and outcome of patients with AAS.^{9,14,18}

IMAGING PROTOCOL

MDCT is the current imaging modality of choice for AAS with a reported sensitivity of 100% and specificity of 98–99%.^{3,19–23} Advantages of MDCT over other imaging modalities in the diagnosis of AAS include rapid image acquisition, near universal emergency access and the ability to image and reconstruct the entire aorta in three-dimensional planes.^{23–25} Furthermore, the ability of MDCT to accurately assess branch vessel involvement and identify aberrant vascular anatomy is vital in determining the surgical and endovascular management of AAS.^{26–28}

MDCT scanners with ≥ 64 detector rows should be used to image patients with suspected AAS, to consistently provide isotropic resolution in three dimensions, enabling the acquired image data set to be reconstructed in the optimal plane with respect to the aorta and its branch vessels.^{25,27–30} The use of electrocardiogram (ECG) gating in the evaluation of AAS is essential to reduce cardiac motion artefacts that may mimic aortic dissection, particularly in the evaluation of the aortic root and ascending aorta (Figures 1 and 2).^{4,31,32} In addition, ECG gating also enables more accurate assessment of the proximal coronary arteries in patients with confirmed aortic dissection.^{28,31} Although ECG-gated imaging of the thoracic aorta is not yet routinely performed in all institutions, it

Figure 1. Non-electrocardiogram-gated multidetector-row CT demonstrates a cardiac motion artefact mimicking a dissection flap in the left anterior aspect of the ascending aorta (white arrow). This site is typical for cardiac motion artefact.

Figure 2. Non-gated multidetector-row CT (MDCT) (a) and prospectively electrocardiogram (ECG)-gated MDCT (b) in a patient transferred to a cardiothoracic centre with suspected aortic dissection on the initial study (white arrow). The suspected dissection flap was confirmed to be cardiac motion artefact by its absence on the subsequently performed prospective ECG-gated study (b). This figure illustrates the use of ECG-gating imaging as a powerful tool to differentiate true dissection flaps from cardiac motion artefacts.

should be noted that this approach may potentially lead to the inappropriate transfer of patients to cardiothoracic surgical centres or even negative open surgery for patients with atypical cardiac motion artefact mimicking acute dissection flaps on non-ECG-gated MDCT (Figure 2).

ECG-gated pre-contrast image acquisition from above the aortic arch to diaphragmatic hiatus to evaluate for the presence of IMH and localized rupture into the pleura or pericardium is an important component of the imaging protocol for patients with suspected AAS and should be performed in all such patients.^{4,31,33} Following the unenhanced study, a bolus-tracked CT angiogram should be performed using 120 ml of 370 mg l ml⁻¹ iodinated contrast medium delivered at a rate of between 4 and 5 ml s⁻¹ via a power injector to achieve a target opacification of the aorta of >250 HU.³⁴ The volume of contrast should be reduced to 80–100 ml in elderly patients with reduced cardiac outputs.³⁴ ECG-gated acquisitions are performed prospectively at mid-diastole with a breath-hold performed whilst scanning the thoracic aorta to the level of the diaphragmatic hiatus, and the patient asked to free breath whilst the sub-diaphragmatic aorta is imaged. Unlike coronary CT angiography studies, pre-medication with β -blockers or nitroglycerine is not required and should be avoided.³¹ Patients with higher heart rates (>70 beats per minute) are prospectively gated at end-systole. Clearly, the specific MDCT parameters for an individual patient should be adjusted depending on the CT vendor used and patient-specific variables such as body mass index. It is important to ensure that contrast injection is via the right arm, as streak artefact arising from hyperdense contrast medium within the left brachiocephalic vein may impair accurate assessment of disease involvement of the head and neck vessels, if contrast is injected via the left arm (Figure 3). In order to further minimize streak artefact arising from the superior

vena cava, a saline flush of 20 ml should be power injected immediately following contrast medium injection using the same flow rate.³⁴

In the evaluation of AAS, craniocaudal coverage should extend from the thoracic inlet to the aortoiliac bifurcation.^{3,27} This range of coverage is important to diagnose major branch vessel involvement and to characterize the presence and severity of the resultant organ malperfusion.^{27,35} Extending the image coverage to include the common femoral arteries may be of use in certain cases, to help decide the site of peripheral vascular access for endovascular management or cannulation for extracorporeal circulation during surgery.^{27,35,36}

While evaluation of the source data provides the foundation of MDCT image interpretation in cases of suspected AAS, multiplanar reformat assessment of the aorta is of particular importance in evaluating the aortic root and arch for disease involvement.^{4,9,11} Two- and three-dimensional reconstruction techniques are also extremely useful in the communication of results to referring clinicians and in the planning of surgical and endovascular procedures in confirmed cases of AAS.^{3,23,27}

In patients with significantly impaired renal function or known intravenous iodinated contrast allergy, an alternative imaging strategy must be utilized in cases of suspected AAS. Both transoesophageal echocardiography and MRI may be used as an alternative to MDCT in this situation.^{2,3,19} A potential disadvantage of both techniques is the relative time required to complete these studies compared with MDCT, with potential delay to definitive treatment as a result.^{23,37} Unenhanced MDCT as an initial screening tool prior to further evaluation with MRI or transoesophageal echocardiography

Figure 3. Streak artefact arising from dense contrast media within the left brachiocephalic vein. While a dissection flap extending into the left subclavian artery is demonstrated (white arrow), the left common carotid and brachiocephalic arteries are partially obscured by streak artefact. Left-arm contrast injections should be avoided in patients imaged for suspected acute aortic syndrome.

may be used to expedite intervention in selected cases. Unenhanced MDCT has been shown to be accurate in the assessment of aortic size and the detection of IMH, intimal calcification displacement and localized rupture into the pericardial or pleural spaces.³⁷

Although MDCT is the principal imaging modality in the acute assessment of AAS, MRI has a significant role in the long-term imaging follow-up of these patients, particularly in young patients or patients with bicuspid aortic valve with associated aortic incompetence.²⁸

ACUTE AORTIC SYNDROME IMAGING APPEARANCES

Acute aortic dissection

Classic aortic dissection originates from a disruption of the aortic intima and inner layer of the aortic media, creating an entrance tear that enables blood to split the aortic media.^{2,38–40} This results in the formation of a double-channel aorta, with a dissection flap dividing the aorta into true lumens (TLs) and false lumens (FLs) (Figures 4 and 5).^{2,38} This so-called “intimal flap” is a misnomer, as the flap tissue is also composed from media tissue stripped from the wall of the aorta and therefore is more accurately termed the intimomедial flap.³⁸ As the proportion of media involved in the intimomедial flap increases, the external wall of the FL becomes thinner, resulting in an increasing risk of catastrophic rupture.⁴¹ The entrance tear is most commonly seen at the sites of the greatest hydraulic stress, located at the right lateral wall of the ascending aorta or the proximal segment of the descending aorta.^{4,38} Many patients have a re-entrance tear and several fenestrations between the TL and FL along the course of the dissected segment of the aorta.^{38,42}

The cardinal features of aortic dissection on MDCT are the presence of an intimomедial flap and a double-channel aorta (Figures 4 and 5).^{4,25,28,32} Once the diagnosis of acute aortic dissection has been made on MDCT, the key is to classify the origin and extent of the dissection, to differentiate the TLs and FLs and to assess for branch vessel involvement and complications.^{3,4,25}

It is important to remember that cardiac motion and streak artefacts may mimic a dissection flap.⁴³ Streak artefacts arising from high-density structures, such as metallic leads or dense contrast media within adjacent veins, can traverse the lumen of the aorta to simulate a dissection flap.⁴³ While a genuine dissection flap is typically thin, with a uniform thickness and consistent orientation on contiguous slices, streak artefacts typically demonstrate an inconsistent orientation on contiguous slices and characteristically radiate away from a high-density focus, with alternating low and high linear densities that increase in width as they fan out (Figure 6).⁴³ Cardiac motion artefact may be differentiated from true aortic dissection by its characteristic position in the right posterior and

Figure 4. Multidetector-row CT (a) and sagittal multiplanar reformat (b) demonstrate the typical appearance of a Type A aortic dissection. FL, false lumen; TL, true lumen.

Figure 5. Multidetector-row CT (a) and sagittal multiplanar reformat (b) demonstrate typical appearance of a Type B aortic dissection. Note the presence of calcification within the outer wall of the true lumen and the intimomedial flap (white arrows) but not in the outer wall of the false lumen (FL). TL, true lumen.

left anterior positions of proximal aorta (Figure 1).^{25,30} The use of ECG gating will circumvent this potential issue (Figure 2).^{4,25,30}

A number of features help accurately differentiate the FLs and TLs. The FL typically demonstrates slower flow and is usually larger, although this is not always a reliable discriminator.^{28,29} A more specific, although uncommon, discriminator is the presence of low attenuation thin strands within the FL, which represent residual threads of incompletely dissected

media tissue and has been termed the “cobweb sign” (Figure 7).^{4,28,31} The most consistently identified feature of the FL is the “beak sign”, demonstrated when the intimomedial flap forms an acute angle with the outer wall of the FL (Figure 8).^{4,25} Another important defining characteristic of the FL is the lack of outer wall calcification, which may be seen within the wall of the TL (Figure 5).^{29,30} The absence of outer wall calcification also helps distinguish a thrombosed FL of an aortic dissection from a wall adherent thrombus of an aortic aneurysm. Accurate differentiation of the TL and FL

Figure 6. Classic appearance of streak artefact arising from dense contrast medium within superior vena cava mimicking an aortic dissection flap. Note the alternating low and high linear densities that increase in width as they fan out away from the high-density source.

Figure 7. Cobweb sign. Low attenuation strands (white arrow) within the false lumen (FL) represent residual threads of incompletely dissected media and are only seen within the FL. TL, true lumen.

Figure 8. Beak sign. The intimal flap forms an acute angle with the outer wall of the false lumen (FL) (white arrow). TL, true lumen.

is of particular significance when endovascular therapy is being considered.^{29,33,44}

Death from aortic dissection is usually the result of major branch vessel occlusion, pericardial tamponade, acute aortic regurgitation or aortic rupture.^{3,5,14} The risk of fatal aortic rupture in patients with dissection involving the ascending aorta is high, with the vast majority occurring in the pericardium, left pleural cavity or mediastinum.^{5,33} The presence of hyperattenuating fluid collections

Figure 9. Rupture of aortic dissection with high attenuation fluid within the pericardium on the initial unenhanced CT study (white arrow).

within these sites on pre-contrast MDCT scans is indicative of aortic rupture (Figure 9).^{4,29,33} Evolving cardiac tamponade secondary to a pericardial haematoma may be inferred by compression of the free wall of the right ventricle.²⁸ In addition, the distance from the dissection flap to the aortic valve should be assessed for all dissections that involve the aortic root, as these patients are at increased risk of aortic valve insufficiency and aortic valve rupture.^{4,25}

Approximately one-third of patients with aortic dissection demonstrate clinical features secondary to end-organ ischaemia caused by branch vessel obstruction.^{3,33} This is commonly owing to obstruction by the dissection flap, which may protrude across the origin of the branch vessel (dynamic occlusion) or directly extend into the wall of the branch vessel (static obstruction) (Figures 10 and 11).^{2,29} Maximum intensity projection reconstructions provide a useful adjunct to axial sections in defining the relationship of the intimomedial flap to the branch vessels and aid identification of end-organ malperfusion.^{29,45,46}

Careful assessment of all the major aortic branch vessels is essential in patients with confirmed aortic dissection and should particularly focus on whether the dissection flap extends into the vessel and whether the branch vessels arise from the TL or FL.^{4,25,28} ECG gating aids accurate assessment of the anatomy of the coronary ostia, the relationship of the coronary ostia to the TLs and FLs and dissection flap extension into the proximal coronary arteries.³¹ It is vital to evaluate the head and neck arteries for disease involvement as these patients have a poorer prognosis.²⁸ Specific comment on the anatomy of the head and neck vessels should be made on patients with acute dissection, as the presence of aberrant subclavian arteries may necessitate alternative approaches to cardiac bypass. The relationship of the origin of the celiac axis, superior mesenteric artery and both renal arteries with the TLs and FLs and the presence of dissection extension into these vessels should be defined, as these patients are at risk of

Figure 10. Aortic dissection with the left renal artery (black arrow) arising from the false lumen with hypoattenuation of the left kidney implying significantly impaired left renal perfusion.

Figure 11. Multidetector-row CT (a) and sagittal multiplanar reformat (b) images from a patient with aortic dissection and malperfusion of the superior mesenteric artery (white arrows) in a patient with associated small bowel infarction.

significant end-organ ischaemia and dysfunction.^{28,31} Finally, the iliac and common femoral arteries should be interrogated for dissection flap extension, not only to identify patients at risk of distal limb ischaemia but also to determine the appropriate vessel to facilitate cardiac bypass in those patients requiring operative intervention.^{27,35,36}

Intramural aortic haematoma

IMH accounts for 10–30% of patients with AAS.^{47,48} Traditionally, IMHs were thought to be characterized by the presence of haemorrhage within the tunica media, presumably owing to rupture of the vasa vasorum, with no evidence of intimal disruption or tear unlike an aortic dissection or PAU.^{28,38} However, in recent years, there has been a greater appreciation in the surgical and imaging literature that IMH and aortic dissection are less pathologically distinct than previously described and that, in reality, IMHs are likely to result from microscopic tears in the intima, which are seen in the majority of patients with IMH at surgery or autopsy.^{4,31} With recent advances in modern imaging technology, small focal communications between the aortic lumen and the aortic wall haematoma are now readily delineated on MDCT in the majority of patients with IMH.^{4,31}

On unenhanced CT, an IMH appears as a crescent-shaped area of high attenuation thickening in the aortic wall. Typically, this crescent demonstrates an attenuation coefficient of >45 HU.⁴⁹ The IMH remains unenhanced after contrast injection and no dissection flap is seen (Figure 12).^{4,50} Multiplanar reconstructions can be of benefit in anatomically differentiating haemorrhagic fluid within the superior pericardial recess from genuine IMH.

The clinical course of an IMH is rather unpredictable and may resolve ($<10\%$) or progress over time to classic aortic dissection, aneurysm formation and rupture.⁵¹ There are a number of features that can help risk stratify patients with IMH (Table 2).^{50–55} The most important of these are the involvement of the ascending aorta and a maximum aortic diameter ≥ 5.0 cm.^{52–54} In addition to these features, the presence of large, haemorrhagic or enlarging pleural or pericardial effusions is suggestive for disease progression.^{2,52}

It is important to differentiate ulcer-like projections from intramural blood pools in patients with IMH. An ulcer-like projection appears as a localized contrasted filled pouch that communicates with the TL, whereas an intramural blood pool

Figure 12. Unenhanced CT (a) demonstrates crescent of high attenuation within the wall of the aorta (white arrows) that does not enhance with contrast (b). This is the typical appearance of an intramural haematoma.

Table 2. Features indicating high risk of disease progression in intramural haematoma

Involvement of ascending aorta
Haematoma thickness >11 mm
Aortic diameter >5 cm
Associated penetrating atherosclerotic ulcer with a diameter of >20 mm or a depth of >10 mm
Large or progressive enlargement of pleural or pericardial effusion
Recurrent chest pain
Progressive enlargement of intramural haematoma on serial imaging

has a very narrow intimal orifice, typically communicates with a lumbar or intercostal artery and does not demonstrate a communication with the TL.⁵⁶ While usually not evident on the initial presentation CT, the development of ulcer-like projections is associated with a poorer prognosis in patients with IMH. Conversely, intramural blood pools are usually present on the initial CT, generally resolve (partially or completely) over interval follow-up and are not typically associated with adverse events in patients with IMH.⁵⁶

Overall, the 5-year mortality of this condition approaches 50%, and owing to the risk of disease progression, follow-up cross-sectional imaging is crucial in patients treated conservatively.^{2,51,52} Interestingly, there is significant international geographic heterogeneity in clinical outcomes of IMH with significantly better mortality rates reported among patients managed conservatively in the Far East than those in Western populations, the cause of which remains unclear to date.²

Penetrating atherosclerotic ulcers

PAUs arise from atherosclerotic aortic lesions that ulcerate and penetrate the internal elastic intima into the media.^{2,38} These may be singular or multiple and are most commonly located within the middle or distal thirds of the descending aorta.^{2,57} PAUs differ from simple ulcerated atherosclerotic plaques by the presence of intimal disruption with subsequent extension of blood into the media.²⁸

A PAU is diagnosed by demonstrating a focal contrast filled out pouching of the aortic wall with jagged edges usually in the presence of extensive aortic atheroma (Figures 13 and 14).^{4,28} Deeper ulcers are often associated with an IMH, and concomitant aneurysms of the thoracic aorta are common.⁵⁷ PAUs and uncomplicated atherosclerotic ulcers share some similar morphological features on MDCT. Uncomplicated simple atherosclerotic ulcers are confined to the intimal layer and are differentiated from PAU on MDCT by the absence of contrast extension beyond the level of the intima.^{4,28,38}

The management of PAU is controversial, with some authors advocating an expectant conservative approach. In 1 study of 105 patients presenting with PAU, involving the aortic arch or descending thoracic aorta, 72% were successfully managed

conservatively. The late survival rate was comparable between patients treated conservatively and those who underwent surgery.⁵⁸ Features predictive of failure of conservative therapy include aortic rupture at presentation and disease involvement of the ascending aorta.^{58,59} Other imaging findings that indicate a need for urgent intervention include extra-adventitial blood, a grossly bulging IMH, progressive haemorrhagic pleural effusion and subadventitial disease.⁶⁰

Currently, there are only few data available in the literature regarding the natural history and follow-up of patients with PAU, particularly on those diagnosed in asymptomatic patients as an incidental imaging finding.⁶¹ One study of 87 patients

Figure 13. Multidetector-row CT (a) and sagittal multiplanar reformat (b) images demonstrating focal out pouching of luminal contrast into the wall of the descending aorta (black arrows). This is the typical appearance of a penetrating atherosclerotic ulcer.

Figure 14. Multidetector-row CT (a), sagittal multiplanar reformat (b) and volume-rendered reconstruction (c) images demonstrating a penetrating atherosclerotic ulcer (white arrows). Multiplanar reformat and volume-rendered images can be of benefit in planning surgical and endovascular interventions in patients with confirmed acute aortic syndrome.

with PAU treated conservatively over a mean imaging follow-up period of 13.9 months, demonstrated that patients with symptomatic PAU were significantly more likely to show disease progression than asymptomatic patients with PAU (43% compared with 17%) and were far more likely to require intervention (36% compared with 8%).⁶² While further work is required in this area, the current limited data suggest that patients with PAU managed conservatively warrant follow-up with imaging to monitor disease progression and to assess for complications that necessitate invasive treatment.^{2,61,62} Complications include false aneurysm formation, focal limited dissection and rupture.^{2,57} Features indicating the need for intervention in patients undergoing follow-up include recurrent or refractory pain, aortic diameter >55 mm, an annual increase in aortic diameter by >10 mm and the presence of a periaortic haematoma.⁶³

CLASSIFICATION AND MANAGEMENT OF ACUTE AORTIC SYNDROME

AAS may be defined as acute if the duration of symptoms is less than 2 weeks and chronic if more than 2 weeks, with the greatest morbidity and mortality occurring during the acute period.^{35,64} AAS is further classified anatomically based on the extent of involvement of the thoracic aorta.³ The original classification was based on the origin of the intimal tear (DeBakey classification system) and was purely concerned with aortic dissection.³ This has since been superseded by the Stanford classification system, which applies to all the conditions that comprise AAS and divides them into two groups based on whether the site of aorta involved is proximal or distal to the origin of the left subclavian artery (Figure 15).^{3,65}

Accurate radiological classification is vital, as it determines the need to proceed with emergency surgery or conservative management.^{3,33}

Stanford Type A dissections account for 75% of all aortic dissections and if untreated have a mortality of 1–2% per hour after the onset of symptoms.^{66,67} The non-operative treatment of patients with acute Type A dissections is associated with a mortality of 24% within 24 h of symptoms and 49% by Day 14.⁵ It is important to note that the management of type A dissections limited to involve just the arch of the aorta ($\pm$ the descending thoracic aorta) distal to the origin of the brachiocephalic artery

Figure 15. Schematic illustrating the classification of acute aortic syndrome.

remains a matter of debate.^{68,69} Some authors have advocated a non-operative management approach in Type A arch dissections that do not involve the ascending aorta, leading to recent proposals to revise the Stanford classification system based on disease involvement proximal to (Type A) and distal to (Type B*) the origin of the brachiocephalic artery and distal to the left subclavian artery (Type B).⁷⁰

Type A IMH is also associated with a high risk of progression to aortic dissection and aortic rupture if left untreated, with an early mortality rate of 8% with surgical repair vs 55% with medical therapy in Western populations.^{51,71,72} Although there are few published data concerning the outcome of Type A vs Type B PAU, current consensus again is that management based on the Stanford classification is appropriate.⁷³ Therefore, a diagnosis of acute Type A AAS mandates immediate referral to tertiary cardiothoracic surgical units for consideration of emergency operative repair.^{2,3,28}

Although most authors advocate that the decision to proceed with surgery for IMH should be determined by the Stanford classification, a few centres have cautiously adopted a “timed urgent surgery” approach to a subgroup of patients with Type A IMH (Table 3).^{74,75} Following initial CT diagnosis, these patients are managed by close inpatient observation for a minimum of 30 days in a tertiary cardiothoracic surgical unit with the decision to proceed to surgery being determined by the development of ominous features on serial imaging (Table 3). This approach

Table 3. Timed surgery criteria for Type A intramural haematoma

Criteria for observation
Haemodynamically stable
Aortic diameter <5 cm
Haematoma thickness <10 mm
No PAU or ulcer-like projections
No large or progressive pericardial or pleural effusion
No evidence of tamponade
No persistent pain
No evidence of end-organ malperfusion secondary to branch vessel involvement
Indications for “timed surgical intervention”
Pain
Increasing aortic diameter
Increasing thickness of intramural haematoma
Increasing pericardial or pleural effusion
Progression to aortic dissection
Development of PAU or ulcer-like projections
Development of tamponade
Development of end-organ malperfusion secondary to branch vessel involvement

PAU, penetrating atherosclerotic ulcer.

requires a protocol of daily transthoracic echocardiography examinations for the first 5 days after diagnosis, followed by repeat CT at Day 5 and then weekly CT or transthoracic echocardiography for a total of 30 days.^{74,75} Although this approach has been successfully applied in centres in Japan and the Republic of Korea, it has yet to be validated in studies of Type A IMH in Western populations.⁷⁵

Owing to a significantly lower risk of fatal aortic rupture and the high morbidity and mortality associated with operative repair of the descending aorta, medical management alone is advocated for uncomplicated Type B dissection with a reported 30-day mortality of 10%.^{35,76} A number of factors have been associated with a greater risk of aortic dilation and adverse outcomes in patients treated conservatively for uncomplicated Type B aortic dissections (Table 4).^{76–79} Patients who develop complications, including disease progression, contained rupture, and renal and mesenteric ischaemia often require more invasive management.^{35,36,80} However, patients who undergo surgery for these indications have a high rate of morbidity with a reported risk of paraplegia of 19% and mortality of 31%.^{14,35,80} Endovascular procedures, including aortic stent grafting, dissection flap fenestration and branch vessel stenting, provide an alternative approach to the management of these patients with a reported 30-day mortality of 6.2% and a neurological complication rate of 2.9%.^{81–86} Reintervention rates of 7.2% and mortality rates of 11.4% reported within 12 months of initial successful thoracic aortic endovascular procedures highlight the importance of close clinical and imaging follow-up in this cohort of patients.⁸⁵

Patients with type B IMH or PAU who present without rupture or imaging features that suggest high risk of rupture can be successfully treated conservatively.⁶⁰ However, it is important to recognize that this cohort of patients have a higher associated mortality and greater risk of rupture than those with classic Type B aortic dissection. As a result, close follow-up with serial imaging and a lower threshold for intervention is required in these patients.^{60,87} As for Type B classic aortic dissection, the initial published experience of endovascular treatment of Type B IMH or PAU is promising with excellent technical success rates and

Table 4. Risk factors for aortic dilation in uncomplicated Type B aortic dissection

Male
Marfan syndrome
Aortic diameter ≥40 mm (initial CT)
Fusiform dilation of proximal descending thoracic aorta
Elliptical shape of true lumen
Saccular FL
Partial thrombosis of FL
FL located on inner curvature of aorta
Single entrance tear
Large entrance tear >10 mm within the proximal aspect of the dissection

FL, false lumen.

relatively low mortality and complication rates reported in the literature to date.^{60,63,82,84,85,87}

CONCLUSION

This review highlights the characteristic MDCT features of the conditions that constitute AAS and demonstrates the vital role of

MDCT in the classification and management of these life-threatening conditions.

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