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REVIEW

Non-traumatic thoracic aortic emergencies: imaging diagnosis and management

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Abstract

The major component of non-traumatic thoracic aortic emergencies is the acute aortic syndromes. These include acute aortic dissection, intramural haematoma and penetrating atherosclerotic ulcer, grouped together because they are indistinguishable clinically and highly fatal. All three entities involve disruption to the tunica intima and media and may be complicated by rupture, end-organ ischaemia or aneurysmal transformation. Early diagnosis is vital to allow timely and appropriate management. Paired unenhanced and electrocardiogram-gated computed tomography angiography of the chest, extending more distally if required, is recommended for diagnosis. Specific computed tomography features of all three entities are reviewed, with a focus on morphological features associated with complications. Those with type A pathology are usually managed with open surgery because this has a high risk of complication. Patients with uncomplicated type B pathology are usually managed with best medical therapy whereas those with complicated type B pathology are usually offered either surgery or thoracic endovascular aortic repair. The limited evidence regarding the use of thoracic endovascular aortic repair in patients with subacute uncomplicated type B pathology is briefly discussed.

Key words: Acute disease; Dissecting aneurysm; Aortic diseases; Aortic rupture; Diagnostic imaging

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Introduction

Thoracic aortic emergencies include aortic transection, ruptured aortic aneurysm and acute aortic dissection. Variants of aortic dissection have classically been grouped together as acute aortic syndromes because they are clinically indistinguishable on presentation. These include acute dissection, intramural haematoma and penetrating atherosclerotic ulcer (Vilacosta and San Román, 2001). Pathologically, all three entities involve disruption to the tunica intima and media, leading intramural blood to dissect through the layers of the aorta, or pass transmurally during rupture. Indeed, all three conditions may co-exist when imaged, rendering it impossible to know which came first.

This article primarily focuses on the imaging of non-traumatic acute aortic syndromes, but briefly reviews the evidence for management of these conditions. Multidetector computed tomography is most commonly used to make the diagnosis, with sensitivity approaching 100%, so will be the focus of this article (Sebastià et al, 1999). While complementary, magnetic resonance imaging, transoesophageal or transthoracic echocardiogram findings are not discussed.

The two traditional subdivisions of acute dissection are the Stanford and DeBakey classifications. The Stanford classification divides aortic dissection into type A (involving the ascending aorta with or without extension into the descending aorta) and type B (involving the descending aorta distal to the origin of the left subclavian artery only). The DeBakey classification divides aortic dissection into type 1 (both ascending and descending aortic involvement), type 2 (ascending aorta only) and type 3 (descending aorta only). The same anatomical classification applies to intramural haematomas and penetrating atherosclerotic ulcers and, unless otherwise stated, the Stanford classification is used.

Risk factors and presentation

Data from the International Registry of Acute Aortic Dissection, a multinational database incorporating over 7300 cases referred to tertiary centres, show that almost two thirds

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of those presenting with acute dissection were male, with a mean age 62±15 years (type A) and 64±14 years (type B). The most common risk factors were hypertension (77%), atherosclerosis (27%) and known aortic aneurysm (16%) (Pape et al, 2015).

Severe pain of acute onset was the most common presenting complaint (≥93% of type A or B acute dissection). Patients with type A acute dissection usually present with chest pain (85% vs 65% in type B) and patients with type B acute dissection more often present with back pain (70% vs 43% in type A acute dissection). Pulse deficits, defined by decreased or absent carotid or peripheral pulses as noted by clinicians, occurred in 31% of patients with type A vs 19% of those with type B acute dissection, and were associated with higher rates of in-hospital complications (Pape et al, 2015).

Conditions associated with abnormal connective tissue (Marfan, Loeys–Dietz, Ehlers–Danlos syndromes, familial thoracic aneurysm and dissection), inflammatory aortitis, Turner syndrome or bicuspid aortic valves are all associated with early onset acute dissection. Accordingly, in those presenting with acute dissection below the age of 40 years, the most common risk factors were bicuspid valve and Marfan syndrome. Furthermore, the majority of patients with Marfan and Loeys–Dietz syndrome carry a lifelong risk of aortic aneurysm and subsequent dissection, with early elective aortic root replacement an established preventative strategy against aortic catastrophe.

Patients with intramural haematoma comprise approximately 7% of patients presenting with acute aortic syndrome. Patients tended to be older than those with acute dissection (mean age 69±10 years), with type B intramural haematoma more common than type A (60% vs 40%) (Evangelista et al, 2005).

The overall incidence of penetrating atherosclerotic ulcers in patients presenting with acute aortic syndrome is estimated to be between 2.3% and 7.6% (Coady et al, 1998). In the largest series of penetrating atherosclerotic ulcers to date, patients tended to be male (60%), even older (mean age 73±9 years), with fewer penetrating atherosclerotic ulcers affecting the aortic arch (7%), compared to the descending thoracic and abdominal aorta (93%). Only a minority (18%) presented symptomatically and hypertension (78%) was the most common risk factor (Nathan et al, 2012).

Image protocolling for thoracic aortic emergencies

Plain chest radiography is typically the initial imaging test performed in the evaluation of acute chest pain. In patients with aortic dissection it may depict mediastinal widening, pleural effusion, blurring of the aortic knuckle and displaced intimal calcification. However, its poor sensitivity and specificity means it has a limited role in definitive diagnosis.

The increasing availability of computed tomography, particularly out-of-hours, means it is the primary modality used for investigation for acute aortic syndrome, with better patient tolerance and shorter acquisition time compared to other modalities. Unenhanced computed tomography of the thorax is vital to differentiate acute intramural haematoma from pre-existing intraluminal thrombus or atheroma. This is followed by computed tomography angiography of the chest, extending more distally if required. The combination of these methods has a pooled sensitivity and specificity close to 100% (Sebastià et al, 1999).

Motion-free (usually meaning electrocardiogram-gated) computed tomography enables the best evaluation of the aortic root and ascending aorta, compared to non-gated studies, where pulsation artefact may mimic a dissection (Roos et al, 2002). When computed tomography technology allows, electrocardiogram-gated computed tomography should be acquired prospectively in late diastole (or in systole if the heart rate is significantly greater than 65 bpm). A prospective acquisition reduces the ionising radiation dose but is susceptible to artefact when the rhythm is irregular (Ohnesorge et al, 2000). Retrospective gating allows for image capture throughout the cardiac cycle, thus enabling image selection at any point along the cardiac cycle (Roos et al, 2002). The coronary arteries are more consistently visualised and information on cardiac function is provided, but this is at the expense of a considerably higher radiation dose so is generally not used (Ohnesorge et al, 2000).

Imaging of acute aortic dissection

Acute dissection is characterised by a tear in the tunica intima of the aorta, with resultant separation of the aortic wall layers. Accumulation of blood in the tunica media results in a true and a false lumen, separated by an intimal and partial medial dissection flap. The purpose of computed tomography in the evaluation of acute dissection is to determine the location of an intimal flap, to classify the anatomical type of dissection (A or B). In addition, computed tomography can identify the entry or re-entry point of the dissection, differentiate the true and false lumens, and evaluate for complications such as branch vessel involvement, end-organ ischaemia, aortic rupture, or aortic root involvement (with resultant aortic regurgitation).

The classical intimal flap on computed tomography angiography is a linear hypodense structure flanked by contrast within the lumen, present in around 70% of cases of dissection (Figure 1) (Sebastià et al, 1999). In the remaining cases where the false lumen is thrombosed, the intimal flap is not obviously demonstrated. On unenhanced computed tomography, there may be inward displacement of calcified atheroma on the intima into the lumen and high attenuation of the false lumen, representing clotted haematoma in acute cases.

In acute dissection, the true lumen is characterised by:

- Continuity with the non-dissected aortic lumen
- Higher contrast attenuation compared to the false lumen
- Typically smaller than the false lumen
- The adjacent dissection flap predominantly forming an obtuse angle with the aortic wall.

With contrast the false lumen may be identified by the presence of the beak sign – the acute angle formed by the edge of the outer aortic wall and the intimal flap (Figure 2) (LePage et al, 2001). The cobweb sign is specific for the false lumen and is characterised by fibroelastic cords projecting from the false lumen wall, thought to represent residual ribbons of media that have been incompletely sheared from the aortic wall during dissection (Williams et al, 1994).

There are multiple complications associated with acute dissection. Dissections involving the ascending aorta may extend into the coronary artery, resulting in occlusion and acute myocardial infarction (Figure 3). Other branch vessel involvement can result in end-organ ischaemia presenting as stroke, renal infarction or bowel ischaemia (Figure 4). Aortic rupture, secondary to dissection, may be indicated by the presence of haemopericardium or haemothorax, with or without cardiac tamponade (Figures 5 and 6). Cardiac tamponade is characteristically seen on computed tomography as dense pericardial fluid with flattening of the right heart, straightening of the interventricular septum, dilation of the superior and inferior vena cava, with reflux of contrast into the inferior vena cava and hepatic veins (Figure 5). The risk of aortic rupture in the context of dissection is increased by the administration of thrombolytic agents, which may be inadvertently administered while attempting to treat suspected stroke or myocardial infarction.

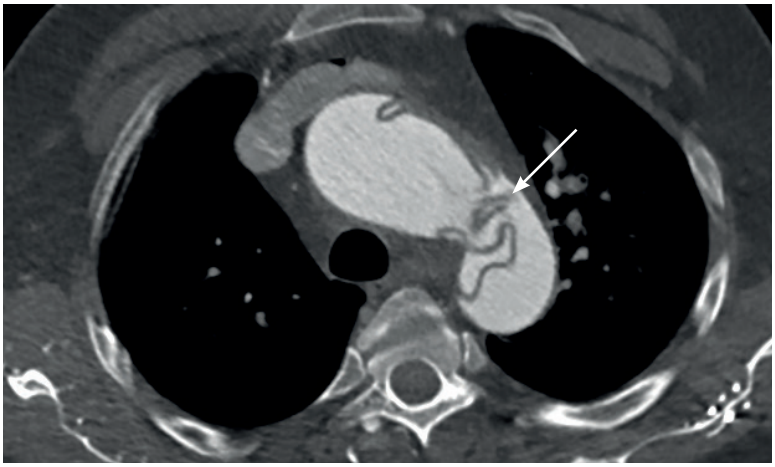

Figure 1. Axial section of a computed tomography angiogram through the aortic arch showing the ribbon-like filling defect consistent with a dissection flap (arrow).

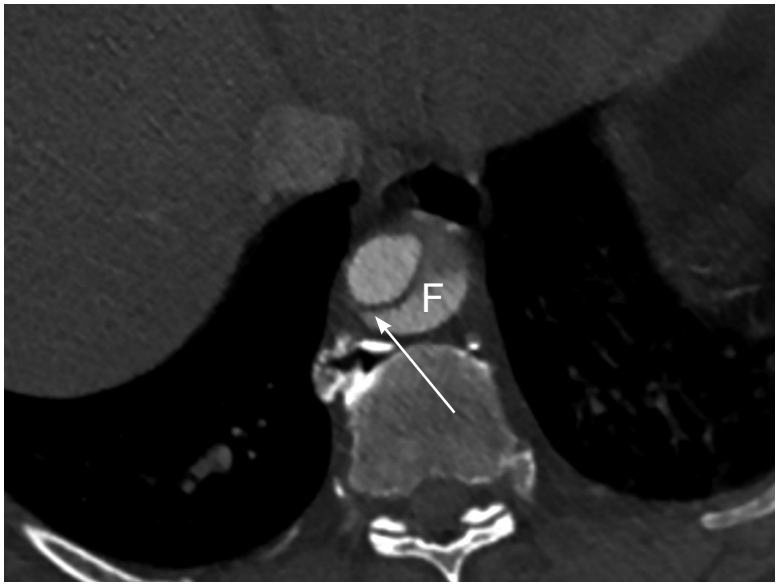

Figure 2. Axial section through the mid ascending and descending aorta on a computed tomography angiogram, demonstrating the acute angle between the dissection flap and the aortic wall (arrow). This acute angle or beak sign occurs on the side of the false lumen (F).

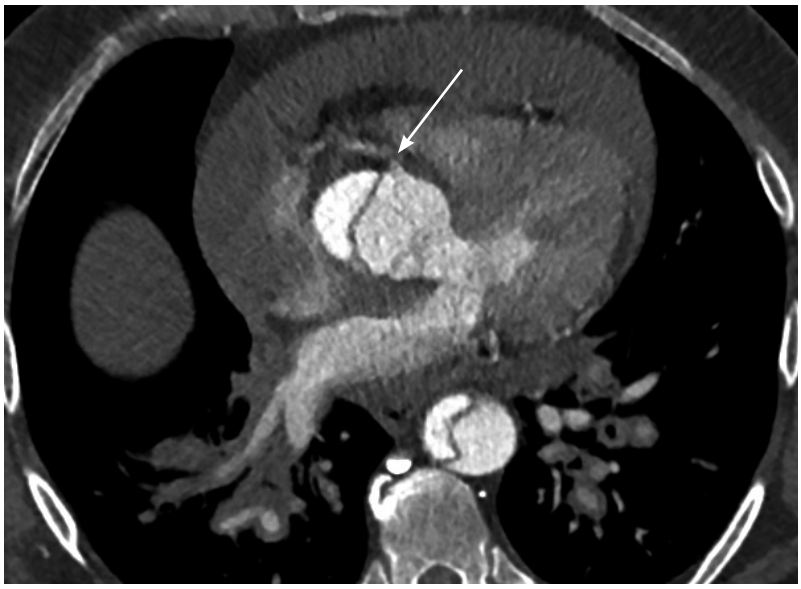

Figure 3. Axial section through the aortic sinus of Valsalva (root) on a computed tomography angiogram demonstrating the dissection flap extending close to the origin of the right coronary artery (arrow). When this extends into the coronary artery it can cause acute occlusion and infarction.

Imaging of intramural haematoma

Traditionally, intramural haematoma was thought to develop secondary to the rupture of the vasa vasorum within the tunica media. More recently, intramural haematoma is thought to arise from small entry tears within the intima, which may subsequently reseal or heal, making them difficult to detect on imaging (Chin et al, 2018).

Intramural haematoma is characterised on unenhanced computed tomography by crescenteric, high-attenuation, aortic wall thickening (typically >5 mm), which does not enhance following administration of contrast (**Figure 7**). As stated previously, type B intramural haematoma is more common than type A, which is thought to be a result of the strong association between intramural haematoma and atherosclerosis (Harris et al, 2012).

Similar to those with acute dissection, patients with type A intramural haematoma are at higher risk of complications. Multiple prospective studies have demonstrated that

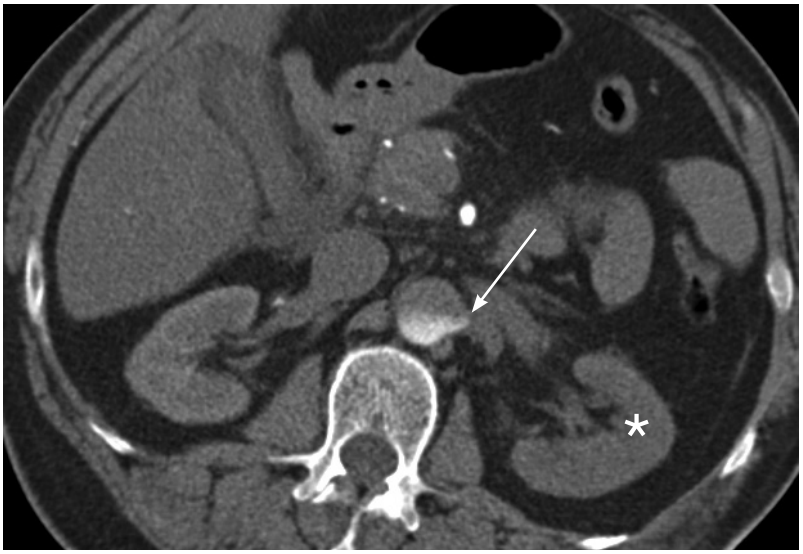

Figure 4. Axial section of a computed tomography angiogram at the level of the renal arteries. The dissection flap extends into the left renal artery (arrow) resulting in reduced enhancement of the left kidney (asterisk) compared to the right kidney.

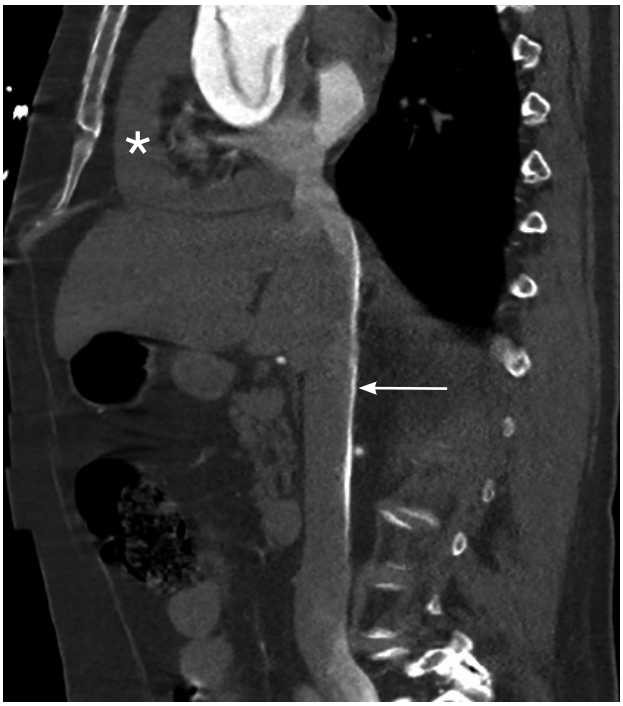

Figure 5. Sagittal section through the inferior vena cava on a computed tomography angiogram. Haemopericardium (asterisk) results in high pressure in the pericardium, preventing normal filling of the right atrium and ventricle, so diverting the contrast into the inferior vena cava (arrow).

intramural haematoma either reabsorbs or is complicated by progression to dissection, periaortic bleeding or contained rupture and aneurysm formation (Evangelista et al, 2004). A maximum aortic diameter of over 40 mm is thought to predict the progression of intramural haematoma to dissection (Sueyoshi et al, 1997). Specifically, type A intramural haematoma is associated with progression to dissection in 15–87.5% of cases (Choi et al, 2001). Other factors that increase the risk of progression of intramural haematoma to dissection include intramural haematoma thickness >10 mm, compression of the true aortic lumen and the presence of progressive pericardial or pleural effusion (Choi et al, 2001).

Ulcer-like projections are a common sequela of intramural haematoma and are another risk factor for progression. Ulcer-like projections may be complicated in up to 70% of

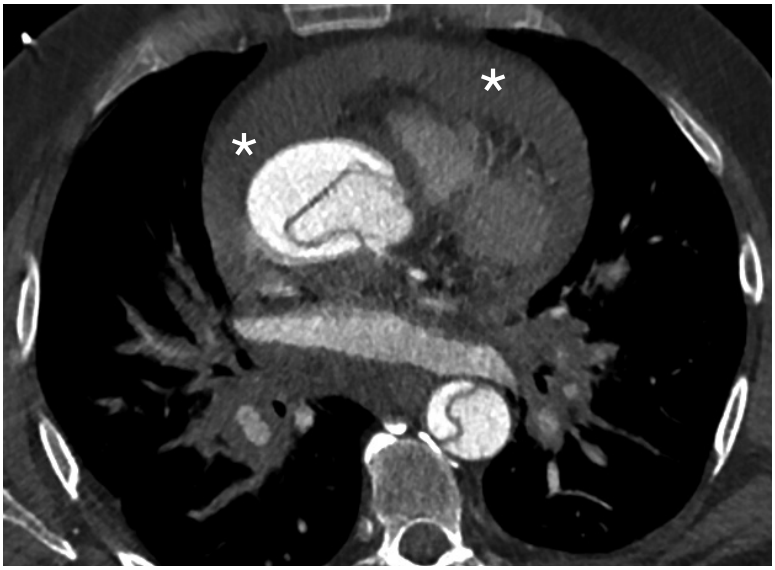

Figure 6. Axial section of a computed tomography angiogram at the level of the heart, showing a haemopericardium (asterisk) secondary to aortic dissection.

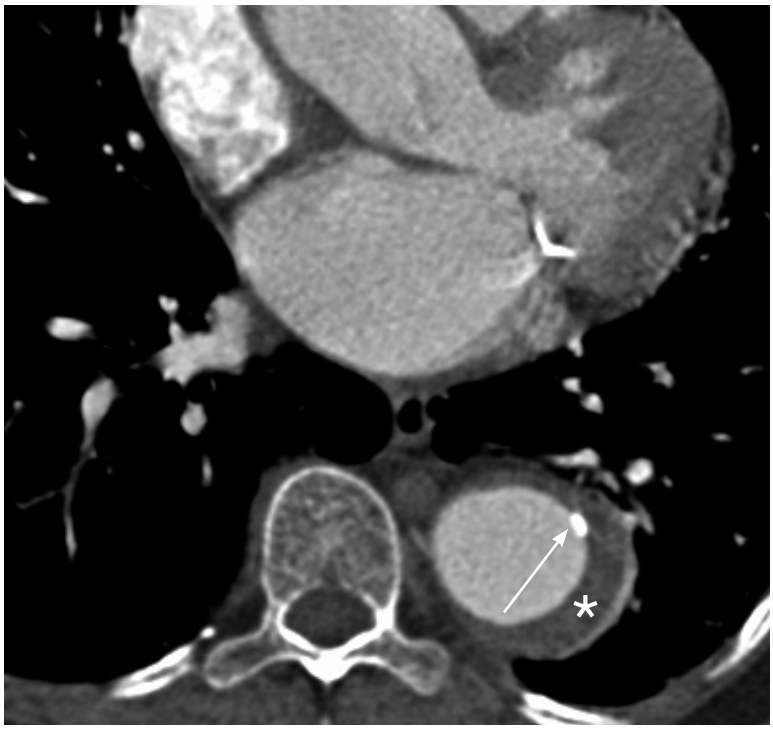

Figure 7. Axial section of a computed tomography angiogram at the level of the mid descending aorta showing an intramural haematoma (asterisk) (in the acute phase this will appear more dense than the true lumen on a scan without contrast). A useful sign is seeing the displaced intimal calcification into the true lumen (arrow).

cases by enlargement, aneurysm, dissection and rupture (Ganaha et al, 2002; Sueyoshi et al, 2002; Kaji et al, 2003). First described in 1965 by Eyler and Clark, ulcer-like projections are thought to represent a defect in the intima, leading to a clotted false channel. The defect may represent a new intimal defect, or a pre-existing intimo-medial defect not seen on initial imaging as a result of thrombosis of the false lumen. On post-contrast images, an ulcer-like projection appears as a contrast-filled pouch which extends into the thrombosed aortic wall and enhances to the same degree as the lumen. Ulcer-like projections are most often seen in areas without atherosclerosis and are not seen on initial imaging, which helps in distinguishing these from a penetrating atherosclerotic ulcer (Sueyoshi et al, 2002).

Aneurysmal dilation of the aorta is the most frequent long-term complication of intramural haematoma, defined as a 50% increase in the normal aortic diameter (Evangelista et al, 2005). Periaortic bleeding is seen as high attenuation fluid outside the outer aortic wall and is thought to be more frequently seen as a complication of a type A intramural haematoma than dissection (Song et al, 2001). Of patients with type A intramural haematoma, 3.3% presented with ST-elevation myocardial infarction secondary to periaortic haematoma causing extrinsic compression of the coronary arteries (Alomari et al, 2014).

Imaging of penetrating atherosclerotic ulcer

A penetrating atherosclerotic ulcer is defined as an atheromatous plaque with disruption of the intima and extension of blood into the media. Typical patients are older, with multiple comorbidities and extensive atherosclerotic disease. A penetrating atherosclerotic ulcer may be seen as a contrast-filled pouch extending into the media, typically within the arch and descending aorta (Figure 8). There is usually outpouching of the outer aortic wall, with jagged edges (Vilacosta et al, 1998). This distinguishes it from an uncomplicated atheromatous ulcer, which is solely limited to the intima. It is also often seen in conjunction with varying degrees of intramural haematoma.

Complications of penetrating atherosclerotic ulcer include aneurysm formation, complete erosion through the media resulting in pseudoaneurysm formation, and aortic dissection (Benitez et al, 1995). Rates of aortic rupture secondary to penetrating atherosclerotic ulcer have been reported at 4.1–38% (Tittle et al, 2002; Nathan et al, 2012). The high rate of rupture reported by Tittle et al (2002) could have be a result of patient selection factors: all cases were symptomatic with involvement of the ascending aorta in 46% of cases, likely representing the most severe subset of patients.

Management of acute aortic syndromes

Acute dissection, intramural haematoma and penetrating atherosclerotic ulcer may be subdivided by duration: acute (<14 days), subacute (15–90 days), and chronic (>90 days). In all three conditions the goal is to reduce the risk of rupture by decreasing aortic

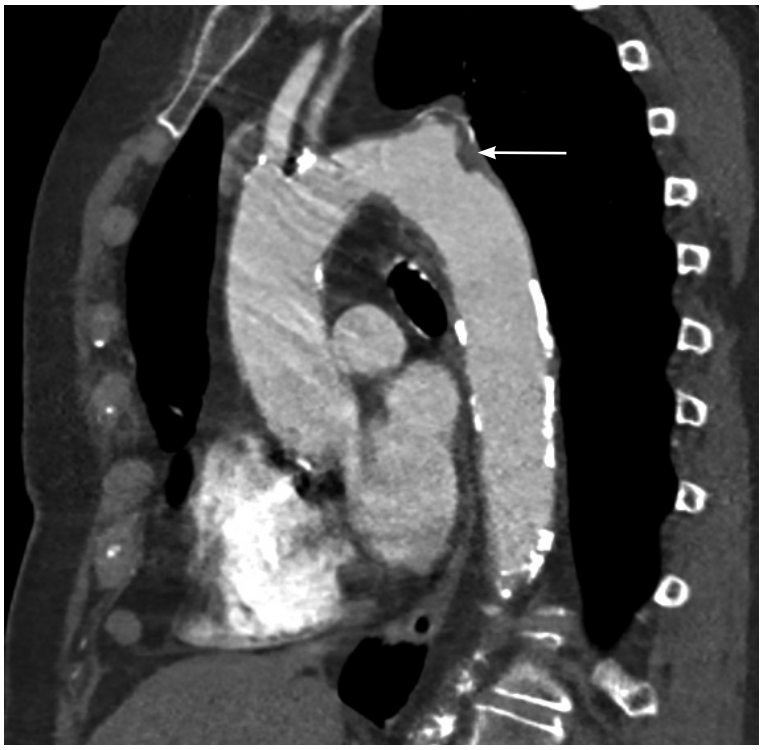

Figure 8. Sagittal oblique section of a computed tomography angiogram in an elderly patient with diffuse aortic atheroma and a focal penetrating atherosclerotic ulcer, resulting in a small focal saccular aneurysm.

wall stress. This is achieved through aggressive control of hypertension (systolic blood pressure <120 mmHg), heart rate (<70 bpm), and pain, first at presentation and then lifelong. In general, clinical features denoting complication include uncontrollable pain, refractory hypertension or hypotension, and end-organ malperfusion. Concerning imaging features denoting complication are associated with a high risk of progression to aneurysm, pseudoaneurysm or dissection, and therefore a high risk of rupture (Table 1). Direct imaging complications may also be demonstrated, such as direct coronary artery or visceral branch vessel occlusion. Therefore a 'complicated' aortic dissection, intramural haematoma or penetrating atherosclerotic ulcer is denoted by the presence of clinical and/or imaging complications. The severity of complications guides the urgency of open surgical or endovascular repair or conservative best medical therapy.

Management of acute aortic dissection

Type A acute aortic dissection

There has been longstanding European and North American consensus on immediate operative management for patients with type A acute dissection (Hiratzka et al, 2010; Erbel et al, 2014).

Type B acute aortic dissection

Current European recommendations are that patients with complicated type B acute dissection (Table 1) be offered thoracic endovascular aortic repair, especially given the favourable risk of mortality compared to open surgery, short- and medium-term outcomes, and complication rates (Moulakakis et al, 2014; Evangelista et al, 2018). Appropriate vascular access and suitable anatomy (in particular adequate landing zones) are a prerequisite.

In patients with uncomplicated type B acute dissection, best medical therapy and imaging follow up has long been considered a safe strategy. Despite this, however,

Table 1. Computed tomography features associated with progression in patients with type B acute dissection, intramural haematoma or penetrating atherosclerotic ulcer

Condition	Computed tomography features		References
Acute dissection	Aortic diameter >40–45 mm at presentation		Schwartz et al (2018)
	Aortic growth >5 mm in 6 months		
	Entry tear >10 mm at presentation		
	False lumen diameter >20–22 mm		
	Patent or only partially thrombosed false lumen		
Intramural haematoma	Initial aortic diameter >40–50 mm at presentation	>40 mm	Sueyoshi et al (1997); Kitai et al (2010)
		≥50 mm	Evangelista et al (2004)
	Haematoma thickness >10 mm		Sueyoshi et al (1997); Choi et al (2001)
	Ulcer-like projection		Ganaha et al (2002); Sueyoshi et al (2002); Kaji et al (2003); Kitai et al (2010); Schlatter et al (2011)
	Progressive pleural effusion		Choi et al (2001); Evangelista et al (2004)
Penetrating atherosclerotic ulcer	Symptomatic		Stanson et al (1986); Nathan et al (2012)
	Presence of intramural haematoma		Ganaha et al (2002)
	Progressive pleural effusion		Ganaha et al (2002)
	Penetrating atherosclerotic ulcer depth >10–15 mm, diameter >20 mm or high growth rate*		Ganaha et al (2002); Jánosi et al (2016)
	*Debated, not shown in		Nathan et al (2012)

From Pape et al (2015)

the long-term risk of progression to complicated dissection or aneurysm is reported at 30–40%, so the potential utility of thoracic endovascular aortic repair in these patients remains a subject of debate (Schwartz et al, 2018).

No mortality benefit of thoracic endovascular aortic repair has been shown in patients with uncomplicated type B acute dissection (Enezate et al, 2018) and there is a risk of retrograde type A dissection, which may occur in up to 2.5% of patients with up to 37% mortality (Chen et al, 2017). Emerging evidence on the use of thoracic endovascular aortic repair in the sub-acute phase suggests morphological and aorta-specific mortality benefit in the medium term, compared to best medical therapy (Nienaber et al, 2013; Hossack et al, 2020). However, the exact mechanisms (Nienaber et al, 2013) and most relevant sub-groups currently remain unknown (Schwartz et al, 2018; Tadros et al, 2019). Thoracic endovascular aortic repair itself carries operative risks, such as stroke in up to 6.25% of patients (vs 5.5% best medical therapy) (Hossack et al, 2020).

Management of intramural haematoma

Type A intramural haematoma

European and North American guidelines (Hiratzka et al, 2010; Erbel et al, 2014) recommend immediate open surgery for patients with type A intramural haematoma, because of the high rate of progression to dissection (16%) and mortality similar to acute dissection at 27% (Evangelista et al, 2018).

Type B intramural haematoma

The European recommendation is that patients with uncomplicated type B intramural haematoma receive medical management and those with complicated intramural haematoma type B be offered thoracic endovascular aortic repair (Erbel et al, 2014). There remains broad agreement on the concerning imaging features associated with progression (Table 1) (Li et al, 2018; Chakos et al, 2019). The role of thoracic endovascular aortic repair in patients with uncomplicated type B intramural haematoma remains unclear.

Management of penetrating atherosclerotic ulcer

The presence of symptoms (Table 1) is key in the management of penetrating atherosclerotic ulcer, with most cases presenting asymptomatically (82%), located in the descending aorta (93%), and uncovered incidentally (Nathan et al, 2012). Asymptomatic penetrating atherosclerotic ulcers are less likely to progress than symptomatic penetrating atherosclerotic ulcers (20% vs 45%). Symptomatic penetrating atherosclerotic ulcers are more likely to develop intramural haematoma (80%) or saccular aneurysm (13%), with rupture rates of up to 38%. As this exceeds the rate for acute dissection, thoracic endovascular aortic repair is recommended (Stanson et al, 1986; Coady et al, 1998; Tittle et al, 2002) and now well established as first-line therapy, with complication rates comparable to thoracic endovascular aortic repair for other indications (Oderich et al, 2019).

Penetrating atherosclerotic ulcer size (diameter and depth) has been shown by some authors to be relevant to progression (Ganaha et al, 2002; Jánosi et al, 2016), and can therefore be an indicator for thoracic endovascular aortic repair. However, almost all patients in these studies presented with symptoms, and size and/or depth was not shown to be a significant predictor of progression in the largest series of penetrating atherosclerotic ulcers so far (Nathan et al, 2012). As there is a paucity of randomised data, most groups essentially advocate watchful waiting and hypertensive control for patients with type B penetrating atherosclerotic ulcers, unless they are symptomatic or associated with complications (Oderich et al, 2019).

Management summary

Open surgery is usually indicated for patients with type A acute dissection, intramural haematoma or penetrating atherosclerotic ulcer because of the high risk of tamponade, aortic rupture, or severe valvular insufficiency, regardless of the presence of complications (Hiratzka et al, 2010; Erbel et al, 2014). Patients with complicated type B acute dissection, intramural haematoma or penetrating atherosclerotic ulcer should be offered thoracic endovascular aortic repair, providing that the anatomy is suitable (Hiratzka et al, 2010; Erbel et al, 2014). In addition to uncontrollable pain, refractory hypertension or hypotension and end-organ

Key points

- In patients with acute aortic dissection, computed tomography can identify the entry or re-entry point of the dissection, the true and false lumens, and complications such as branch vessel involvement, end-organ ischaemia, aortic rupture, or aortic root involvement.
- On unenhanced computed tomography intramural haematoma is characterised by crescenteric, high-attenuation, aortic wall thickening which does not enhance following administration of contrast.
- A penetrating atherosclerotic ulcer may be seen as a contrast-filled pouch extending into the media with outpouching of the outer aortic wall. This distinguishes it from an uncomplicated atheromatous ulcer, which is solely limited to the intima. Penetrating atherosclerotic ulcers may be associated with varying degrees of intramural haematoma.
- Open surgery is usually indicated for type A acute dissection, intramural haematoma, and penetrating atherosclerotic ulcer because of the high risk of tamponade, aortic rupture, or severe valvular insufficiency, regardless of the presence of complication.
- Patients with complicated type B acute dissection, intramural haematoma or penetrating atherosclerotic ulcer are usually offered thoracic endovascular aortic repair, providing that the anatomy is suitable.
- Patients with uncomplicated type B acute dissection, intramural haematoma or penetrating atherosclerotic ulcer are usually offered best medical therapy and imaging follow up.
- Limited evidence suggests a role for thoracic endovascular aortic repair in subacute, uncomplicated type B acute dissection pathology but this remains the subject of debate.

malperfusion, the specific computed tomography features associated with an increased risk of progression are summarised in Table 1. Those with uncomplicated type B acute dissection, intramural haematoma or penetrating atherosclerotic ulcer are offered best medical therapy and imaging follow up, with European guidelines advising computed tomography before discharge, at 1, 3, 6, and 12 months, and annually thereafter (Erbel et al, 2014). Limited evidence suggests a role for thoracic endovascular aortic repair in patients with subacute, uncomplicated type B acute dissection pathology but this remains the subject of debate.

Conclusions

This article presents some of the key imaging features and briefly summarises some of the latest evidence and guidance on the management of acute aortic syndromes. Endovascular repair is now well established in the treatment of patients with complicated type B pathology. Its utility in patients with type A dissection, intramural haematoma or uncomplicated type B acute dissection is of interest, although this remains incompletely delineated at present.

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Conflicts of interest

The authors declare no conflicts of interest.

References

Alomari IB, Hamirani YS, Madera G et al. Aortic intramural hematoma and its complications. Circulation. 2014;129(6):711–716. https://doi.org/10.1161/CIRCULATIONAHA.113.001809

Benitez RM, Gurbel PA, Chong H et al. Penetrating atherosclerotic ulcer of the aortic arch resulting in extensive and fatal dissection. Am Heart J. 1995;129(4):821–823. https://doi.org/10.1016/0002-8703(95)90335-6

Chakos A, Twindyawardhani T, Evangelista A et al. Endovascular versus medical management of type B intramural hematoma: a meta-analysis. Ann Cardiothorac Surg. 2019;8(4):447–455. https://doi.org/10.21037/acs.2019.06.11

Chen Y, Zhang S, Liu L et al. Retrograde type A aortic dissection after thoracic endovascular aortic repair: a systematic review and meta-analysis. J Am Heart Assoc. 2017;6(9):e004649. https://doi.org/10.1161/JAHA.116.004649

Chin AS, Willemink MJ, Kino A et al. Acute limited intimal tears of the thoracic aorta. J Am Coll Cardiol. 2018;71(24):2773–2785. https://doi.org/10.1016/j.jacc.2018.03.531

Choi S, Choi S-J, Kim J et al. Useful CT findings for predicting the progression of aortic intramural hematoma to overt aortic dissection. J Comput Assist Tomogr. 2001;25(2):295–299. https://doi.org/10.1097/00004728-200103000-00025

Coady MA, Rizzo JA, Hammond GL et al. Penetrating ulcer of the thoracic aorta: What is it? How do we recognize it? How do we manage it? J Vasc Surg. 1998;27(6):1006–1016. https://doi.org/10.1016/S0741-5214(98)70003-5

Enezate TH, Omran J, Al-Dadah AS et al. Thoracic endovascular repair versus medical management for acute uncomplicated type B aortic dissection. Catheter Cardiovasc Interv. 2018;91(6):1138–1143. https://doi.org/10.1002/ccd.27406

Erbel R, Aboyans V, Boileau C et al. 2014 ESC Guidelines on the diagnosis and treatment of aortic diseases: document covering acute and chronic aortic diseases of the thoracic and abdominal aorta of the adult. The Task Force for the Diagnosis and Treatment of Aortic Diseases of the European Society of Cardiology (ESC). Eur Heart J. 2014;35(41):2873–2926. https://doi.org/10.1093/eurheartj/ehu281

Evangelista A, Dominguez R, Sebastia C et al. Prognostic value of clinical and morphologic findings in short-term evolution of aortic intramural haematoma: therapeutic implications. Eur Heart J. 2004;25(1):81–87. https://doi.org/10.1016/j.ehj.2003.10.011

Evangelista A, Isselbacher EM, Bossone E et al. Insights from the international registry of acute aortic dissection: a 20-year experience of collaborative clinical research. Circulation. 2018;137(17):1846–1860. https://doi.org/10.1161/CIRCULATIONAHA.117.031264

Evangelista A, Mukherjee D, Mehta Rajendra H et al. Acute intramural hematoma of the aorta. Circulation. 2005;111(8):1063–1070. https://doi.org/10.1161/01.CIR.0000156444.26393.80

Ganaha F, Miller DC, Sugimoto K et al. Prognosis of aortic intramural hematoma with and without penetrating atherosclerotic ulcer: a clinical and radiological analysis. Circulation. 2002;106(3):342–348. https://doi.org/10.1161/01.CIR.0000022164.26075.5A

Harris KM, Braverman AC, Eagle KA et al. Acute aortic intramural hematoma. Circulation. 2012;126(11_suppl_1):S91–S96. https://doi.org/10.1161/CIRCULATIONAHA.111.084541

Hiratzka LF, Beckman JA, Bersin RM et al. 2010 ACCF/AHA/AATS/ACR/ASA/SCA/SCAI/SIR/STS/SVM guidelines for the diagnosis and management of patients with thoracic aortic disease: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines, American Association for Thoracic Surgery, American College of Radiology, American Stroke Association, Society of Cardiovascular Anesthesiologists, Society for Cardiovascular Angiography and Interventions, Society of Interventional Radiology, Society of Thoracic Surgeons, and Society for Vascular Medicine. Circulation. 2010;121(13):e266–369. https://doi.org/10.1161/CIR.0b013e3181d4739e

Hossack M, Patel S, Gambardella I et al. Endovascular vs. medical management for uncomplicated acute and sub-acute type B aortic dissection: a meta-analysis. Eur J Vasc Endovasc Surg. 2020;59(5):794–807. https://doi.org/10.1016/j.ejvs.2019.08.003

Jánosi RA, Gorla R, Tsagakis K et al. Thoracic endovascular repair of complicated penetrating aortic ulcer: an 11-year single-center experience. J Endovasc Ther. 2016;23(1):150–159. https://doi.org/10.1177/1526602815613790

Kaji S, Akasaka T, Katayama M et al. Long-term prognosis of patients with type B aortic intramural hematoma. Circulation. 2003;108(90101):II307–311. https://doi.org/10.1161/01.cir.0000087425.86049.74

Kitai T, Kaji S, Yamamuro A et al. Impact of new development of ulcer-like projection on clinical outcomes in patients with type B aortic dissection with closed and thrombosed false lumen. Circulation. 2010;122(11_suppl_1):S74–80. https://doi.org/10.1161/CIRCULATIONAHA.109.927517

LePage MA, Quint LE, Sonnad SS et al. Aortic dissection. Am J Roentgenol. 2001;177(1):207–211. https://doi.org/10.2214/ajr.177.1.1770207

Li L, Jiao Y, Zou J et al. Thoracic endovascular aortic repair versus best medical treatment for high-risk type B intramural hematoma: a systematic review of clinical studies. Ann Vasc Surg. 2018;52:273–279. https://doi.org/10.1016/j.avsg.2018.03.010

Moulakakis KG, Mylonas SN, Dalainas I et al. Management of complicated and uncomplicated acute type B dissection. A systematic review and meta-analysis. Ann Cardiothorac Surg. 2014;3(3):234–246. https://doi.org/10.3978/j.issn.2225-319X.2014.05.08

Nathan DP, Boonn W, Lai E et al. Presentation, complications, and natural history of penetrating atherosclerotic ulcer disease. J Vasc Surg. 2012;55(1):10–15. https://doi.org/10.1016/j.jvs.2011.08.005

Nienaber CA, Kische S, Rousseau H et al. Endovascular repair of type B aortic dissection: long-term results of the randomized investigation of stent grafts in aortic dissection trial. Circ Cardiovasc Interv. 2013;6(4):407–416. https://doi.org/10.1161/CIRCINTERVENTIONS.113.000463

Oderich GS, Kärkkäinen JM, Reed NR et al. Penetrating aortic ulcer and intramural hematoma. Cardiovasc Intervent Radiol. 2019;42(3):321–334. https://doi.org/10.1007/s00270-018-2114-x

Ohnesorge B, Flohr T, Becker C et al. Cardiac imaging by means of electrocardiographically gated multisection spiral CT: initial experience. Radiology. 2000;217(2):564–571. https://doi.org/10.1148/radiology.217.2.r00nv30564

Pape LA, Awais M, Woznicki EM et al. Presentation, diagnosis, and outcomes of acute aortic dissection: 17-year trends from the international registry of acute aortic dissection. J Am Coll Cardiol. 2015;66(4):350–358. https://doi.org/10.1016/j.jacc.2015.05.029

Roos JE, Willmann JK, Weishaupt D et al. Thoracic aorta: motion artifact reduction with retrospective and prospective electrocardiography-assisted multi–detector row CT. Radiology. 2002;222(1):271–277. https://doi.org/10.1148/radiol.2221010481

Schlatter T, Auriol J, Marcheix B et al. Type B intramural hematoma of the aorta: evolution and prognostic value of intimal erosion. J Vasc Interv Radiol. 2011;22(4):533–541. https://doi.org/10.1016/j.jvir.2010.10.028

Schwartz SI, Durham C, Clouse WD et al. Predictors of late aortic intervention in patients with medically treated type B aortic dissection. J Vasc Surg. 2018;67(1):78–84. https://doi.org/10.1016/j.jvs.2017.05.128

Sebastià C, Pallisa E, Quiroga S et al. Aortic dissection: diagnosis and follow-up with helical CT. Radiographics. 1999;19(1):45–60. https://doi.org/10.1148/radiographics.19.1.g99ja0945

Song J-K, Kim H-S, Kang D-H et al. Different clinical features of aortic intramural hematoma versus dissection involving the ascending aorta. J Am Coll Cardiol. 2001;37(6):1604–1610. https://doi.org/10.1016/S0735-1097(01)01184-6

Stanson AW, Kazmier FJ, Hollier LH et al. Penetrating atherosclerotic ulcers of the thoracic aorta: natural history and clinicopathologic correlations. Ann Vasc Surg. 1986;1(1):15–23. https://doi.org/10.1016/S0890-5096(06)60697-3

Sueyoshi E, Matsuoka Y, Imada T et al. New development of an ulcerlike projection in aortic intramural hematoma: CT evaluation. Radiology. 2002;224(2):536–541. https://doi.org/10.1148/radiol.2242011009

Sueyoshi E, Matsuoka Y, Sakamoto I et al. Fate of intramural hematoma of the aorta: CT evaluation. J Comput Assist Tomogr. 1997;21(6):931–938. https://doi.org/10.1097/00004728-199711000-00016

Tadros RO, Tang GHL, Barnes HJ et al. Optimal treatment of uncomplicated type B aortic dissection: JACC review topic of the week. J Am Coll Cardiol. 2019;74(11):1494–1504. https://doi.org/10.1016/j.jacc.2019.07.063

Tittle SL, Lynch RJ, Cole PE et al. Midterm follow-up of penetrating ulcer and intramural hematoma of the aorta. J Thorac Cardiovasc Surg. 2002;123(6):1051–1059. https://doi.org/10.1067/mtc.2002.121681

Vilacosta I, San Román JA. Acute aortic syndrome. Heart Br Card Soc. 2001;85(4):365–368. https://doi.org/10.1136/heart.85.4.365

Vilacosta I, San Román JA, Aragoncillo P et al. Penetrating atherosclerotic aortic ulcer: documentation by transesophageal echocardiography. J Am Coll Cardiol. 1998;32(1):83–89. https://doi.org/10.1016/S0735-1097(98)00194-6

Williams DM, Joshi A, Dake MD et al. Aortic cobwebs: an anatomic marker identifying the false lumen in aortic dissection–imaging and pathologic correlation. Radiology. 1994;190(1):167–174. https://doi.org/10.1148/radiology.190.1.8259399