Acute aorta, overview of acute CT findings and endovascular treatment options

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Acute aortic pathologies include acute aortic syndrome (aortic dissection, intramural hematoma, penetrating aortic ulcer), impending rupture, aortic aneurysm rupture and aortic trauma. Acute aortic syndrome, aortic aneurysm rupture and aortic trauma are life-threatening conditions requiring prompt diagnosis and treatment. The basic imaging modality for "acute aorta" is CT angiography with typical findings for these aortic pathologies. Based on the CT, it is possible to classify aortic diseases and anatomical classifications are essential for the planning of treatment. Currently, endovascular treatment is the method of choice for acute diseases of the descending thoracic aorta and is increasingly indicated for patients with ruptured abdominal aortic aneurysms.

Key words: acute aortic syndrome, aortic dissection, intramural hematoma, penetrating aortic ulcer, aortic aneurysm rupture, CT angiography, endovascular treatment

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INTRODUCTION

Acute aortic syndrome (aortic dissection, intramural hematoma, penetrating aortic ulcer), impending rupture, aortic rupture and aortic trauma are life-threatening conditions requiring early diagnosis and treatment. The basic imaging modality for “acute aorta” is CT angiography. Both early diagnosis and immediate treatment are essential in these cases. Currently, endovascular treatment is the method of choice for these acute diseases of the descending thoracic aorta. Further, patients with ruptured aortic aneurysms are increasingly indicated for stentgraft implantation with encouraging results.

ACUTE AORTIC SYNDROME

Acute aortic syndrome is a life-threatening non-traumatic disease of the thoracic aorta. This syndrome includes aortic dissection, intramural hematoma (IMH) and penetrating aortic ulcer (PAU). Acute aortic syndrome usually presents with acute central chest pain that radiates to the back, but symptoms vary. Syncope can result from hypotension secondary to cardiac tamponade or from obstruction of the cerebral blood vessels. Rupture into the mediastinum can cause severe hemorrhagic pleural effusions or pericardial tamponade which may be fatal.

Early diagnosis and treatment are essential and are associated with improved clinical outcomes in acute aortic syndrome. CT angiography is the basic imaging modality1,2. CT angiography has a sensitivity and specificity approaching 100% (ref.3).

CT angiography protocol used by the authors includes:

1. Non-enhanced CT with 5 mm thin sections, interval 5 mm, 120 kV, 50 mA (manual). Non-enhanced CT is very important for the detection of hemopericardium, hemothorax, hemomediastinum, intramural hematoma, which are better seen than after contrast medium administration3. 2. CT angiography with 1.25 mm thin section, interval 1.25 mm (overlap 0.625), 120 kV, 150-800 mA (auto mA), 80 mL of non-ionic iodinated contrast medium 370-400 mg I/mL followed by 40 mL of saline, injection flow rates 3.5-4 mL/s (4 mL/s for contrast medium, 3.5 mL/s for saline). ROI (Region of interest) is placed in the descending thoracic aorta (2 ROI - in true and false lumens). Scan direction is cranial to caudal from neck to groins (chin to the head of the femoral bones).

Acute aortic dissection

Aortic dissection is caused by a tear in the intima of the aorta wall that allows blood flow into the aortic wall and creation of a new “false” lumen (separation of part of the media from outer layers). Propagation of dissection can be anterograde or retrograde and dissection can involve aortic valve and any side aortic branches and thus cause complications. Prognosis depends on the localisation of dissection. Dissection of ascending thoracic aorta is an acute threat to life due to the potential involvement of aortic valves, coronary arteries and arteries originating from aortic arch or pericardial tamponade.

Dissection arises based on degenerative changes of aortic media or it can be a progression of intramural hematoma and penetrating aortic ulcer4. The most important risk factor for acute aortic dissection is hypertension (72%) (ref.5). Other risk factors are smoking, drugs, connective tissue disease (Marfan syndrome - in 15%, especially in young patients, Ehlers-Danlos syndrome, Loeys-Dietz syndrome, Turner syndrome), hereditary
vascular disease (bicuspid aortic valve, coarctation), vasculitis (autoimmune - giant-cell arteritis, Takayasu arteritis, Behcet disease, Ormond disease, infection - syphilis, tuberculosis), trauma, iatrogenic injury (catheterisation, cardiac surgery) (ref.7,77).

Acute aortic dissection presents with acute severe pain (chest, back, abdomen) in 96% of patients with different localisation and intensity according to the type of dissection. Other symptoms with which dissection can be manifested are syncope (cerebral, vascular, neurological) (9%), ischemic stroke (6%) or acute heart failure (7%) (ref.7).

Dissection can be complicated by acute aortic valve insufficiency and acute heart failure. Hemopericardium can lead to acute cardiac tamponade. Dissection can involve aortic side branches and cause malperfusion. Malperfusion syndrome (cerebral and/or visceral malperfusion) occurs in 25-30% of dissection type A.10. Involvement of coronary arteries can lead to myocardial infarction. Neurological complications (ischemic stroke, paraplegia) are due to involvement of aortic arch arteries. Compromise of visceral arteries (superior mesenteric artery, renal arteries) can cause mesenteric ischemia (3-5%) and renal failure (5-8%) (ref.9,11). Involvement of the iliac arteries can result in acute limb ischemia.

Classification

Aortic dissection is classified according to the involved segment of aorta and the origin of primary intimal tear by Stanford classification and by DeBakey (Table 1) (ref.12,13). The Stanford classification is based on indication for treatment and divides dissection into type A (involving ascending aorta and can continue to descending aorta) and type B (involving descending aorta). Dissection type A is more frequent, 60-70% (ref.14). Untreated type A dissection is associated with high mortality, over 50% within 48 h and 80% within 14 days15.

CT findings

If we evaluate CT angiography, it is necessary to assess carefully the native scans and subsequent CT angiography.

Non-enhanced haemorrhage, including hemopericardium, hemomediasinum, hemotorax, intramural hematoma. Hemopericardium and hemotorax due to rupture of dissected aorta to these spaces are associated with poor prognosis and frequently fatal. Pericardial fluid with a density more than 20 HU (Hounsfield units) indicates hemopericardium and is highly suspicious of incipient perforation of dissected aorta into the pericardial space with high risk of acute tamponade (Table 3, Fig. 1 in supplement) (ref.7).

The following parameters are evaluated on CT angiography. It is very important to distinguish between type A and B dissections in relation to indication for treatment. Primary entry of dissection type A is on the ascending aorta, usually on the right anterior wall. The most common primary entry of type B dissection is located in the aortic isthmus, distal to the origin of the left subclavian artery. Dissection tends to have a spiral course. Re-entry for type A and B dissection is usually in the left common iliac artery. Aortic valve insufficiency can be evaluated only indirectly as dilatation of the annulus. It is necessary to evaluate a potential compromise of aortic branches, which can cause malperfusion syndrome. Occlusion of aortic branch vessels can be static or dynamic16. In a static obstruction the intimal flap enters the artery origin without re-entry and occludes the artery. Dynamic obstruction affects arteries arising from the true lumen where intimal flap prolapses and covers the artery origin. Malperfusion syndrome can be caused without damage of branches itself, due to the compression of true lumen by false lumen.

Furthermore, it is important to distinguish between true and false lumen on CT angiography (Table 3, Fig. 2 in supplement). Differentiation is possible by tracking the lumen from entry of dissection. True lumen is very often compressed, in 80% of the cases. False lumen has a larger cross-section area. If the lumen is thrombosed, it is a false lumen. Lumen with delayed enhancement and washout of contrast medium is false. Beak sign is typical for false lumen (sharp angle between the intimal flap and the wall of the aorta). If one lumen encircles the other, inside one is the true lumen. This is best seen in the aortic arch. Calcifications of the aortic wall are on the side of the true lumen. Cobweb sign in the false lumen is formed by hypodense thin strands of incompletely separated media17.

Dissection must be distinguished from pulsation artefacts and artefacts from high density contrast in central veins. Prevalence of pulsation (motion) artefacts is 57% in non-electrocardiographic-gated CT angiographic studies18. Motion artefacts are symmetric on both sides of the aorta and create a double contour of aortic wall (Table 3, Fig. 3 in supplement). Artefacts from contrast medium form thin hypodense strips (Table 3, Fig. 4 in supplement).

Treatment

Untreated type A dissection (involving the ascending aorta) is associated with poor prognosis and is strictly indicated for therapy. Surgery is still the treatment of choice19.

Uncomplicated type B dissection (without malperfusion or/and progression, without uncontrolled hypertension, uncontrolled pain) is usually managed conservatively with control of pain and blood pressure. Indication of endovascular treatment of uncomplicated dissection type B is still controversial. One existing randomised control trial compared patients with uncomplicated dissection type B treated conservatively (optimal medical treatment) and by stentgraft20. The risk of all-cause mortality (11.1% vs 19.3%), aorta-specific mortality (6.9% vs 19.3%), and progression (27.0% vs 46.1%) after 5 years was lower with endovascular treatment than with optimal medical treatment alone. The weak point of this study is that the investigators in this trial also evaluated patients with early chronic dissection.

Endovascular therapy (stentgraft implantation) is the method of choice for the treatment of complicated acute type B dissection21. Complicated dissection is dissection with persistent or recurrent pain, uncontrolled hypertension, aortic expansion, malperfusion and rupture.
Table 1. Classification of aortic dissection by DeBakey and Stanford.

<table>
<thead>
<tr>
<th>Classification of aorta</th>
<th>DeBakey</th>
<th>Stanford</th>
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<tr>
<td>Ascending aorta + descending aorta</td>
<td>I</td>
<td>A</td>
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<tr>
<td>Ascending aorta</td>
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<td>Descending aorta</td>
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<tr>
<td>Descending thoracic aorta</td>
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<td>B</td>
</tr>
<tr>
<td>Descending thoracic and abdominal aorta</td>
<td>IIIb</td>
<td>B</td>
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</table>

Table 2. Morphological criteria for standard endovascular treatment of AAA.

Morphological criteria for endovascular treatment of AAA

- Length of proximal neck ≥ 15 mm
- Diameter of neck < 32 mm
- Angulation of aorta < 60º
- Iliac arteries without excessive tortuosity or stenosis minimally on one side
- Diameter of external iliac arteries > 8 mm or 6-8 mm without calcifications

Table 3. Figures in supplement.

- Acute aortic dissection type A (hemoperitoneum, active bleeding) Fig. 1 A, B
- Acute aortic dissection type A (true x false lumen) Fig. 2 A-F
- Motion artefacts (ascending aorta) Fig. 3
- Artefacts from contrast medium Fig. 4 A, B
- Intramural hematoma type A Fig. 5 A, B
- Intramural hematoma type B, pseudoaneurysm Fig. 6 A-C
- Penetrating aortic ulcer of ascending aorta Fig. 7
- Classification of AAA (EUROSTAR/Schumacher) Fig. 8
- Ruptured thoracic aneurysm (hemothorax, active bleeding) Fig. 9 A, B
- Ruptured AAA (retroperitoneal hematoma) Fig. 10
- Ruptured AAA (active bleeding) Fig. 11
- Impending rupture, crescent sign Fig. 12
- Impending rupture, retroperitoneal fat stranding Fig. 13
- Impending rupture, bulging, fat stranding, merging wall Fig. 14 A-C
- Impending rupture, increased aneurysm size, bulging Fig. 15 A-C
- Impending rupture, prominent patent lumen Fig. 16
- Traumatic intimal tear of abdominal aorta Fig. 17
- Traumatic intimal tear of aortic isthmus Fig. 18
- Traumatic intramural hematoma of descending aorta Fig. 19
- Traumatic pseudoaneurysm of aortic isthmus Fig. 20
- Traumatic pseudoaneurysm of aortic isthmus, hemopericardium Fig. 21 A, B

The principle of endovascular therapy is closure of primary tear and exclusion of false lumen from blood circulation.

This technique of closure of primary entry by stent-graft can also resolve malperfusion syndrome (visceral, peripheral) and stops the true lumen from compression by the false lumen. If this is not sufficient, other interventions are required (stent implantation, fenestration). The type of endovascular treatment of visceral and peripheral malperfusion is based on type of arterial obstruction – dynamic or static. Dynamic occlusion can be resolved with closure of primary entry by stentgraft. If this technique is not possible, endovascular fenestration (creation of a wide communication between false and true lumen through the intimal flap with angioplasty balloon dilatation) is indicated. Static obstruction is managed with stent implantation of the affected artery.

Prospective randomised control trials for endovascular treatment of complicated acute type B dissection are not reported. Prospective multicentre registry demonstrated a 30-day mortality of 8% and stroke and spinal cord ischaemia of 8% and 2% respectively.

See case reports 1-3 (Table 4, Fig. 22-24 in supplement).

Intramural hematoma (IMH)

Intramural hematoma is caused by spontaneous bleeding from vasa vasorum in the media of the aortic wall without intimal tear. IMH represents 5-30% of acute aortic
syndromes\textsuperscript{23,24}. IMH can precede aortic rupture or aortic dissection\textsuperscript{25}.

Spontaneous intramural hematoma develops due to pre-existing atherosclerosis\textsuperscript{23}. Hypertension is a risk factor for IMH (ref.\textsuperscript{23}). Intramural hematoma is presented with chest or/and back pain in 80\% patients\textsuperscript{24}. Complications of IMH include aortic dissection in 28-47\%, aneurysm/pseudoaneurysm or rupture in 20-45\% (periaortic hematoma, hemothorax, hemomediatinum, hemopericardium), pericardial effusion and pleural effusion\textsuperscript{23}. Progression of IMH is more frequent with persisting or recurring pain or when presenting with penetrating aortic ulcer. Younger age, diameter of aorta < 4-4.5 cm and diameter of hematoma < 1 cm are associated with better prognosis\textsuperscript{26,27}.

**Classification**

Intramural hematoma is similarly classified as aortic dissection to type A (involving the ascending aorta and can continue to descending aorta) and type B (involving descending aorta). IMH type B is more frequent, 60-70\% (ref.\textsuperscript{23}).

**CT findings**

An unenhanced CT is essential for the diagnosis of IMH (ref.\textsuperscript{23}). Intramural hematoma manifests as hyperdense (60-70 HU) circular or crescent shape thickening of the aortic wall (> 5 mm) without enhancement after contrast medium administration (crescent sign) (Table 3, Fig. 5 in supplement). Intramural hematoma has a higher attenuation than intraluminal blood on unenhanced CT. CT angiography (unenhanced and enhanced scans) has 94\% sensitivity and 99\% specificity for detection of IMH (ref.\textsuperscript{23}). Pleural and/or pericardial effusion can be present\textsuperscript{24}.

It is usually easy to distinguish IMH from another aortic wall thickening (aortitis, periaortic lymphoma, periaortic fibrosis, atherosclerosis, wall thrombus) due to the presence of acute symptoms.

Patients with intramural hematoma type B should be carefully followed-up (clinical follow-up, CT angiography) due to risk of small intimal tear and blood leak into the intramural hematoma and subsequently development of pseudoaneurysm (Table 3, Fig. 6 in supplement).

**Treatment**

Intramural hematoma type A is indicated for urgent surgery, especially in patients with pericardial effusion, periaortic hematoma or aneurysm. Surgery is required in most patients with IMH type A (ref.\textsuperscript{19}). In high risk patients without aortic dilatation (< 50 mm) and wall hematoma < 11 mm only medical therapy (hypertension and pain control) can be considered as initial treatment with follow-up CT examination\textsuperscript{19,29}.

Intramural hematoma type B is managed conservatively. If treatment is indicated, endovascular therapy with stentgraft is the method of choice. Endovascular treatment of intramural hematoma type B is indicated in patients with progression and/or complications of IMH (dissection, rupture) or persistent chest pain (unresponsive to antihypertensive treatment) (ref.\textsuperscript{30}).

See case reports 4-6 (Table 4, Fig. 25-27 in supplement).

**Penetrating aortic ulcer**

Penetrating aortic ulcer (PAU) is defined as the ulceration of an atherosclerotic plaque which penetrates through the internal elastic lamina into media\textsuperscript{31}. Atherosclerotic ulcer compared with PAU is limited to intima. PAU can be multiple in 10\%. The most common localisation is descending thoracic aorta, in 94\% (ref.\textsuperscript{31}). Penetrating aortic ulcer is usually asymptomatic. If PAU is symptomatic, the main symptom is severe acute chest pain.

Classic PAU can result to localized hematoma inside media, the most common localisation being the middle or distal descending thoracic aorta. PAU may develop into a focal dissection or may penetrate through the media and create saccular pseudoaneurysm. Incidence of pseudoaneurysm is 25\% with increased risk of rupture\textsuperscript{4}.

**CT findings**

A typical CT image of ulcer is bulging of contrast medium outside of the lumen with separation of the wall calcification (Table 3, Fig. 7 in supplement).

**Table 4. Case reports in supplement.**

<table>
<thead>
<tr>
<th>Case reports</th>
<th>Acute aortic dissection</th>
<th>Intramural hematoma</th>
<th>PAU</th>
<th>Aortic rupture</th>
<th>Aortic trauma</th>
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<td>Acute aortic dissection type A</td>
<td>Case report 1</td>
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<td>Uncomplicated acute aortic dissection type B</td>
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<td>Complicated acute aortic dissection type B</td>
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<tr>
<td>Acute intramural hematoma type A</td>
<td>Case report 4</td>
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<td>Uncomplicated acute intramural hematoma type B</td>
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<tr>
<td>Complicated acute intramural hematoma type B</td>
<td>Case report 6</td>
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<td>Aortic arch pseudoaneurysm on the basis of PAU</td>
<td>Case report 7</td>
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<td>Ruptured AAA</td>
<td>Case report 8</td>
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<tr>
<td>Traumatic pseudoaneurysm of thoracic aorta</td>
<td>Case report 9</td>
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Penetrating aortic ulcer of descending thoracic aorta has a better prognosis. Indication for treatment of PUA in this localisation is similar as in the treatment of acute aortic dissection type B. Uncomplicated PUA can be treated conservatively (correction of hypertension). Treatment is indicated in case of persistent or worsening pain, rapid progression of ulcer, aortic rupture or distal embolization of thrombus and hemodynamic instability. Endovascular treatment is the method of choice.

Patients with asymptomatic penetrating aortic ulcer are indicated for treatment depending on ulcer size. Ulcer diameter > 20 mm and/or an ulcer depth > 10 mm is associated with high risk of progression of disease and is indicated for early endovascular or surgical repair.

See case report 7 (Table 4, Fig. 28 in supplement).

**AORTIC ANEURYSM RUPTURE, SYMPTOMATIC ANEURYSM, IMPENDING AORTIC ANEURYSM RUPTURE**

Aneurysm is defined as the dilation of the aorta by more than 50% of the expected diameter of the aorta. Aneurysm can have different localization and extent (thoracic, thoracoabdominal, abdominal), different etiology (degenerative, infective, inflammatory, traumatic, anastomotic, congenital) and different clinical presentation (asymptomatic, symptomatic, ruptured – free rupture, contained rupture, rupture with fistula development into adjacent organs) (ref.35).

CT angiography protocol used by the authors includes:
- Abdominal aorta: 1. Non-enhanced CT with 5 mm thin section, interval 5 mm, 120 kV, 50 mA (manual). 2. CT angiography with 0.625 mm thin section, interval 0.625 mm, 120 kV, 150-800 mA (auto mA), 70 ml of non-ionic iodinated contrast medium 320-400 mgI/mL followed by 40 ml of saline, injection flow rates 3.5-4 mL/s (4 mL/s for contrast medium, 3.5 mL/s for saline). ROI is placed in the abdominal aorta above the renal arteries. Scan direction is craniocaudal from diaphragm to the heads of the femur.
- Thoracic aorta: 1. Non-enhanced CT with 5 mm thin section, interval 5 mm, 120 kV, 50 mA (manual). 2. CT angiography with 0.625 mm thin section, interval 0.625 mm, 120 kV, 150-800 mA (auto mA), 70 mL of non-ionic iodinated contrast medium 320-400 mgI/mL followed by 40 mL of saline, injection flow rates 3.5-4 mL/s (4 mL/s for contrast medium, 3.5 mL/s for saline). ROI is placed in the abdominal aorta above the renal arteries. Scan direction is cranio-caudal from diaphragm to the chins of the femur.

**Aneurysm rupture, symptomatic aneurysm**

Thoracic aortic aneurysms are less frequent and often asymptomatic. When the aneurysm grows, symptoms may occur. Aneurysm can cause tenderness or pain in the chest, back pain, hoarseness, cough and dyspnea. Ruptured aneurysm is associated with very high mortality rate. 49% (51% arrive alive at the hospital), overall mortality is 97–100% (ref.36). Rupture can induce sudden severe sharp pain in the upper back that radiates downward, pain in chest, jaw, neck or arms and dyspnea.

Most abdominal aneurysms are asymptomatic. Aneurysm can become symptomatic, when it expands. Symptoms of abdominal aortic aneurysm include abdominal pain, chest pain, and pain in the lower back or scrotum. Symptomatic aneurysms have a high risk of rupture. Rupture is associated with very high mortality, 65–85% (ref.37). Rupture is usually signaled by a triad of symptoms which are hypotension, sudden onset of abdominal pain or pain in hip and the presence of pulsating abdominal mass. The bleeding can lead to hypovolemic shock. Rupture of anterolateral aneurysm wall into the peritoneal cavity is usually dramatic and is often associated with sudden death. Conversely, rupture of the posterolateral wall into retroperitoneal space is more favourable and patients can survive. Less common are aneurysm rupture into adjacent organs. Very rarely aneurysms may rupture into the duodenum (massive gastrointestinal bleeding) or into the inferior vena cava (swollen legs and sudden heart failure) (ref.38,39).

**Classification of aneurysms**

Anatomical classification is essential in the planning of treatment (endovascular or surgical). Aneurysms can be divided into thoracic (TAA), thoracoabdominal (TAAA) and abdominal (AAA).

Thoracic aortic aneurysms can be classified according to localisation – ascending aortic aneurysm, aneurysm of aortic arch and descending aortic aneurysm. Aneurysm of ascending aorta is the most common thoracic aortic aneurysm, but aneurysm can extend to the entire thoracic aorta.

The Crawford classification divides thoracoabdominal aortic aneurysms on the basis of the extent of aneurysm. Crawford classification of TAAA: type I – most of the descending thoracic and upper abdominal aorta, type II – most of the descending thoracic aorta and most or all of the abdominal aorta, type III – the distal descending thoracic aorta and varying segments of abdominal aorta, type IV – most or all of the abdominal aorta including the segments from which the visceral vessels arise. Modified Crawford classification of TAAA by Safi: type I – from distal to the origin of the left subclavian artery to suprarenal aorta, type II – from distal to the origin of the left subclavian artery to the aortic bifurcation, type III – from the sixth intercostal space to the aortic bifurcation, type IV – from the diaphragm to the aortic bifurcation (total abdominal aorta), type V – from the sixth intercostal space to suprarenal aorta.

Crawford classification of AAA: type I – infrarenal (subrenal), type II – juxtarenal, type III – pararenal, type IV – suprarenal (extend above the renal arteries = thoracoabdominal aneurysm type IV).

Schumacher and EUROSTAR classifications of AAA are based on aneurysm morphology according to indications for endovascular treatment.
Classification EUROSTAR/Schumacher (Table 3, Fig. 8 in supplement): type A (I) - proximal and distal neck of aneurysm exists, type B (IIa) - aneurysm extends to the bifurcation without distal neck, C (IIb) - aneurysm extends to the proximal part of common iliac artery on one or both sides, D (Iic ) - aneurysm extends to the entire common iliac artery on one or both sides, E (III) - aneurysm without proximal neck.

Advantage of endovascular treatment of RAAA is that it avoids laparotomy, thus avoiding a significant reduction in blood pressure leading to the circulation collapse upon release to the retroperitoneum. Endovascular treatment reduces cardiorespiratory load and minimizes blood loss. Endovascular therapy is indicated in hemodynamically stable patients with suitable morphology of the abdominal aorta and pelvic arteries (Table 2). However this is often associated with a delay in time-consuming preoperative CT angiography. Suitable morphology is presented in only half of these patients. A comparison of the results of surgical and endovascular treatment is very difficult.

First prospective randomized study from Nottingham comparing endovascular treatment and surgical treatment for RAAA was published in 2006 (ref.46). This study, another prospective randomized studies (AJAX, IMPROVE) and meta-analysis showed no difference in perioperative mortality. Perioperative mortality is 20-50% (ref.45,49). The results of above mentioned studies AJAX and IMPROVE have recently been questioned on methodological errors and it is stated that the results of endovascular treatment of RAAA could have been better compared with surgical repair. However, the benefits of endovascular treatment were definitely found in the reduction of the length of patients’ stay on intensive care units, the length of hospitalization, need of mechanical ventilation, blood loss and similarly in the number of re-interventions during hospitalization. But benefits of endovascular treatment of RAAA are reflected only at hospitals with high volume of elective endovascular procedures.

Ruptured juxtarenal AAA are morphologically unsuitable for immediate endovascular treatment, but not impossible. Chimney technique can be used with higher risk for proximal perigraft type Ia endoleak.

With increasing surgical specialisation and centralisation, some surgical emergencies are treated only in specialised centres. The same applies to endovascular treatment. The necessary condition for endovascular treatment is wide range of stentgrafts on shelf (range of different sizes) and an experienced team.

Ruptured ascending thoracic aneurysm and aneurysm of aortic arch are indicated for cardiac surgery. Ruptured aneurysms of descending thoracic aorta can be treated surgically or endovascularly. Endovascular treatment has equivalent mortality, complication rates and failure compared with open repair.

CT findings and treatment of symptomatic aneurysms

In symptomatic thoracic and abdominal aneurysms, the signs of rupture are looked for on a CT angiography. The signs of impending rupture should also be evaluated carefully (see below) because the presence of these signs is an indication for immediate treatment. The type of treatment is indicated according to the location of symptomatic aneurysm (see above).

See case report 8 (Table 4, Fig. 29 in supplement).

Impending aortic aneurysm rupture

A significant portion of symptomatic aneurysms and even some asymptomatic aneurysms may show signs of...
imperitect rupture. Early diagnosis and treatment of these aneurysms with signs of impending rupture is very important and can be lifesaving.

CT findings

Crescent sign is formed by acute bleeding to pre-existing thrombus or to the aortic wall. Crescent sign is associated with high risk of rupture. Sensitivity of crescent sign as an indication of complicated aneurysm is 77% and specificity is 93% (ref.57). On CT acute hematoma creates crescent shape hyperdensity inside wall thrombus. It is the best seen on non-enhanced scans (Table 3, Fig. 12 in supplement).

Increased density or stranding of retroperitoneal fat around the aneurysm indicates leak, respectively peri-aortic oedema. CT shows stripes of increased density of retroperitoneal fat surrounding abdominal aortic aneurysm or of mediastinal fat around thoracic aortic aneurysm (Table 3, Fig. 13 in supplement).

Draped aorta and blurred aneurysm wall is another sign of impending rupture. A dorsal aortic wall does not form a distinct line and merges with the spine (Table 3, Fig. 14 in supplement). The aneurysm wall cannot be thus distinguished from adjacent structures. Fat between aneurysm and spine is lost. Draped aorta is a sign of unreconised contained aneurysm rupture.

Focal bulging of aneurysm with wall discontinuity (focal disruption of wall calcifications) can represent past local bleeding (contained rupture) (ref.57) (Table 3, Fig. 15 in supplement). The same findings (aneurysm “bleb”) can be found, if the aneurysm wall is weakened (inflammation, changes of elastic fibres) (ref.58). Bulging is usually localised on the posterolateral wall. Bulging with wall discontinuity indicates risk of rupture. This sign is similar to draped aorta.

Prominent patent lumen into the aneurysm wall thrombus often in the shape of a nose is a sign of imminent rupture (Table 3, Fig. 16 in supplement).

AORTIC TRAUMA

Aortic trauma is a serious life-threatening condition. The aorta is most commonly injured in traffic accidents, usually associated with blunt trauma. The mechanism of aortic trauma is usually rapid deceleration. The most frequent localisation (90%) of trauma is isthmus (relatively mobile aortic arch can be moved against the descending aorta fixed by ligamentum arteriosum). Other localisations are peridiahfragmatic, ascending aorta, and aortic arch (10%). Trauma of the abdominal aorta is very rare and is much less common than thoracic aortic injury (Table 3, Fig. 17 in supplement). Trauma of the abdominal aorta is very rare and is much less common than thoracic aortic injury (Table 3, Fig. 17 in supplement). Eighty percent of patients with blunt thoracic aortic injury die before reaching a hospital. Mortality for patients who reach hospital within 24 h is 50% (ref.59).

CT angiography protocol used by the authors includes:

Aortic trauma is usually diagnosed in polytrauma patients in whom this CT protocol is used: 1. Non-enhanced CT – brain, cervical spine. 2. Enhanced CT of thorax and abdomen with 5 mm thin section (reconstruction 1.25, 120 kV, 150-800 mA (auto mA), 80 mL of non-ionic iodinated contrast medium 320-400 mgI/mL followed by 30 mL of saline, injection flow rates 1.8 mL/s. Scan direction is craniodaorva from cervical spine C6 to the distal margin of pelvis. In our experience this CT polytrauma protocol is sufficient for the diagnosis of aortic trauma.

Classification

Although multiple classification systems exist for evaluating the magnitude of aortic injury, the most recent and now widely accepted classification system is the Vancouver simplified blunt aortic injury grading system. This is easy to use and has low inter-observer variability and correlates with clinical outcome. According to this classification traumatic aortic injury is classified into four grades: grade I – intimal flap, thrombus, or intramural hematoma < 1 cm (Table 3, Fig. 18 in supplement), grade II – intimal flap, thrombus, or intramural hematoma > 1 cm (Table 3, Fig. 19 in supplement), and grade III – pseudoaneurysm (simple or complex, no extravasation) (Table 3, Fig. 20 in supplement), grade IV – rupture, contrast extravasation (with or without pseudoaneurysm).

Abdominal aorta zones of injury are ideally classified according to possible surgical approaches. Zone I injuries occur from diaphragmatic hiatus to the superior mesenteric artery. Zone II injuries include the superior mesenteric artery and the renal arteries. Zone III injuries extend from the inferior aspect of the renal arteries to the aortic bifurcation.

CT findings

CT has a sensitivity of 98% for diagnosis of aortic trauma and specificity (direct signs are present) is 100% (ref.63,64). Mediastinal hematoma is the most common indirect sign of injured thoracic aorta, but only 20% of mediastinal hematoma is associated with aortic trauma. If periaortic fat is seen, cause of mediastinal hematoma is most probably not aortic trauma. Thereafter it is necessary to look for another source of bleeding (intercostal arteries, internal mammary arteries, and other branches of aortic arch). Mediastinal hematoma has soft tissue density. Direct findings are mural thrombus (intraluminal filling defect), intimal flap (linear intraluminal filling defect), traumatic pseudoaneurysm (contained rupture by adventitia or surrounding tissue) and intramural hematoma (Table 3, Fig. 21 in supplement). Other signs are abnormal aortic contour and local changes of aortic diameter. Extravasation of contrast medium (active bleeding) is extremely rarely seen, because patients usually bleed to death immediately.

In relation to the consideration of endovascular treatment it is important to evaluate the diameter of thoracic aorta proximal to the injury (important in choosing the stentgraft size), length of the proximal landing zone (healthy aorta between left subclavian artery and injured aorta), patency of vertebral arteries (stentgraft implantaion sometimes requires extension of proximal landing zone across left subclavian artery – right vertebral artery
Treatment of aortic trauma

Thoracic aortic trauma is much more frequent. Patients with Grade I injury should be managed conservatively with blood pressure control using β-blockers, with a target systolic blood pressure of < 120 mm Hg and heart rate < 60 beats / min, if tolerated, coupled with antiplatelet therapy when it is not contraindicated by concomitant injury. Grade II and III injuries are indicated for invasive treatment. The basic treatment of thoracic aortic trauma for patients without contraindications is thoracic endovascular aortic repair (TEVAR) with stentgraft. Stable patients should be immediately treated and no later than within 24 h (ref.66). Endovascular treatment is minimally invasive, avoids thoracotomy and has lower morbidity and mortality compared with open surgical repair. The mortality of patients who undergo endovascular repair is 9%, whereas it is 15% in open repair and 46% in nonoperative management. In grade IV injuries patients exsanguinate immediately allowing no time for treatment. Trauma of abdominal aorta is less frequent. Zone I injuries may require extensive open exposure, while the endovascular approach is also a good option in the acute setting. Zone II injuries are not amenable to endovascular placement of covered stent without fenestration for the SMA and renal arteries. This may be possible in a stable patient provided the stentgraft and the covered stents are available. Zone III injuries are amenable to an open or an endovascular approach, although in cases of concomitant visceral injury with peritoneal spillage, the endovascular approach offers a practical alternative.

See case report 9 (Table 4, Fig. 30 in supplement).

CONCLUSION

Acute aortic syndrome (aortic dissection, intramural hematoma, penetrating aortic ulcer), impending rupture, aortic rupture and aortic trauma are life-threatening aortic conditions requiring early diagnosis and treatment. The basic imaging modality for this “acute aorta” is CT angiography. The acute aortic syndrome and ruptured aortic aneurysm can clinically mimic other pathologies. It is necessary to keep this fact in mind during evaluation of acute CT examinations done for other suspected pathologies and to carefully search for any aortic pathology. Like polytrauma patients, the recommendation is to carefully evaluate the aorta given that some traumatic changes, such as subtle intimal tear left untreated can be fatal.

IMH, intramural hematoma; PAU, penetrating aortic ulcer; ROI, region of interest; VRT, volume rendering technique; MIP, maximum intensity projection; TAA, thoracic aortic aneurysm; TAAA, thoracoabdominal aortic aneurysm; AAA, abdominal aortic aneurysm; RAAA, ruptured abdominal aortic aneurysm; RTAA, ruptured thoracic aortic aneurysm.

Search strategy and selection criteria

Data for this article were identified by searches of MEDLINE, PubMed, and references from relevant articles using the terms “acute aortic syndrome”, “aortic dissection”, “intramural hematoma”, “penetrating aortic ulcer”, “aortic aneurysm rupture”, “CT angiography”, “endovascular treatment” and combinations of these terms. We gave preference to publications from the past 10 years. Only articles published in English were included. Citations from journals with high impact factors were given special weight. Our own experience was also included.

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