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 haemorrhagic 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 modality[1,2]. 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 administration[4]. 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 ulcer[5]. The most important risk factor for acute aortic dissection is hypertension (72%) (ref.[6]). 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 diseasse, Ormond disease, infection - syphilis, tuberculosis), trauma, iatrogenic injury (catheterisation, cardiac surgery) (ref.[7,8]).

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 (cardiac, vascular, neurological) (9%), ischemic stroke (6%) or acute heart failure (7%) (ref.[9]).

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 days[15].

CT findings

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

Non-enhanced examination helps in identifying haemorrhage, including hemopericardium, hemomediastinum, hemothorax, intramural hematoma[4]. Hemopericardium and hemothorax 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.[4]).

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 dynamic[16]. 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 media[17].

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 studies[18]. 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 choice[19].

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 stentgraft[20]. 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 dissection[21]. Complicated dissection is dissection with persistent or recurrent pain, uncontrolled hypertension, aortic expansion, malperfusion and rupture. The

Table 1. Classification of aortic dissection by DeBakey and Stanford.

	DeBakey	Stanford
Ascending aorta + descending aorta	I	A
Ascending aorta	II	A
Descending aorta	III	B
Descending thoracic aorta	IIIa	B
Descending thoracic and abdominal aorta	IIIb	B

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

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 ischemia of 8% and 2% respectively[22].

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

Table 4. Case reports in supplement.

Case reports		
Acute aortic dissection	Acute aortic dissection type A	Case report 1
	Uncomplicated acute aortic dissection type B	Case report 2
	Complicated acute aortic dissection type B	Case report 3
Intramural hematoma	Acute intramural hematoma type A	Case report 4
	Uncomplicated acute intramural hematoma type B	Case report 5
	Complicated acute intramural hematoma type B	Case report 6
PAU	Aortic arch pseuodaneurysm on the basis of PAU	Case report 7
Aortic rupture	Ruptured AAA	Case report 8
Aortic trauma	Traumatic pseudoaneurysm of thoracic aorta	Case report 9

syndromes[23,24]. IMH can precede aortic rupture or aortic dissection[25].

Spontaneous intramural hematoma develops due to pre-existing atherosclerosis[23]. Hypertension is a risk factor for IMH (ref.[23]).

Intramural hematoma is presented with chest or/ and back pain in 80% patients[24]. Complications of IMH include aortic dissection in 28-47%, aneurysm/pseudoaneurysm or rupture in 20-45% (periaortic hematoma, hemothorax, hemomediastinum, hemopericardium), pericardial effusion and pleural effusion[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[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.[23]).

CT findings

An unenhanced CT is essential for the diagnosis of IMH (ref.[28]). 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.[27]). Pleural and/or pericardial effusion can be present[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.[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[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.[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[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.[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[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).

Treatment

Penetrating aortic ulcer of ascending aorta is very rare. Due to high risk of rupture surgical treatment is clearly recommended[32].

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[32].

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[33].

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[34]. 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 4 mL/s. ROI is placed in ascending thoracic aorta. Scan direction is caudocranial from diaphragm to the chin. In the case of endovascular treatment planning, CT angiography of abdominal aorta and iliac arteries can be performed to evaluate vascular access for stentgraft placement.

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[40]. 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[41].

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[42,43].

Classification EUROSTAR/Schumacher (Table 3, Fig. 8 in supplement): type A (1) – 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.

CT findings
Ruptured thoracic aortic aneurysms

The rupture of thoracic aorta aneurysm (RTAA) can be detected in CT where hemomediastinum (hyperdensity or striped opacities in the mediastinum), hemopericardium and hemothorax (Table 3, Fig. 9 in supplement) may be present. Haemorrhagic effusion has a density > 30 HU and is better seen on non-enhanced CT (ref.[4]). Haemorrhage from ascending aorta may enter the perivascular connective tissue of the pulmonary artery and then extend into the interstitium of the lungs[4].

Ruptured abdominal aortic aneurysms

In ruptured abdominal aortic aneurysm (RAAA) retroperitoneal hematoma is seen (soft tissue density mass or stranding of retroperitoneal fat). Free intraperitoneal hematoma is detected on CT very rarely, it is indicative of a very unstable situation[4]. Retroperitoneal hematoma has different density. Acute and subacute hematoma is heterogeneously hyperdense (Table 3, Fig. 10 in supplement). Active bleeding manifests as a contrast leakage outside the aorta (Table 3, Fig. 11 in supplement).

The following parameters (morphology of aorta) are evaluated on CT angiography in relation to the evaluation of aneurysm suitability for endovascular treatment: proximal landing zone – proximal neck – the "healthy" part of the aorta between the renal arteries and the aneurysm sac (diameter, length, shape, thrombus, calcification), aneurysm sac (diameter, extent, thrombus), angle of longitudinal axis of the proximal neck – longitudinal axis of the aneurysm, distal neck – the "healthy" part of the aorta below aneurysm sac, diameter of aorta bifurcation (space for stentgraft), potential aneurysmal impairment of iliac arteries, quality of external iliac arteries (diameter, calcification, tortuosity) (vascular access for stentgraft placement), distal landing zones for stentgraft – "healthy" iliac arteries, vascular anomaly (accessory renal arteries, retroaortic left renal vein).

From a technical point of view the most important parameters influencing the possibility of standard endovascular treatment are the length of the proximal neck, angulation of the subrenal aorta and quality of iliac arteries (tortuosity, calcification, stenosis).

Treatment of ruptured aneurysms

Basic treatment for ruptured AAA is surgery with resuscitation of hypovolemic shock. Ruptured part of aorta is reconstructed with a prosthetic graft. Patients with ruptured aneurysms have recently been increasingly indicated for endovascular treatment by stentgraft implantation with encouraging results.

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[44]. 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.[45]). This study, another prospective randomized studies (AJAX, IMPROVE) and meta-analysis showed no difference in perioperative mortality[45-49]. Perioperative mortality is 20-50% (ref.[48,49]). 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[48,50,51].

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[52,53].

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[54].

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

impending 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[55]. Sensitivity of crescent sign as an indication of complicated aneurysm is 77% and specificity is 93% (ref.[55]). 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[56]. A dorsal aneurysm 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 unrecognised contained aneurysm rupture[56].

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[57]. Bulging with wall discontinuity indicates risk of rupture[57,58]. 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 peridiaphragmatic, 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). 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 cranicaudal 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[60-62]. 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[62]. Zone 1 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[65]. 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 implantation sometimes requires extension of proximal landing zone across left subclavian artery – right vertebral artery

must be patent and must be sufficient for vertebrobasilar circulation).

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[62]. 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[66]. 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[67]. The mortality of patients who undergo endovascular repair is 9%, whereas it is 19% in open repair and 46% in nonoperative management[67]. In grade IV injuries patients exsanguinate immediately allowing no time for treatment. Trauma of abdominal aorta is less frequent. Zone 1 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[62,68].

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.

ABBREVIATIONS

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.

Conflict of interest statement: The authors state that there are no conflicts of interest regarding the publication of this article.

REFERENCES

1. Erbel R, Aboyans V, Boileau C, Bossone E, Bartolomeo RD, Eggebrecht H, Evangelista A, Falk V, Frank H, Gaemperli O, Grabenwöger M, Haverich A, Iung B, Manolis AJ, Meijboom F, Nienaber CA, Roffi M, Rousseau H, Sechtem U, Sirnes PA, Allmen RS, Vrints CJ. 2014 ESC Guidelines on the diagnosis and treatment of aortic diseases. Eur Heart J 2014;35(41):2873-926.
2. Hiratzka LF, Bakris GL, Beckman JA, Bersin RM, Carr VF, Casey DE Jr, Eagle KA, Hermann LK, Isselbacher EM, Kazerooni EA, Kouchoukos NT, Lytle BW, Milewicz DM, Reich DL, Sen S, Shinn JA, Svensson LG, Williams DM. 2010 ACCF/AHA/AATS/ACR/ASA/SCA/SCAI/SIR/STS/SVM Guidelines for the diagnosis and management of patients with thoracic aortic disease: Executive summary. Anesth Analg 2010;111(2):279-315.
3. Abbas A, Brown IW, Peebles C, Harden SP, Shambrook JS. The role of multidetector-row CT in diagnosis, classification and management of acute aortic syndrome. Br J Radiol 2014 Aug 1. [Epub ahead of print] doi:10.1259/bjr.20140354
4. Prokop M, Engelke C. Vascular system. Aorta. In: Prokop M, Galanski M, editors. Spiral and multislice Computed Tomography of Stuttgart, New York: Thieme; 2001. p.870-891.
5. Larson EW, Edwards WD. Risk factors for aortic dissection: a necropsy study of 161 cases. Am J Cardiol 1984;53(6):849-55.
6. Baguet JP, Chavanon O, Sessa C, Thony F, Lantelme P, Barone-Rochette G, Mallion JM. European Society of Hypertension scientific newsletter: hypertension and aortic diseases. J Hypertens 2012;30(2):440-3.
7. Cregler LL. Aortic dissection and cocaine use. Am Hearth J 1992;124(6):1665.
8. Nienaber CA, Clough RE. Management of acute aortic dissection. Lancet 2015 Feb 6. [Epub ahead of print] doi: 10.1016/S0140-6736(14) 61005-9
9. Sheik AS, Ali K, Mazhar S. Acute aortic syndrome. Circulation 2013;128(10):1122-7.
10. Geirsson A, Szeto WY, Pochettino A, McGarvey ML, Keane MG, Woo YJ, Augoustides JG, Bavaria JE. Significance of malperfusion syndromes prior to contemporary surgical repair for acute type A dissection: outcomes and need for additional revascularization. Eur J Cardiothorac Surg 2007;32(2):255-62.
11. Lauterbach SR, Cambria RP, Brewster DC, Gertler JP, Lamuraglia GM, Isselbacher EM, Hilgenberg AD, Moncure AC. Contemporary management of aortic branch compromise resulting from acute aortic dissection. J Vasc Surg 2001;33(6):1185-92.

12. DeBekey ME, Henly WS, Cooley DA, Morris GC Jr, Crawford ES, Beall AC Jr. Surgical management of dissection aneurysms of the aorta. J Thorac Cardiovasc Surg 1965;49:130-49.

13. Daily PO, Trueblood HW, Stinson EB, Wuerflein RD, Shumway NE. Management of acute aortic dissections. Ann Thorac Surg 1970;10(3):237-347.

14. Dohnert W. Cardiovascular disorders: aortic dissection. In: Radiology review manual. 5th ed. Philadelphia, Pa: Lippincott Williams & Wilkins; 2005.p. 607-9.

15. Karmy-Jones R, Aldea G, Boyle EM Jr. The continuing evolution in the management of thoracic aortic dissection. Chest 2000;117(5):1221-3.

16. Castañer E, Andreu M, Gallardo X, Mata JM, Cabezuelo MA, Pallardó Y. CT in nontraumatic acute thoracic aortic disease: typical and atypical features and complications. RadioGraphics 2003;23(Spec No):S93-S110.

17. Williams DM, Joshi A, Dake MD, Deeb GM, Miller DC, Abrams GD. Aortic cobwebs: an anatomic marker identifying the false lumen in aortic dissection—imaging and pathologic correlation. Radiology 1994;190(1):167-74.

18. Ko SF, Hsieh MJ, Chen MC, Ng SH, Fang FM, Huang CC, Wan YL, Lee TY. Effects of heart rate on motion artefacts of the aorta on non–ECG-assisted 0.5-sec thoracic MDCT. AJR Am J Roentgenol 2005;184(4):1225-30.

19. Erbel R, Aboyans V, Boileau C, Bossone E, Bossone E, Bartolomeo RD, Eggebrecht H, Evangelista A, Falk V, Frank H, Gaemperli O, Grabenwöger M, Haverich A, Iung B, Manolis AJ, Meijboom F, Nienaber CA, Roffi M, Rousseau H, Sechtem U, Sirnes PA, Allmen RS, Vrints CJ; ESC Committee for Practice Guidelines. 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):2873-926.

20. Nienaber CA, Kische S, Rousseau H, Eggebrecht H, Rehders TC, Kundt G, Glass A, Scheinert D, Czerny M, Kleinfeldt T, Zipfel B, Labrousse L, Fattori R, Ince H; INSTEAD-XL trial. 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):407-16.

21. Grabenwoger M, Alfonso F, Bachet J, Bonser R, Czerny M, Eggebrecht H, Evangelista A, Fattori R, Jakob H, Lönn L, Nienaber CA, Rocchi G, Rousseau H, Thompson M, Weigang E, Erbel R. Thoracic Endovascular Aortic Repair (TEVAR) for the treatment of aortic diseases: a position statement from the European Association for Cardio-Thoracic Surgery (EACTS) and the European Society of Cardiology (ESC), in collaboration with the European Association of Percutaneous Cardiovascular Interventions (EAPCI). Eur Heart J 2012;33(13):1558-63.

22. Heijmen RH, Thompson MM, Fattori R, Goktay Y, Teebken OE, Orend KH. Valiant thoracic stent-graft deployed with the new captivia delivery system: procedural and 30-day results of the Valiant Captivia registry. J Endovasc Ther 2012;19(9):213-25.

23. Evangelista A, Mukherjee D, Mehta RH, O'Gara PT, Fattori R, Cooper JV, Smith DE, Oh JK, Hutchison S, Sechtem U, Isselbacher EM, Nienaber CA, Pape LA, Eagle KA; International Registry of Aortic Dissection (IRAD) Investigators. Acute intramural hematoma of the aorta: a mystery in evolution. Circulation 2005;111(8):1063-70.

24. Maraj R, Rerkpattanapipat P, Jacobs LE, Makornwattana P, Kotler MN. Meta-analysis of 143 reported cases of aortic intramural hematoma Am J Cardiol 2000;86(6):664-8.

25. Macura KJ, Corl FM, Fishman EK, Bluemke DA. Pathogenesis in acute aortic syndromes: aortic dissection, intramural hematoma, and penetrating atherosclerotic aortic ulcer. AJR Am J Roentgenol 2003;181(2):309-16.

26. Sundt TM. Intramural hematoma and penetrating aortic ulcer. Curr Opin Cardiol 2007;22(6):504-9.

27. Mohr-Kahaly S, Erbel R, Kearney P, Puth M, Meyer J. Aortic intramural hemorrhage visualized by transesophageal echocardiography: findings and prognostic implications. J Am Coll Cardiol 1994;23(3):658-64.

28. Lemos AA, Pezzullo JC, Gullo M, Giannitto C, Lo Gullo R, Biondetti PR. Can the unenhanced phase be eliminated from dual-phase CT angiography for chest pain? Implications for diagnostic accuracy in acute aortic intramural hematoma. AJR Am J Roentgenol 2014;203(6):1171-80.

29. Kitai T, Kaji S, Yamamuro A, Tani T, Tamita K, Kinoshita M, Ehara N, Kobori A, Nasu M, Okada Y, Furukawa Y. Clinical outcomes of medical therapy and timely operation in initially diagnosed type A aortic intramural hematoma: a 20-year experience. Circulation 2009;120(11 Supp):S292-S298.

30. Svensson LG, Kouchoukos NT, Miller DC, Bavaria JE, Coselli JS, Curi MA, Eggebrecht H, Elefteriades JA, Erbel R, Gleason TG, Lytle BW, Mitchell RS, Nienaber CA, Roselli EE, Safi HJ, Shemin RJ, Sicard GA, Sundt TM 3rd, Szeto WY, Wheatley GH 3rd; Society of Thoracic Surgeons Endovascular Surgery Task Force. Expert consensus document on the treatment of descending thoracic aortic disease using endovascular stentgrafts. Ann Thorac Surg 2008;85(1 Supp):S1-S41.

31. Stanson AW, Kazmier FJ, Hollier LH, Edwards WD, Pairolero PC, Sheedy PF, Joyce JW, Johnson MC. Penetrating atherosclerotic ulcers of the thoracic aorta: natural history and clinicopathologic correlations. Ann Vasc Surg 1986;1(1):15-23.

32. Baikoussis NG, Apostolakis EE. Penetrating atherosclerotic ulcer of the thoracic aorta: diagnosis and treatment Hellenic J Cardiol 2010;51(2):153-7.

33. Ganaha F, Miller DC, Sugimoto K, Do YS, Minamiguchi H, Saito H, Mitchell RS, Dake MD. Prognosis of aortic intramural hematoma with and without penetrating atherosclerotic ulcer: a clinical and radiological analysis. Circulation 2002;106(3):342-8.

34. Johnston KW, Rutherford RB, Tilson MD, Shah DM, Hollier L, Stanley JC. Suggested standards for reporting on arterial aneurysms. J Vasc Surg 1991;13(3):452-8.

35. Chaikof EL, Blankensteijn JD, Harris PL, White GH, Zarins CK, Bernhard VM, Matsumura JS, May J, Veith FJ, Fillinger MF, Rutherford RB, Kent KC; Ad Hoc Committee for Standardized Reporting Practices in Vascular Surgery of The Society for Vascular Surgery/American Association for Vascular Surgery. Reporting standards for endovascular aortic aneurysm repair. J Vasc Surg 2002;35(5):1048-60.

36. Johansson G, Markström U, Swedenborg J. Ruptured thoracic aortic aneurysms: a study of incidence and mortality rates. J Vasc Surg 1995;21(6):985-8.

37. Kniemeyer HW, Kessler T, Reber PU, Ris HB, Hakki H, Widmer MK. Treatment of ruptured abdominal aortic aneurysm, a permanent challenge or a waste of resources? Prediction of outcome using a multi-organ-dysfunction score. Eur J Vasc Endovasc Surg 2000;19(2):190-6.

38. Davis PM, Gloviczki P, Cherry KJ jr, Toomey BJ, Stanson AW, Bower TC, Hallett JW jr. Aorto-caval and ilio-iliac arteriovenous fistulae. Am J Surg 1998;176(2):115-8.

39. Parry DJ, Waterworth, Kessel D, Robertson I, Berridge DC, Scott DJ. Endovascular repair of inflammatory abdominal aortic aneurysm complicated by aortoduodenal fistulation with an unusual presentation. J Vasc Surg 2001;33(4):874-9.

40. Crawford ES, Crawford JL, Safi HJ, Coselli JS, Hess KR, Brooks B, Norton HJ, Glaeser DH. Thoracoabdominal aortic aneurysms: preoperative and intraoperative factors determining immediate and longterm results of operations in 605 patients. J Vasc Surg 1986;3(3):389-404.

41. Safi HJ, Huynh TTT, Estera AL, Miller CC III, Porat EE. Thoracoabdominal aortic aneurysm. In: Franco KL, Verrier ED, editors. Advanced Therapy in Cardiac Surgery. BC Decker Inc.: Hamilton; 2003. p. 371-84.

42. Schumacher H, Allenberg JR, Eckstein HH. Morphological classification of abdominal aortic aneurysm in selection of patients for endovascular grafting. Br J Surg 1996;83(7):949-50.

43. Harris PL, Buth J, Miahle C, Myhre HO, Norgren L. The need for clinical trials of endovascular abdominal aortic aneurysm sten-graft repair: The EUROSTAR project. J Endovasc Surg 1997;4(1):72-7.

44. Hoornweg LL, Wisselink W, Vahl A, Balm A. The Amsterdam acute aneurysm trial: Suitability and application rate for endovascular repair of ruptured abdominal aortic aneurysm. Eur J Vasc Endovasc Surg 2007;33(6):679-83.

45. Hinchliffe RJ, Bruijstens L, MacSweeney STR, Braithwaite BD. A randomised trial of endovascular and open surgery for ruptured abdominal aortic aneurysm – results of pilot study and lessons learned for future studies. Eur J Vasc Endovasc Surg 2006;32(5):506-13.

46. Reimerink JJ, Hoornweg LL, Vahl AC, Wisselink W, van den Broek TA, Legemate DA. Endovascular repair versus open repair of ruptured abdominal aortic aneurysms: a multicenter randomized controlled trial. Ann Surg 2013;258(2):248-56.

47. Powell JT, Sweeting MJ, Thompson MM, Ashleigh R, Bell R, Gomes M, Greenhalgh RM, Grieve R, Heatley F, Hinchliffe RJ, Thompson SG, Ulug P. Endovascular or open repair strategy for ruptured abdominal aortic aneurysm: 30 day outcomes from IMPROVE randomized trial. BMJ 2014;348: f7661.

48. van Beek SC, Conijn AP, Koelemay MJ, Balm R. Endovascular aneurysm repair versus open repair for patients with a ruptured abdominal aortic aneurysm: a systemic review and meta-analysis of short-term survival. Eur J Vasc Endovasc Surg 2014;47(6):593-602.

49. Badger S, Bedenis R, Blair PH, Ellis P, Kee F, Harkin DW. Endovascular treatment for ruptured abdominal aortic aneurysm. Cochrane Database Syst Rev 2014 Jul 21. [Epub ahead of print] doi: 10.1002/14651858. CD005261.pub3

50. McPhee J, Eslami MH, Arous EJ, Messina LM, Schanzer A. Endovascular treatment of ruptured abdominal aortic aneurysms in the United States (2001-2006): a significant survival benefit over open repair is independently associated with increased institutional volume. J Vasc Surg 2009;49(4):817-26.

51. Raupach J, Dobes D, Lojik M, Chovanec V, Ferko A, Gunka I, Maly R, Vojacek J, Havel E, Lesko M, Renc O, Hoffmann P, Ryska P, Krajina A. Integration of endovascular therapy of ruptured abdominal and iliac aneurysms in the treatment algorithm: a single-center experience in a medium-volume vascular center. Vasc Endovasc Surg 2014;48(5-6):412-20.

52. Ketelsen D, Thomas C, Schmehl J, König CW, Syha R, Rittig K, Balletshofer B, Claussen CD, Brechtel K. Endovascular repair of abdominal aortic aneurysms: standards, technical options and advanced indications. Fortschr Röntgenstr 2014;186(4):337-47.

53. Wilson A, Zhou S, Bachoo P, Tambyraja AL. Systemic review of chimney and periscope grafts for endovascular aneurysm repair. Br J Surg 2013;100(12):1557-64.

54. Gopaldas RR, Dao TK, LeMaire SA, Huh J, Coselli JS. Endovascular versus open repair of ruptured descending thoracic aortic aneurysms: a nationwide risk-adjusted study of 923 patients. J Thorac Cardiovasc Surg 2011;142(5):1010-18.

55. Mehard WB, Heiken JP, Sicard GA. High-attenuating crescent in abdominal aortic aneurysm wall at CT: a sign of acute or impending rupture. Radiology 1994;192(2):359-62.

56. Halliday KE, al-Kutoubi A. Draped aorta: CT sign of contained leak of aortic aneurysms. Radiology 1996;199(1):41-3.

57. Fillinger MF, Racusin J, Baker RK, Cronenwett JL, Teutelink A, Schermerhorn ML, Zwolak RM, Powell RJ, Walsh DB, Rzucidlo EM. Anatomic characteristics of ruptured abdominal aortic aneurysm on conventional CT scans: Implications for rupture risk. J Vasc Surg 2004;39(6):1243-52.

58. Hunter GC, Leong SC, Yu GS, McIntyre KE, Bernhard VM. Aortic blebs: possible site of aneurysm rupture. J Vasc Surg 1989;10(1):923-99.

59. Teixeira PG, Inaba K, Barmparas G, Georgiou C, Toms C, Noguchi TT, Rogers C, Sathyavagiswaran L, Demetriades D. Blunt thoracic aortic injuries: an autopsy study. J Trauma 2011;70(1):197-202.

60. Starnes BW, Lundgren RS, Gunn M, Quade S, Hatsukami TS, Tran NT, et al, A new classification scheme for treating blunt aortic injury. J Vasc Surg 2012;55(1):47-54.

61. Lamarche Y, Berger FH, Nicolaou S, Bilawich AM, Louis L, Inacio JR, et al. Vancouver simplified grading system with computed tomographic angiography for blunt aortic injury. J Thorac Cardiovasc Surg 2012;144(2):347-1.

62. Shalhub S, Starnes BW, Tran NT, Hatsukami TS, Lundgren RS, Davis CW et al. Blunt abdominal aortic injury. J Vasc Surg 2012;55(5):1277-86.

63. Fabian TC, Davis KA, Gavant ML, Croce MA, Melton SM, Patton JH Jr, Haan CK, Weiman DS, Pate JW. Prospective study of blunt aortic injury: helical CT is diagnostic and antihypertensive therapy reduces rupture. Ann Surg 1998;227(5):666-76.

64. Melton SM, Kerby JD, McGiffin D, McGwin G, Smith JK, Oser RF, Cross JM, Windham ST, Moran SG, Hsia J, Rue LW 3rd. The evolution of chest computed tomography for the definitive diagnosis of blunt aortic injury: a single-center experience. J Trauma 2004;56(2):243-50.

65. Steenburg SD, Ravenel JG, Ikonomidis JS, Schönholz C, Reeves S. Acute traumatic aortic injury: imaging evaluation and management. Radiology 2008 Sep;248(3):748-62.

66. Fox N, Schwartz D, Salazar JH, Haut ER, Dahm P, Black JH, Brakenridge SC, Como JJ, Hendershot K, King DR, Maung AA, Moorman ML, Nagy K, Petrey LB, Tesoriero R, Scalea TM, Fabian TC. Evaluation and management of blunt traumatic aortic injury: a practice management guideline from the Eastern Association for the Surgery of Trauma. J Trauma Nurs 2015;22(2):99-110.

67. Lee WA, Matsumura JS, Mitchell RS, Farber MA, Greenberg RK, Azizzadeh A, Murad MH, Fairman RM. Endovascular repair of traumatic thoracic aortic injury: clinical practice guidelines of the Society for Vascular Surgery. J Vasc Surg 2011;53(1):187-92.

68. Halkos ME, Nicholas J, Kong LS, Burke JR, Milner R. Endovascular management of blunt abdominal aortic injury. Vascular 2006;14:223-6.

Supplemental Material: The online version of this article (doi: 10.5507/bp.2016.060) offers supplemental material.