CT imaging of non traumatic aortic emergencies: acute aortic syndrome and beyond

Poster No.: C-1286
Congress: ECR 2016
Type: Educational Exhibit
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Keywords: Arteries / Aorta, Emergency, CT-Angiography, CT, Diagnostic procedure, Education, Dissection, Aneurysms, Embolism / Thrombosis
DOI: 10.1594/ecr2016/C-1286

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Learning objectives

Acute onset of chest, abdominal or back pain are nonspecific symptoms and clinical manifestation of several life threatening diseases like acute myocardial infarction, acute pulmonary embolism, acute aortic injuries, ruptured viscus and many others. Imaging is key to the diagnosis of these diseases and aids for prompt and appropriate management. Non-traumatic aortic injuries are not common but life threatening.

The aim of this poster is to:

1. Review the anatomy and imaging protocols of the aorta with emphasis to CT angiogram of the aorta.

2. Discuss and illustrate the imaging features of acute aortic syndromes, which include intramural hematoma, penetrating atherosclerotic ulcer and aortic dissection, and their complications.

3. Discuss the imaging manifestations of diseases causing aortic stenosis or occlusion which also present with fatal emergency complications.

Background

Acute aortic diseases are life threatening with significantly high mortality. Imaging plays a vital role with multidetector CT scan being almost 100% sensitive and 98 to 99% specific for the diagnosis. Acute aortic syndrome encompasses the major cause of nontraumatic aortic emergency and includes aortic dissection, intramural hematoma and penetrating atherosclerotic ulcer. Aortic occlusive disease, be it secondary to thrombosis or inflammation, may also present with emergency conditions like bowel wall ischemia or perforation, infarction of the major organs like the brain, kidneys, and many others. This article will discuss and illustrate the imaging features of the acute aortic syndromes and aortic occlusive disease presenting with emergency complications.

Anatomy

The aorta is grossly divided into thoracic and abdominal segments.
**Thoracic segment** Fig. 1 on page 19

1. Aortic root Fig. 2 on page 20 - most proximal and bulbous portion comprised of the aortic valve annulus (also called basal ring or ventriculo-aortic junction), sinuses of Valsalva and sinotubular junction
2. Ascending aorta - immediately distal to the sinotubular junction up to the origin of the brachiocephalic artery
3. Aortic arch - origin of the brachiocephalic artery to the attachment of the ligamentum arteriosum
4. Descending aorta - begins at the ligamentum arteriosum to the aortic hiatus in the diaphragm

**Abdominal segment** (Fig. 3 on page 21): extends from the aortic hiatus to the aortic bifurcation into common iliac arteries. It can be further divided into the following:

1. Suprarenal segment - above the renal arteries
2. Juxtarenal segment - at the level (within 5 mm of the origin) of the renal arteries
3. Infrarenal segment - below the renal arteries
4. Inframesenteric segment - distal to the origin of the inferior mesenteric artery

**Histology**

The aortic wall consists of three layers (Fig. 4 on page 22), namely:

1. Intima - innermost layer, composed of endothelium on a basement membrane with ground substance and connective tissue
2. Media - sheets of elastic tissue, responsible for the elasticity, distensibility and tensile strength
3. Adventitia - outermost layer, consists of collagen, vasa vasorum (arteries supplying the aortic wall) and nervi vascularis (bundle of nerves responsible for pain during the acute stretching of aorta like in dissection).

**Maximum Aortic Diameter**

The diameter of aorta varies depending on the segment as well as in the age and sex of the patient. In general, the maximum diameters of the ascending aorta, descending aorta, and abdominal aorta should be 4, 3 and 2 cm, respectively.

**IMAGING OF THE AORTA**

**Multidetector computed tomography (MDCT)**

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MDCT scan study is the ideal imaging technique for assessment of the aorta with sensitivity 100% sensitive and 98% to 99% specificity. CT scanners with 16 or greater detector rows is recommended.

CT Protocol:

Non contrast and contrast studies to evaluate vascular structures from the thoracic inlet to the pelvis including the femoral and iliac arteries. Non contrast study is particularly helpful for the detection of acute intramural hematoma and identify intimal calcifications.

Technical considerations:

- 3 mm or less slice thickness with reconstruction interval of 50% or less
- Tube rotation of 1 sec or less and tube kilo voltage of 120 to 140 kVp

ECG gating is commonly used to reduce motion artifacts which can mimic dissection or luminal irregularity, especially at the areas of greatest motion like ascending aorta. Prospective and retrospective ECG gating are two forms of ECG gating being used. Prospective ECG gating, where the images are acquired at diastole of the cardiac cycle has demonstrated significant dose reduction compared to retrospective gating where the images are acquired at the entire cardiac cycle. In addition ECG gating is also helpful in concomitant evaluation of coronary arteries.

The practical recommendation would be to perform ECG gating for evaluation of ascending aorta and proximal aortic arch. The evaluation of the descending arch, descending aorta and follow up of previous type B dissection may be performed without ECG gating.

Contrast study: The entire aorta as described has to be imaged following intravenous (IV) contrast. Higher concentration of iodine 350 to 375 mg/ml is advised for better assessment. The contrast is to be administered at the rate of 3 to 5 ml/sec followed by saline bolus and total dose not to exceed 150 ml. Bolus tracking should be done and triggered at 200 HU for aorta for 64-section scanner and 230-250 HU for 128-section or higher. Images should be acquired at arterial and venous phases whereas delayed venous phase (after 60 sec) is used for evaluation of the aorta following endovascular repair, particularly to detect endoleaks.

Radiation:

Table1. Estimated radiation dose in each procedure
<table>
<thead>
<tr>
<th>Procedure (CT angiography of aorta)</th>
<th>radiation dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prospective ECG gating</td>
<td>3.65 mSv</td>
</tr>
<tr>
<td>Retrospective ECG gating</td>
<td>8.85 mSv</td>
</tr>
<tr>
<td>Non ECG gating</td>
<td>4.50 mSv</td>
</tr>
</tbody>
</table>

**Other imaging modalities:**

**Magnetic resonance imaging (MRI)**

MRI is also equally sensitive and specific to CT scan for evaluation of the aorta. MRI has advantage for it being able to better assess the aortic valve pathology and left ventricular dysfunction without radiation exposure and use of iodinated contrast. However longer acquisition time limits its role in emergency setting, in addition claustrophobic patients and the ones with metallic implants and cardiac pacemakers are also contraindications for MRI.

MRI is preferred over serial follow up with ECG gated CT angiography particularly for younger individuals with hereditary connective tissue disorders like Marfans or Ehlers danlos syndrome. Serials of follow up study and repeated exposure may lead to radiation hazards to these individuals.

**Chest x-ray (CXR)**

CXR has limited role and may be a primary screening method for identification of other pathology with similar clinical symptoms as aortic emergency. On instances widening of the mediastinum due to ruptured aneurysm may be detected on initial evaluation.

**Measurement and reporting**

External diameters are measured and should be precise, accurate and taken at reproducible landmarks in a plane perpendicular to the axis of blood flow. The accurate assessment of these diameters is critical as the size of endovascular graft will highly rely on the measurements provided by imaging studies. Studies show the measurement on diastole provides the best reproducibility. Inter and intraobserver variability ranges from 3 to 5 mm, so any measurement with differences greater than 5 mm is significant and indicates error.
On evaluation of aortic root, measurement of the annulus is essential for selection of implant size for transcatheter implantation of aortic valves. The three sinuses of valsalva are located immediately above the aortic valves and are the right and left coronary and the non coronary sinus. These sinuses taper to a sinotubular junction also known as sinotubular ridge. The suggested anatomic locations of measurements in a standard report describing the aorta are described in Table 2 and illustrated in Fig. 5 on page 23.

Table 2. Suggested anatomic locations of aortic measurements

1. Annulus
2. Sinuses of valsalva
3. Sinotubular junction
4. Midascending aorta (midpoint between sinotubular junction and proximal aortic arch)
5. Proximal aortic arch (aorta at origin of brachiocephalic trunk)
6. Midaortic arch (just distal to left common carotid artery)
7. Proximal descending aorta (2 cm distal to left subclavian artery)
8. Mid descending aorta
9. Aorta at diaphragm (2 cm above celiac axis origin)
10. Abdominal aorta at celiac axis origin
11. Abdominal aorta at most cephalic renal artery
12. Abdominal aorta at most caudal renal artery
13. Infrarenal abdominal aorta (15 mm below most caudal renal artery)
14. At the level of inferior mesenteric artery
15. Aorta just above the bifurcation
16. Aneurysm (maximum diameter at the location specified)

ACUTE AORTIC SYNDROME

Acute aortic syndrome is a spectrum of emergency life threatening conditions of the aortic disease, characterized with similar clinical characteristics like sudden onset of acute intense chest or back pain and includes aortic dissection, penetrating atherosclerotic ulcer and intramural hematoma as its components. The global death rate secondary to acute aortic disease is 2.49 to 2.78 deaths per 100,000 persons.

Aortic dissection

Aortic dissection is the most common aortic emergency and pathologically characterized by disruption of the aortic media that leads to bleeding within and along the wall of aorta and separation of the layers (Fig. 6 on page 24). The initial event is an intimal tear
that causes blood pooling to the aortic media. This blood pool further causes longitudinal separation of aortic intima and adventitia and forms the false lumen, which has pressure greater than or equal to the true lumen. Clinically characterized by sudden onset of chest or back pain. The disease can be life threatening requiring emergent management. Hypertension is the most common associated risk factor. Hereditary conditions like Marfan’s syndrome, turner’s syndrome, vascular Ehlers Danlos, Loeys-Dietz syndrome, bicuspid aortic valve and other connective tissue disorders are also the risk factors. The incidence is 2 to 3.5 cases per 100 000 persons. Nearly about forty percent (40%) of the patients die immediately.

Classifications of aortic dissection (Fig. 7 on page 25)

**Stanford classification** is more widely used and consists of two (2) types:
- *type A* - involves the ascending thoracic aorta and arch. The dissecting flap may extend to the descending thoracic or abdominal aorta and accounts for 60-70 % of the cases.
- *type B* - does not involve ascending aorta and arch. Originates at the level distal to the left subclavian artery and accounts for 30-40% of the cases.

**DeBakey system** divides dissection into three (3) types:
- *DeBakey I* - originates at the ascending aorta and involves at least the aortic arch and may extend to the descending aorta
- *DeBakey II* - involves the ascending aorta only

DeBakey types I and II are equivalent to Stanford type A
- *DeBakey III* - begins in the descending aorta usually distal to the origin of the subclavian artery and is equivalent to Stanford type B. It is further classified to type a and b.

- **IIIA** refers to dissections that originate distal to the left subclavian artery but extend proximally or distally, mostly above the diaphragm

- **IIIB** is similar dissection that originate distal to the left subclavian artery however extends below the diaphragm

**Why classify aortic dissection?**
The management of aortic dissection relies on its location. Stanford Type A is an indication for surgical management. There is almost fifty percent (50%) mortality if surgery is delayed more than 48 hrs. Stanford type B can be managed conservatively until there are no ischemic complications like visceral ischemia or organ malperfusion, signs of impending rupture or frank rupture, hemodynamically unstable and descending aorta diameter greater than 6 cm.

**Complications of aortic dissection:**

Multiple systems may be involved as complication to aortic dissection and includes the following among many others.

End-organ ischemia or malperfusion is secondary to either static and dynamic obstruction.

- *Dynamic obstruction* is direct obstruction from the prolapsed dissecting flap to the origin of the supplying branch vessel, like a curtain. This leads to increased pressure and ischemia.
- *Static obstruction* occurs where there is direct entry of the prolapsed dissecting flap to the supplying vessel without reentry point. This leads to increased pressure and thrombosis of the false lumen and eventually stenosis of the false lumen and end organ ischemia. Renal and mesenteric ischemia are the common forms or this complication.

Cardiac complications: Acute aortic regurgitation is the most common acute cardiac complication. Other complications include aortic rupture myocardial ischemia, congestive heart failure, pericardial effusion or cardiac tamponade. Death is mainly due to aortic regurgitation, cardiac tamponade or aortic rupture.

Pulmonary complications: rare and includes compression of pulmonary artery, aortopulmonary fistula, left sided pleural effusion, usually sympathetic effusion.

CNS complications: syncope (9 - 20%) and may be secondary to cardiac tamponade or obstruction to cerebral vessels

**Imaging features (Fig. 11 on page 27 & Fig. 12 on page 27)**

Multidetector CT scan is superior to all other imaging modalities almost 100% sensitive and 98% specific. Transthoracic echocardiography is less sensitive and specific (59%-83% sensitivity and specificity of 63%-93%) for the diagnosis of aortic dissection.
Key CT imaging features:

Unenhanced CT:

Medial or inward displacement of the aortic calcification is noted.

Contrast-enhanced CT:

Detection of intimal flap, seen in almost 70 % of cases. Likewise true and false lumens are also identified. The table below shows the differentiating features of true and false lumen.

Table 3. True lumen versus False lumen

<table>
<thead>
<tr>
<th></th>
<th>True lumen</th>
<th>False lumen</th>
</tr>
</thead>
<tbody>
<tr>
<td>Caliber</td>
<td>smaller</td>
<td>larger</td>
</tr>
<tr>
<td>Intimal calcification</td>
<td>medially displaced and lines the true lumen</td>
<td>away from the wall of false lumen</td>
</tr>
<tr>
<td>Convex margin</td>
<td>away from the true lumen</td>
<td>towards the true lumen</td>
</tr>
<tr>
<td>Attenuation</td>
<td>high/relatively hyperdense</td>
<td>slow flowing blood , comparatively low attenuating</td>
</tr>
<tr>
<td>Beak sign</td>
<td>negative</td>
<td>present</td>
</tr>
<tr>
<td>Cobweb sign</td>
<td>negative</td>
<td>present</td>
</tr>
</tbody>
</table>

*Beak sign* is a wedge of hematoma that creates a space for propagation of false lumen.

*Cob web sign* are linear low attenuation changes, reflective of incompletely dissected flaps.

Rarely *Mercedes Benz sign* three channel dissection where secondary dissection occurs in one of the lumen.

What needs to be mentioned on the CT report

- Length and diameter of the aorta, dimensions of the true and false lumen
- Involvement of the major organs
- Distance of the intimal tear to the major vascular branches
Mimickers of the dissection flap:

- Pericardial recess
- Mural thrombus in a fusiform aneurysm
- Periaortic fibrosis
- Anemia with apparent high attenuation of the aortic wall

**Intramural hematoma**

Intramural hematoma (IMH) is a crescentic thickening of the aortic wall that appears hyperdense compared to lumen on non contrast study (thickness greater than 5-7 mm and CT Hounsfield units of 60-70). Hematoma develops within media of the aortic wall and demonstrates no enhancement following contrast injection.

IMH accounts for ten to twenty five percent (10-25 %) of the acute aortic syndrome cases. Descending thoracic aorta is the most common location seen in approximately seventy percent (70 %) of the cases followed by ascending aorta (30%) and aortic arch (10%).

**Pathogenesis (Fig. 8 on page 25)**

The old literature describes hemorrhage within the vasa vasorum of the medial layer as the cause for development of IMH, however recent studies show there are microscopic intimal tear leading to IMH formation.

**Imaging features (Fig. 13 on page 28 )**

Multidetector CT scan is highly, almost 96% sensitive in detection of intramural hematoma.

Noncontrast CT scan demonstrates hyper dense crescent of blood surrounding the aortic lumen.

Contrast CT: Lack of contrast enhancement of the affected aortic wall.

IMH may mimic thrombosed false lumen of aortic dissection where there is inward displacement of the intimal calcification on both of these conditions; however the calcifications are semilunar or circumferential following the configuration of aortic wall in intramural hematoma. The wall of the thrombosed aortic dissection lumen spirals longitudinally.
MRI: helpful in differentiation of intramural hematoma, thrombus or thrombosed dissection where CT diagnosis is not definite and characterizing the age of hematoma.

*Digital subtraction angiography (DSA): not useful and less sensitive (83%)*

Stanford classification similar to aortic dissection is used for intramural hematoma based on its location

*Management again depends on the imaging features*

Type A IMH: Emergency surgery indicated in pericardial effusion, periaortic hematoma or large aneurysms. However for elderly patients with significant co morbidities wait and watch strategy may be done for IMH with thickness < 11 mm and aortic diameter less than 50 mm

Type B IMH: treatment similar to type B aortic dissection

*Prognosis of acute intramural hematoma:*

  - Spontaneous resolution - 10%
  - Progression to aortic dissection - 28 to 47%
  - Aortic rupture - 20 to 45%
  - Development of ulcer like projection

*Poor prognostic indicators*

  - Stanford type A IMH > 48-56 mm and Stanford Type B > 41 mm
  - Intramural hematoma thickness > 10 mm

*Differential diagnosis:*

  - Retroperitoneal fibrosis
  - Periaortic lymphoma
  - Both of these conditions enhances on post contrast studies and demonstrate irregular walls in contradiction to intramural hematoma which has smooth walls and no contrast enhancement.

*Intramural blood pool*

Intramural blood pool (IBP) is an intramural contrast medium-filled pool with a tiny intimal orifice with or without connection with an intercostal or lumbar artery in an intramural
hematoma (IMH) Fig. 14 on page 29. IBP is morphologically distinct from an ulcer-like projection, which has a wider intimal opening to the lumen. This is more common in patients less than 70 yrs and intramural hematoma thickness of more than 10 mm. This has relatively benign course and resorption occurs in approximately 86% of cases. This should be distinguished from ulcer-like projection.

**Ulcer-like projection (ULP)**

Localized blood filled pouch protruding into the intramural hematoma (< 20 mm). Development of ULP, not detected on initial CT scan suggests it being secondary to new intimal disruption and carries poor prognosis as compared to penetrating atherosclerotic ulcer (PAU), frequently progressing to aortic rupture, dissection or aneurysm.

**Imaging features**

ULP shows the same degree of contrast enhancement as the aortic lumen on post contrast CT images.

Differentiating feature of ULP from IBP:

- ULP has wider orifice (> 3 mm) than IBP.

Differentiating features of ULP from penetrating atherosclerotic ulcer:

The focal intima of ULP usually shows no atherosclerotic plaque unlike frequently noted in a penetrating atherosclerotic ulcer and it is commonly located at the ascending aorta and aortic arch unlike penetrating atherosclerotic ulcer seen commonly at descending thoracic and abdominal aorta (Fig. 15 on page 30)

**Penetrating atherosclerotic ulcer (PAU)**

PAU is ulceration of the atherosclerotic ulcer plaque penetrating through the internal elastic lamina to media and subsequent hematoma formation (Fig. 9 on page 26). This accounts for 2-7% of acute aortic syndrome and seen in elderly with atherosclerosis and conditions predisposing to it like peripheral arterial occlusive disease, coronary artery syndromes and abdominal aortic aneurysm. In addition to atherosclerosis PAU can occur in younger patients with connective tissue disorders. The most common location (> 90%) is seen at mid and descending thoracic aorta.
Imaging features

Noncontrast CT: may be similar to IMH

Contrast CT: outpouching of the aortic wall that tend to project beyond the aortic margin with irregular edges in the presence of atheromatous changes or extensive intimal calcifications. (Fig. 16 on page 31 & Fig. 17 on page 32)

Prognosis:

PAU may lead to intramural hematoma, pseudoaneurysm, aortic rupture or acute aortic dissection. IMH with PAU has progressive disease whereas PAU without IMH has stable course. PAU tends to have higher incidence of aortic rupture than aortic dissection. PAU greater than 20 mm and neck diameter greater than 10 mm have greater risk of disease progression

Indications for surgical intervention:

Recurrent and refractory pain, signs of contained rupture, rapidly growing aortic ulcer, associated periaortic hematoma or pleural effusion

Aortic aneurysm

Aneurysm is defined as a permanent localized dilatation of an artery having at least 50% increases in diameter compared to expected normal diameter of the artery in question. It is the second most frequent disease of the aorta after atherosclerosis. The overall incidence of aortic aneurysm is 10.4 cases per 100,000 person. The most common location is abdominal aorta (30.9%), followed by ascending aorta (22.1%), aortic arch (11.6%), descending thoracic aorta (7.6%) and thoracoabdominal aneurysms (2.6%).

Risk factors: Aortic aneurysms are mostly secondary to degenerative disease. Other risk factors are hypertension, alcohol, COPD, hereditary connective tissue disorders, bicuspid aortic valve among many others.

Ascending aorta or the root of aorta is most common site for thoracic aortic aneurysm, whereas infrarenal aneurysm is the most common location for abdominal aortic aneurysm and accounts for almost 90% of cases. Abdominal aortic aneurysm occurs in 28%
of patients with thoracic aortic aneurysm. Likewise abdominal aortic aneurysm is seen concomitantly in 42.1% of patients with penetrating aortic ulcers and 29.4% of patients with intramural hematoma.

Thoracic aortic aneurysms are commonly detected incidentally on chest x-ray and are mostly asymptomatic. The rare presentation may be secondary to compression to adjacent structures like trachea, esophagus or recurrent laryngeal nerve resulting to dyspnea, dysphagia or hoarsness of voice respectively.

The table below shows the normal dimension of the segments of aorta and threshold for considering aneurysm:

Table 4. Normal and abnormal aortic diameters

<table>
<thead>
<tr>
<th>Aortic segment</th>
<th>maximum normal diameter</th>
<th>non-aneurysmal dilatation</th>
<th>aortic aneurysm</th>
</tr>
</thead>
<tbody>
<tr>
<td>ascending aorta &amp; arch</td>
<td>4 cm</td>
<td>&gt;4 to &lt;5 cm</td>
<td>#5 cm</td>
</tr>
<tr>
<td>descending aorta</td>
<td>3 cm</td>
<td>&gt;3 to &lt;4 cm</td>
<td>#4 cm</td>
</tr>
<tr>
<td>abdominal aorta</td>
<td>2 cm</td>
<td>&gt;2 to &lt;3 cm</td>
<td>#3 cm</td>
</tr>
</tbody>
</table>

Morphologically aneurysms are classified as *Fusiform and Sacular aneurysms*

*Fusiform aneurysm* - symmetrical dilatation that involves the entire circumference of the aortic wall with a uniform shape. (Fig. 18 on page 33)

*Sacular aneurysm* - localized out pouching of the aortic wall and are usually pseudo aneurysm

*Annuloaortic ectasia*: dilated sinus of annulus and sinuses of valsalva with effacement of the sinotubular junction producing a pear shaped aorta that tapers to the normal aortic arch, most commonly associated with Marfans syndrome

**Significance of Imaging**
Multidecetor CT scan is primarily used for assessment of the anatomic location and size of the aneurysm, for follow up monitoring and preoperative planning for either open or endovascular repair of the aneurysm.

The followings are to be mentioned in the report for imaging of aneurysm (Fig. 10 on page 26):

- Aneurysm dimensions measured on maximal antero-posterior, transverse and craniocaudal dimensions
- Aneurysm angulation
- Proximal aortic neck length from the origin of the nearest major vessel
- Proximal aortic neck diameter - anteroposterior and craniocaudal diameter
- Proximal aortic neck angulation
- Distal aortic neck diameter

The rest of measurements should be described as mentioned on table 1.

**Indications for surgical intervention**

Normal growth rate for an aneurysm is 1-4 mm/year however it relies on the size of aneurysm. Studies show the growth rate is 2-4 mm for aneurysm smaller than 4 cm, 2-5 mm for aneurysms 4-5 cm and 3-7 mm for aneurysms greater than 5 cm in sizes.

**Indication for elective surgery**

Aneurysm greater than 5.4 cm in the greatest diameter

Growth rate more than 5-7 mm per 6 months and more than 10 mm per year

**Indication for elective surgery in special circumstances**

Thoracic aortic aneurysm greater than 5.5 cm for degenerative aneurysms and 5.0 cm for bicuspid aortic valve or aneurysm growth rate of more than 1 cm/year

4.5 cm for family history of dissection or rupture < 5.0 cm

Descending thoracic aortic aneurysm size 6.5 cm

Marfans syndrome and vascular Ehlers danlos syndrome - 4.5 to 5.0 cm or growth rate more than 5 mm/year
Loeys dietz syndrome - 40 mm or growth rate > 5mm/year

Aortic rupture:

Aortic rupture is the most fatal complication of aortic disease and may be associated with acute aortic syndrome or aortic aneurysm. The size of aneurysm is directly related to the risk of aortic rupture. Greater the size, higher the risk of rupture and overall the size greater than 6 cm has high risk of rupture. Female sex has higher risk of rupture, 2 to 4 times more than men.

Radiological features

Multidetector CT scan of the aorta is the imaging modality of choice. (Fig. 19 on page 34)

CT scan demonstrates features to distinguish complete and impending rupture as tabulated:

Table 5. Features of complete rupture and impending rupture

<table>
<thead>
<tr>
<th>Complete rupture</th>
<th>Impending rupture</th>
</tr>
</thead>
<tbody>
<tr>
<td>Focal wall discontinuity</td>
<td>Rapid enlargement of the aneurysm</td>
</tr>
<tr>
<td>Intraperitoneal or retroperitoneal hematoma</td>
<td>Hyperattenuating crescent sign</td>
</tr>
<tr>
<td>Extravasation of contrast material</td>
<td>Thrombus fissurization and periaortic stranding</td>
</tr>
<tr>
<td>Aorto-enteric or aorto-caval fistula</td>
<td>Draped aorta sign</td>
</tr>
</tbody>
</table>

Aortic rupture can extend to the mediastinum, pericardium, pleural space or to the lumen of esophagus or air way. The communication of ruptured thoracic aortic aneurysm to air way lead to formation of aortobronchial fistula and to esophagus leads to aortoesophageal fistula

Hyperattenuating crescent sign - periluminal crescentic hyperattenuation along the wall or thrombus of the aorta.
Draped aorta sign - sign of contained rupture where there is new sacular out pouching of aortic wall and draping of the posterior wall of aneurysm over the adjacent vertebral body. The wall of aorta may also be indistinguishable from the adjacent vertebral body and psoas muscle associated with loss of intervening peri aortic fat plane.

**Aortic Pseudoneurysm:**

Fewer than three layers and contained by adventitia or periadventitial tissues. These are mostly sacular aneurysm with narrow neck and mainly secondary to trauma, mycotic aneurysm or penetrating atherosclerotic ulcer.

Mycotic aneurysm is a pseudo aneurysm secondary to non-syphilitic infection of the aorta with predisposing conditions like bacterial endocarditis, drug abuse, atherosclerosis or trauma.

**Imaging features of mycotic aneurysm**

Mycotic aneurysm are usually saccular, eccentric thrombus, mostly involve ascending aorta apparently in proximity to the region of endocarditis. Periaortic thickening or stranding densities are also noted. (Fig. 20 on page 35)

**Aortic occlusive disease**

Narrowing of the aortic lumen be it secondary to intraluminal thrombus, inflammation or extra luminal compression can be life threatening. Congenital narrowing of the aorta or coarctation of aorta is typically seen at the thoracic aorta at the isthmus. Mid aortic dysplastic syndrome is narrowing of the aorta seen at the thoracoabdominal junction, whereas aorto iliac occlusive disease or Leriche’s syndrome typically occurs at below the level of renal arteries and causes complete occlusion of the aortic bifurcation. Aortic inflammatory disease, infectious or non infectious in etiology may also lead to narrowing of the aortic lumen.

Acute aortic syndrome has well been described in the literature for its emergency complications. Aortic occlusive disease may also lead to fatal emergency complications. This section of the article is aimed to focus on possible emergency complication of aortic occlusive disease and described the imaging features which has been less described unlike acute aortic syndrome. Imaging features of aorto iliac occlusive disease also
known as Leriche's syndrome and aortic inflammatory disease in particularly Takayasu Arteritis will be discussed.

**Aorto-iliac occlusive disease or Leriche's syndrome**

Leriche's syndrome is a triad of claudication, impotence and decreased femoral pulses and commonly seen in elderly men with risk factors like hypertension and diabetes. Initially described for occlusion of aortic bifurcation and iliac arteries, it may also involve major branches like renal arteries, superior and inferior mesenteric artery. Occlusion of these major vessel leads to fatal complication of ischemia or infarction of kidneys, liver, spleen and even myocardium and brain. Bowel wall ischemia secondary to mesenteric vessel occlusion is also a well known fatal complication. Chronic occlusion however may lead to collaterals formations.

**Imaging features**

Multidetector CT scan shows the occlusion of aorta and involved vessels (Fig. 21 on page 36). Infarction to liver, spleen or kidneys are demonstrated as wedge shaped areas with no enhancement on post contrast study. Mesenteric vascular occlusion can lead bowel wall ischemia demonstrated as thickened/edematous non enhancing bowel walls (Fig. 22 on page 37) also known as thumb printing sign. Prolong ischemia can lead to penumatosis intestinalis or bowel wall perforation demonstrated as penumoperitoenum on imaging.

**Inflammatory/infectious disease of the aorta**

Inflammation of the aortic wall may be infective or non infective in etiology. Syphilis was previously the common form of infectious aortitis. Non infective causes of aortitis are takayasu arteritis, rheumatic fever, ankylosing spondylitis, giant cell arteritis and relapsing polychondritis.

**Takayasu arteritis:**

Takayasu arteritis is a common form of vasculitis with multisystem involvement with predilection to the large vessels. Takayasu arteritis initially causes mural thickening and if untreated leads to transmural fibrosis, stenosis, calcifications or thrombosis. Aneurysmal dilatation of the aorta and its branches is the uncommon form and is the late stage of presentation. Four different forms of takayasu arteritis has been described depending
on the location of the vessels involved. The complications related to takayasu arteritis may be fatal similar to other occlusive disease with ischemia or infarction of major organs including brain and myocardium. Aneurysm and related complications are uncommon.

**Imaging features**

Early changes: mural thickening which appears as diffuse enhancement of the aortic walls on CT and MR

Late changes: stenosis, calcifications and aneurysm( Fig. 23 on page 38)

Knowledge of various forms of aortic occlusive disease and its complications will help in timely diagnosis and management of the possible life threatening complications.

**Post-operative imaging of aorta**

The detailed discussion regarding post operative imaging of the aorta is beyond the scope this article. Radiologist should be familiar with post operative changes with both open and endovascular repair of the aorta which at instances may be fatal requiring urgent surgical management. The most common complication seen on imaging are endoleaks for endovascular repair of aorta. Delayed images are mandatory for evaluation of endoleaks. Five types of endoleaks has been described as follows:

- Type I - endoleaks at the graft attachment site either proximally or distally
- Type II - most common type and results from persistence pressure to the aneurysmal sac by retrograde flow from the aortic collaterals, mainly the inferior mesenteric and lumbar artery.
- Type III - leak through the graft defects ( Fig. 24 on page 39 )
- Type IV - leak through graft fabric as a result of graft porosity
- Type V - endodistension or expansion of the aneurismal sac without demonstrable endoleaks

**Images for this section:**
Fig. 1: Segments of the thoracic aorta

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Fig. 2: Aortic root

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Fig. 3: Segments of the abdominal aorta

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**Fig. 4:** Layers of the aortic wall

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**Fig. 5:** Suggested anatomic locations for measuring aortic diameters: annulus (a), sinus of Valsalva (b), sinotubular junction (c), midascending aorta (d), proximal aortic arch (e), midaortic arch (f), proximal descending aorta (g), mid descending aorta (h), aorta at diaphragm (i), abdominal aorta at celiac axis origin (j), abdominal aorta at most cephalic renal artery (k), abdominal aorta at most caudal renal artery (l), infrarenal abdominal aorta (m), aneurysm (n), and aorta just above the bifurcation (o).

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**Fig. 6:** Pathogenesis of aortic dissection: Starts as a tear at the intima which allows blood to pool into the media separating the intimal layer from the rest of the aortic wall.
Fig. 7: Classifications of aortic dissection

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Fig. 8: Pathogenesis of intramural hematoma: Illustration of classic intramural hematoma resulting from rupture of the vasa vasorum supplying the aortic wall. Recent studies show that intramural hematomas may also result from microtears at the intima.

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Fig. 9: Penetrating atherosclerotic ulcer (PAU) resulting from erosion of atherosclerotic plaque

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Fig. 10: Aortic measurements prior to aneurysm repair: diameter (D1) and length (L1) of the aneurysm; diameters of proximal (D2) and distal (D3) aortic necks, proximal aortic neck length (L2), aortic diameter at the adjacent branch (e.g., renal artery) level (D4), angulation of the proximal landing zone/proximal aortic neck (#), and angulation of the aneurysm (#).

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Fig. 11: Stanford A (DeBakey I) acute aortic dissection. 3D reformatted image (a) of the aorta demonstrating aortic dissection with the arrows pointing the intimal flap extending from the aortic root down to the aortic bifurcation. Axial post contrast images (b and c) again demonstrate aortic dissection with the larger caliber false lumen with relatively low attenuation compared to the true lumen.

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Fig. 12: Stanford A, DeBakey II aortic dissection. Axial and coronal post contrast images (a, b and c) demonstrate acute aortic dissection (arrows) involving the ascending aorta and aortic arch up to the level of the brachiocephalic trunk. The arrows point to the dissection extending to right common carotid artery (image b and c).

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Fig. 13: Stanford B acute Intramural Hematoma. Axial non contrast image (a) demonstrates crescentic shaped hyper density circumferentially along the aortic wall (small arrow). Axial and sagittal post contrast study images (b and c) demonstrate no enhancement of the intramural hematoma (small and large arrows) arising distal to the left subclavian artery and extending up to the aortic hiatus.

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Fig. 14: Intramural Blood Pool (IBP). Axial non contrast image (a) demonstrates hyperdense crescent along the aortic wall indicative of acute intramural hematoma. Post contrast axial (b) and focused sagittal (c) images demonstrate no enhancement of the intramural hematoma and two small contrast outpouchings connected to the descending aorta through a narrow orifice, typical for intramural blood pooling. Partially visualised yellow line is a localiser. The blood pool arising from the intercostal artery is also demonstrated (image b).

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Fig. 15: Ulcer like projection (ULP). Axial post contrast images demonstrates intramural hematoma with irregular appearing out pouching with a wide opening connected to the descending thoracic aorta. No significant atherosclerotic calcification was noted in the vicinity of the ulcer like projection. Bilateral minimal pleural effusion was also noted.

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Fig. 16: Penetrating atherosclerotic ulcer and intramural hematoma. Axial non-contrast image (a) demonstrates relatively hyper dense aortic wall indicative of intramural hematoma with atherosclerotic intimal calcifications. Post contrast axial and coronal images (b and c) demonstrate no enhancement of the intramural hematoma and an irregular focal out pouching connected to the aorta with a wide orifice, typical for penetrating atherosclerotic ulcer. Fluid collection is noted in the mediastinum compressing the carina and bilateral bronchus. Likewise, left sided pleural effusion is also noted.

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Fig. 17: Penetrating atherosclerotic ulcer, intramural hematoma and intramural thrombus in a large thoracic aorta aneurysm. Axial non contrast image (a) demonstrates relatively hyper dense aortic wall indicative of intramural hematoma with atherosclerotic intimal calcifications. Post contrast axial and coronal images (b and c) demonstrates no enhancement of the intramural hematoma and an irregular focal out pouching connected to the aorta with a wide orifice, typical for penetrating atherosclerotic ulcer. Large thoracic aneurysm is noted at the descending thoracic aorta with significant intramural thrombus extending to the mediastinum compressing the carina and bilateral bronchus. Likewise, left-sided pleural effusion is also noted.

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Fig. 18: Infrarenal abdominal aortic aneurysm with intramural thrombus formation. Axial, coronal and sagittal images (a,b,c) demonstrate an infrarenal abdominal aortic aneurysm with significant intramural thrombus formation. Aneurysm should be described in detailed with regards to measurements and location. No contrast dissection or contrast extravasation is noted. Thoracic aortic aneurysm was also identified in the patient, not shown in the images.

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Fig. 19: Ruptured abdominal aortic aneurysm. Axial non contrast and contrast CT images (a and b) and contrast coronal image (c) demonstrate a large infrarenal aortic aneurysm with high attenuating fluid at the periaortic and retroperitoneal space with significant fat stranding suggestive of hematoma and indicative of a ruptured aneurysm. Hyperdense wall is also noted.

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Fig. 20: Mycotic aneurysm. Axial, sagittal and coronal post-contrast images (a,b,c and d) demonstrate an irregular lobulated saccular aneurysm arising from the medial aspect of the proximal abdominal aorta with surrounding fat stranding densities, suggestive of a mycotic or inflammatory aneurysm.

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**Fig. 21:** Leriche's syndrome, also known as aorto-iliac occlusive disease. MIP, coronal and axial post-contrast images (a,b and c) demonstrates severe atherosclerosis with calcification of the abdominal aorta, iliac and femoral arteries. There is complete occlusion of the infrarenal abdominal aorta and iliac arteries. Mesenteric collaterals (white open arrow in image a) demonstrates superior and inferior mesenteric arteries supplying each other through the Riolan's arcade. Likewise, the inferior intercostal arteries supply the external iliac arteries through the superficial and deep iliac circumflex arteries (red arrow in image a).

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Fig. 22: Leriche’s Syndrome with suspicious bowel ischemia. Coronal post-contrast images (a and b) of the same patient (Fig 14) demonstrate occlusion of jejunal branch of superior mesenteric artery (white arrows) with apparent bowel wall thickening of the adjacent jejunum (yellow arrow) and highly suspicious for ischemic enteritis.

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Fig. 23: Takayasu arteritis. Axial and post-contrast images of the aorta demonstrate complete occlusion of the right subclavian artery (red arrow), irregular configuration of the left renal artery (yellow arrow in image) with almost complete occlusion of the inferior segmental artery and area of non enhancement likely infarction of inferior pole cortex of the left kidney (yellow arrow in fig c). Likewise noted is a fusiform aneurysm of the descending thoracic aorta with intramural thrombus formation (white arrows in image d and e). The overall findings are indicative of various stages of Takayasu arteritis.

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**Fig. 24:** Post-endovascular repair of the ruptured aneurysm again with features of impending rupture and endoleaks. Non-contrast axial CT images (a) demonstrate hyperdense attenuation at the periphery of large infrarenal abdominal aneurysm with endovascular stents in place. Paraortic and retroperitoneal fat stranding densities are also noted. Post-contrast images demonstrate no contrast extravasation from the aneurysms, however multiple ill-defined site of endoleaks (likely Type III) are noted. Linear hyperdensities are noted within the aortic wall suggestive of thrombus fissurization, which is also indicative of impending rupture. Delayed images are mandatory for assessment for endoleaks since arterial images at instances may not detect endoleaks.

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Findings and procedure details

CT angiogram of the thoracic and abdominal aorta was done and all performed on 256 slice CT scanner machine.

The images with corresponding findings are described with their respective figure numbers.

Images for this section:

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Conclusion

Acute aortic injuries are broadly divided as traumatic and non-traumatic based on their etiology. Acute aortic syndromes, which include intramural hematoma, penetrating atherosclerotic ulcer and aortic dissection, demonstrate typical imaging manifestations, as discussed. CT angiogram is the ideal method for diagnosis, preoperative planning and follow-up monitoring. Clinical conditions leading to aortic stenosis or occlusive disease also lead to fatal complications and radiologists should be aware about the imaging features and possible complications of these uncommon clinical entities.

Personal information

References