Acute Aortic Syndrome: Beyond aortic dissection.

Poster No.: C-2204
Congress: ECR 2014
Type: Educational Exhibit
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Keywords: Arteriosclerosis, Computer Applications-3D, Arthrography, Arterial access, CT, Arteries / Aorta
DOI: 10.1594/ecr2014/C-2204

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Learning objectives

Acute aortic syndrome (AAS) describes the acute presentation of patients with one of several life threatening aortic pathologies (non-traumatic).

The purposes of this educational exhibit are:

1. Revise the concept of AAS and its pathogenesis.
2. Describe the characteristics of the different entities in the AAS and the relationship between them.
3. Set the key to a correct and early diagnosis of these entities, using the CT as a radiological technique.
4. And describe the classification, complications and treatment of each one.
Background

Introduction

Acute aortic syndrome refers to a group of pathological conditions affecting the aortic wall (non-traumatic) and are clinically indistinguishable from each other. It can affect the aorta at any location, but the involvement of the thoracic aorta, is undoubtedly the most common location and characteristics of the AAS.

Nontraumatic acute aortic syndromes, are medical emergencies typically presenting with severe acute pain and characterized by a high risk of aortic rupture and sudden death. This complex group of diseases that share relatively similar clinical presentation and imaging protocols include:

- Typical Aortic dissection,
- Intramural haematoma (atypical aortic dissection),
- Penetrating atherosclerotic ulcer

All disorders giving rise to AAS can be distinguished in terms of their a etiology and radiological appearance. There is however considerable overlap with the possibility of progression from one pathological process to another and can also occur simultaneously in the same patient.

Clinical and epidemiological characteristics

The AAS occurs mainly between the sixth and seventh decade of life, affecting men more often with a prevalence compared to women of 2:1.

Risk factors that favor the development of SAA are varied, but hypertension is the main risk factor especially for the development of classic aortic dissection and intramural hematoma (Table 1). However, atherosclerosis is the most important in the penetrating ulcer.

Table 1: Risk factors and comorbidity related to acute aortic syndrome.

<table>
<thead>
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<th>Risk Factors</th>
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Chronic hypertension  
Coronary artery disease
Collagen vascular disorders (for example, Marfans)  
Cerebrovascular disease
Previous aortic valve surgery  
Peripheral artery disease
Atherosclerosis*  
Renal disease
Pregnancy  
Diabetes
Cocaine use...

*Atherosclerosis is the leading cause of penetrating aortic ulcer, however, most dissections are not associated with atherosclerosis. In fact fibrosis and calcification may limit the progression of dissection

Clinical Manifestations

The main clinical manifestation is acute onset of pain, whose location varies depending on whether the damage is in of the thoracic or in the abdominal aorta.

As mentioned above, the most frequent location is in the thoracic aorta, so that the most common clinical presentation is going to be **chest pain**. This pain has been variously described as severe, ripping, and migratory chest pain that may radiate anteriorly to the neck or posteriorly between the scapula when it affects the ascending or descending aorta respectively. The pain may be accompanied by other signs or symptoms associated commitment aortic branches and are the complications of these entities:

- **Syncope** can result from hypotension secondary to cardiac tamponade
- **Cerebrovascular manifestations**, occurs in up to 5%-10% of patients (1). This can include involvement of one or more branch vessels.
- **Cardiac involvement** in patients with proximal dissections
- **Limb ischemia** by commitment supraaortic trunks
- **Abdominal pain** can be an indicator of abdominal branch involvement. Main abdominal arterial branch involvement has been reported in 27% of cases. Occlusion of the celiac trunk may lead to splenic or hepatic infarction with pain and abnormal liver blood test results. Involvement of the mesenteric branches can lead to mesenteric ischemia
- **Oliguria, anuria and abnormal renal blood** parameter, are indicators of renal artery involvement.
- **Compression of adjacent structures** such as the superior vena cava, left laryngeal nerve, bronchi, and esophagus.
- Extension of the dissection flap into the common femoral arteries may cause signs and symptoms of lower limb ischemia
Diagnosis

The AAS is a medical emergency, so the diagnosis must be quick and proper to begin an early and appropriate treatment, especially in the case of acute aortic dissection, because it is the most frequent cause of AAS and a real threat to life. The need to consider and highlight acute thoracic pathology as an AAS is clear.

Furthermore, thoracic aortic pathology can be a difficult diagnosis to make. Clinical findings are often absent, the chest radiograph may be normal, and symptoms may be confused with acute myocardial infarction, which should be excluded.

In the approach to patients with suspected acute aortic syndrome, the radiologist's role is fundamental for correct diagnostic and therapeutic management. The tools available in these cases are:

- **Aortography**
- **Computed Tomography** (CT),
- **Transoesophageal echocardiography** (TEE),
- **Magnetic Resonance Imaging** (MRI).

**Aortography**, has been considered for many years the technique of choice in the diagnosis of dissection. The development and improvement of Noninvasive techniques has changed the concept.

**Computed Tomography** (CT), because of its speed and its wide availability, is currently the most common diagnostic imaging method for the study of acute aortic dissection. Helical CT enables the diagnosis of acute aortic dissection with a sensitivity and specificity of nearly 100%. Modern multisection CT allows rapid image acquisition and data reconstruction and aids in treatment planning. It helps differentiate type A from type B dissection, may localize the intimal entry site, and helps assess branch-vessel involvement and compromise and the relationship of the branch vessels to the true or false lumen. This information aids in planning treatment with either root replacement, intravascular stent placement, or fenestration.

However, it has some disadvantages, such as the use of nephrotoxic contrast, trouble viewing the gateway and the absence of hemodynamic information.
Transoesophageal echocardiography (TEE), is reported to have a sensitivity of 94%-100% and a specificity of 77%-100% for identifying an intimal flap. However, the distal part of the ascending aorta and the branches of the aortic arch may not be adequately evaluated with TEE. Neither TEE can be determine the extent of the dissection in the abdominal aorta and iliac arteries. However, to determine the existence, degree and the mechanism of aortic regurgitation and ventricular function.

Magnetic Resonance Imaging (MRI), provide a complete and dynamic display of aortic dissection, help determine the type of dissection, display the true and false lumina, the intimal flap, the location and size of the initial entry and its relationship with the neighboring arterial orifice, the origin of the aortic branches, and the presence and amount of thrombus in the false lumen, which is important for planning of surgical and endovascular therapy.

Both TEE and MRI has advantages of not radiate and the possibility of functional assessment of the heart chambers and aorta. But they also have limitations such as operator-dependent, as in the cases of the TEE, longer acquisition time and it is not appropriate for patients with implanted electronic devices... in the case of the MRI.

Definitely, acute aortic syndrome is a radiological emergency, where time is very important, could say that "time is life".
Findings and procedure details

TYPICAL AORTIC DISSECTION

Aortic dissection is the most common acute emergency condition of the aorta, and the most serious, often resulting in the death of the patient.

Pathogeny

An aortic dissection is characterized by an entrance tear within the intima and media of the aorta, which allows blood flow to create a false lumen within the medial layers of the aortic wall. The media separates in a course parallel to that of the flow of blood. This results in two lumina: a true lumen and a false lumen.

The false lumen lies within the outer half of the media, so that the outer wall of the false lumen is very thin, usually only about one quarter as thick as the original aortic wall. The thinness of the outer wall of the false lumen explains its tendency to rupture. The tearing of the intima generates an intimal flap that is also formed by the middle layer (fig. 1).
Fig. 1: Schematic of aortic layers in typical aortic dissection shows a tear of the intimal layer, which has resulted in the formation of two lumina (one false, one true).

References: Castañer E et al. CT in Nontraumatic Acute Thoracic Aortic Disease: Typical and Atypical Features and Complications. RadioGraphics 2003; 23

Classification

The original system for classification of aortic dissection, the DeBakey system, based on the anatomical and pathological features, has been superseded by the Stanford system, which includes two types based on the need for surgical intervention (fig. 2).
• **Stanford type A** (DeBakey types I and II), dissection involves the ascending aorta or the aortic arch. Such dissections account for 60%-70% of cases and typically require urgent surgical intervention.

• **Stanford type B** (DeBakey type III), dissection involves the descending thoracic aorta distal to the left subclavian artery and accounts for 30%-40% of cases.

**Fig. 2**: Figure 2: Drawing shows the Stanford classifications of aortic dissections and the equivalent DeBakey classifications.

**References**: Castañer E et al. CT in Nontraumatic Acute Thoracic Aortic Disease: Typical and Atypical Features and Complications. RadioGraphics 2003; 23

**Imaging Features of Aortic Dissection**

**Unenhanced CT**

Although the primary role of unenhanced CT is to detect intramural hematoma, in the case of aortic dissection, one can see, occasionally, the internal displacement of intimal
calcifications. This finding can be problematic because it may be confused with an aneurysm with calcified mural thrombus (fig. 3).

**Fig. 3:** Unenhanced CT scan depicts calcified thrombus (arrow) in the descending aorta, which mimics displaced intimal calcification.

**References:** Servicio de Radiodiagnóstico, Hospital regional universitario Carlos Haya - Málaga/ES

**Contrast-enhanced CT**

The main finding on contrast-enhanced CT scans of aortic dissection is an **intimal flap** that separates the true lumen from the false lumen (fig. 4). The intimal flap tends to acquire a spiral arrangement in the longitudinal images (fig. 5).
**Fig. 4:** Contrast-enhanced CT scans of Stanford type B (a) and type A (b) typical aortic dissection show the intimal flaps (arrows) in the descending aorta (a) and the aortic arch (b).

**References:** Servicio de Radiodiagnóstico, Hospital regional universitario Carlos Haya - Málaga/ES

**Fig. 5:** Contrast-enhanced CT, sagittal reconstruction (a) and volume rendered (b), the intimal flap is seen with a spiral arrangement.
Identifying true and false lumen on Aortic dissection is important for planning surgical repair or endovascular treatment. Among the findings that can be found to differentiate both lumen include:

Findings that indicate the **true lumen** include (fig. 6-7):

- True lumen is **smaller** than the false lumen
- and **increased attenuation** due to increased blood flow velocity.
- In 80% of cases it is compressed by the false lumen.
- The true lumen is **in continuity with the undissected** portion of the aorta.
- **Atherosclerotic calcification** in the outer wall aortic lumen indicates the true lumen, in a acute aortic dissection. However, in chronic dissection the outer wall of the false lumen can endotelizar and calcify, so that the sign can not be valid.
- Given an aortic segment with three lumen, the center is the true lumen and the other corresponding to the false lumen.

![Fig. 6](image-url): Stanford type A typical aortic dissection. Sequential contrast-enhanced CT scans show a true lumen (*) smaller that false lumen. The true lumen of the ascending aorta is compressed
Fig. 7: Contrast-enhanced CT scans of Stanford type A typical aortic dissection show atherosclerotic calcification in the outer wall aortic lumen indicating which is the true lumen.

References: Servicio de Radiodiagnóstico, Hospital regional universitario Carlos Haya - Málaga/ES
Fig. 8: Stanford type A typical aortic dissection. Given an aortic segment with three lumen, the center is the true lumen and the other corresponding to the false lumen. References: Servicio de Radiodiagnóstico, Hospital regional universitario Carlos Haya - Málaga/ES

Important signs that indicate the **false lumen** include (fig. 9-10):

- **Larger cross-sectional area**
- The "**cobweb sign**" as slender linear areas of low attenuation specific to the false lumen due to residual ribbons of media that have incompletely sheared away during the dissection process.
- "**Beak sign**", it is seen at cross-sectional imaging, and the acute angle formed between the junction of intimal flap and the outer wall of the false lumen, which is formed as the wedge hematoma that cleaves a space for the propagation of the false lumen.
- The false lumen may become **thrombosed**.
Fig. 9: Stanford type A typical aortic dissection. Sequential contrast-enhanced CT scans show a the beak sign (arrow), a wedge of hematoma is thought to create a space for the development of the false lumen

References: Servicio de Radiodiagnóstico, Hospital regional universitario Carlos Haya - Málaga/ES
**Fig. 10:** Stanford type A typical aortic dissection. Sequential contrast-enhanced CT scans show a cobweb sign (arrow), linear traces of low attenuation in the false lumen

**References:** Servicio de Radiodiagnóstico, Hospital regional universitario Carlos Haya - Málaga/ES

**Evolution and complications**

The possible evolution of aortic dissection may be the following (fig. 11):

- **Rupture**, the risk of fatal aortic rupture in patients with untreated proximal aortic dissection is approximately 90%.
- **Reentry** of dissecting hematoma. In this case be an aorta with double light, the false usually be greater than the true.
- Total or partial **thrombosis** of the false lumen (fig. 12).
- **Compression** of the true lumen. When the flap adopts a concave stance toward the false lumen and the true light is C-shaped, there is a deficit of pressure in the true lumen and aortic branches in irrigated through this light. As a result of this malperfusion syndrome occurs.
- **Aortic remodeling**, Occurs over time, both in cases with double lumen patency as where there has been a thrombosis of the false lumen.
Fig. 11: Schematic of the evolution of aortic dissection.

Fig. 12: Stanford type B aortic dissection. Contrast-enhanced CT scans show a thrombosed false lumen (*). Arrow indicates a small residual flow channel in the false lumen.

References: Servicio de Radiodiagnóstico, Hospital regional universitario Carlos Haya - Málaga/ES
Fig. 13: contrast-enhanced CT scans show a aortic dissection in which is observed as the celiac trunk arises from the false lumen.

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**Fig. 14**: Axial images through the abdomen shows dissection flap involving the origin of the superior mesenteric artery and along its path (arrow). The dissection flap is seen to enter the origin of the artery, thus compromising its lumen.

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**Fig. 15**: Iliac arteries. Axial image of the abdomen demonstrates a dissection flap in the iliac arteries.

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**INTRAMURAL HEMATOMA:**

**Pathogeny**

Vasa vasorum (VV) are small arteries which penetrate the arterial wall both through the luminal surface (vasa vasorum internae) and the adventitia (vasa vasorum externae). Adventitial VV ramify into smaller vessels supplying the outer media layers. Venous VV are more numerous and also supply mainly the outer media.

VV seem to play a fundamental role in the physio-pathology of the three acute aortic syndrome entities.

An intramural hematoma results from rupture of the vasa vasorum and hemorrhage into the arterial media, which leads to weakening of the aortic wall. The distinguishing feature of this entity is an absence of the intimal disruption that characterizes classic aortic dissection (Fig 16 y 17).

**Fig. 16**: Diagrams show the pathogenesis of aortic intramural hematoma. Hemorrhage resulting from rupture of the vasa vasorum weakens the media of the aortic wall and leads to enlargement of the aortic diameter. In an intramural hematoma, unlike an aortic dissection or penetrating ulcer, the intima remains intact.

**References**: Chao CP et al. Natural History and CT Appearances of Aortic Intramural Hematoma. RadioGraphics 2009; 29
**Fig. 17**: Diagram shows events leading to intramural hematoma from ruptura of vasa vasorum feeding aortic media to creation of intramedial hematoma with intact intimal layer

**References**: old.ctisus.com

**Classification.**

Intramural hematomas are classified as aortic dissection by Standford Rating described above.

**Imaging Features of Intramural hematoma.**

**Unenhanced CT**

Unenhanced axial CT images, a crescentic, eccentric, hyperattenuating region of thickening of the aortic wall (diameter, >7 mm; attenuation, 60-70 HU) is considered diagnostic of acute intramural hematoma (Fig. 18).

In intramural hematoma as in aortic dissection, intimal calcifications may be displaced inward; however, in the presence of intramural hematoma, such calcifications usually appear in a semicircular or circular curvilinear configuration rather than the linear configuration seen in the presence of an intimal flap (fig. 19).
Fig. 18: Type B IMH. (a) Unenhanced CT scan depicts crescent-shaped areas with high attenuation (arrows) extending along the walls of the descending aorta.

References: Servicio de Radiodiagnóstico, Hospital regional universitario Carlos Haya - Málaga/ES
Fig. 19: Unenhanced axial image shows intimal calcifications in a curvilinear configuration and a crescentic hyperattenuating intramural fluid collection

References: Servicio de Radiodiagnóstico, Hospital regional universitario Carlos Haya - Málaga/ES

Contrast-enhanced CT

The intramural fluid collection appears as a non-enhancing, smooth, crescentic region of aortic wall thickening that extends partially or entirely around the opacified aortic lumen, and no spiraling of an intimal flap is seen (fig. 20).

In addition, the absence of a dissection flap, intimal tear, or penetrating atherosclerotic ulcer is a prerequisite for the diagnosis of intramural hematoma.
Fig. 20: Contrast material-enhanced axial image depicts a smooth, nonenhancing, crescentic region of aortic wall thickening without a spiraling intimal flap.

References: Servicio de Radiodiagnóstico, Hospital regional universitario Carlos Haya - Málaga/ES

Evolution

The natural history of an intramural hematoma may include periods of stabilization, regression, and resolution or may consist of continuous disease progression. Complications may occur at any stage in the evolution of an intramural hematoma. Potential complications include progression to overt aortic dissection, development of ulcerlike projections of the aorta, and formation of an aortic aneurysm (Fig. 21-22).
**Fig. 21**: Schematic of the evolution of intramural hematoma. Based at Vilacosta I. Síndrome aórtico agudo.

**References**: Vilacosta I. Síndrome aórtico agudo. Rev Esp Cardiol 2003;56

**Fig. 22**: Type B IMH, sequential contrast-enhanced CT scans acquired 1 week later because the patient reported persistent pain shows aortic dilatation and dissection of the lumen (arrows).
Penetrating atherosclerotic ulcer refers to an ulcerating atherosclerotic lesion that penetrates the elastic lamina and is associated with hematoma formation within the media of the aortic wall.

Initially, atheromatous ulcers develop in patients with advanced atherosclerosis. At this stage, the lesions are usually asymptomatic and confined to the intimal layer. In the next stage, the lesion progresses to a deep atheromatous ulcer that penetrates through the elastic lamina and into the media (fig. 23).

Hematoma formation may extend along the media, resulting in either "double-barreled" or "thrombosed" aortic dissection. Double-barreled aortic dissection demonstrates communication between the true and false lumina, whereas thrombosed aortic dissection shows no opacification of the false lumen. Thrombosed aortic dissection is probably more common than double-barreled aortic dissection because severe atherosclerosis is believed to prevent the extension of hematoma and the creation of reentry.
The most common site of penetrating ulcers is the descending thoracic aorta, followed by the abdominal aorta, but as with other atherosclerotic lesions, can be found all throughout the aorta. They can be single or multiple and, in some cases, bilobed.

**Imaging Features of Penetrating Atherosclerotic Ulcer.**
**Unenhanced CT**

On unenhanced axial CT include focal involvement with adjacent subintimal hematoma located beneath the frequently calcified and inwardly displaced intima in the middle or distal third of the thoracic aorta.

**Contrast-enhanced CT**

On contrast-enhanced CT scans of penetrating atherosclerotic ulcer, a collection of contrast material is seen outside the aortic lumen. the ulcer is often associated with thickening or enhancement of the aortic wall (fig. 24).

Atheromatous ulcers that are confined to the intimal layer sometimes appear radiologically similar to penetrating atherosclerotic ulcers. The extension of the penetrating atherosclerotic ulcers beyond theoretical edge of the aortic wall and the presence of a cap of hematoma on the ulcer help differentiating this one against the atheromatous ulcer, that occurs more frequently.
**Fig. 24:** contrast-enhanced CT scans of penetrating atherosclerotic ulcer, a collection of contrast material is seen outside the aortic lumen (arrow).

**References:** Servicio de Radiodiagnóstico, Hospital regional universitario Carlos Haya - Málaga/ES

**Evolution**

The natural history of a penetrating atherosclerotic ulcer may include (Fig.25):

- Intramural hematoma
- Saccular aneurysm
- Subadventitial pseudoaneurysm
- Aortic dissection,
- Aortic rupture.
The most common ulcer development is dilation progressive aortic aneurysm. It can also complete destruction of the middle layer occur and formed a pseudoaneurysm.

Although many patients with penetrating atherosclerotic ulcer have some degree of hematoma, its propagation along the descending aorta is hampered by the fibrosis and calcification of the aortic wall, and therefore, usually a small hematoma extension longitudinal

Penetrating atherosclerotic ulcer may be the origin dissection. Here the door entry would be the ulcer crater. The differential characteristics dissection of such are: intimomedial flap thick, calcified, irregular, frayed and stationary or small amplitude oscillatory; extension longitudinal limited, location in the descending aorta. The true lumen size is equal to or greater than that of the false light and often is retrograde because
arteriosclerosis and the adjacent aortic calcification will sometimes require extended in this direction.

**TREATMENT**

Once the diagnosis is made the treatment of the various entities that make up the AAS will be determined by the location of the affected aorta, the patient's clinical situation and accompanying complications.

**Treatment of type A (ascending aortic):**

Dissection, IMH, or PAU located in the ascending aorta is a strong indicator of disease progression. It can rapidly extend towards the heart and cause death. Type A IMH, PAU, and thoracic aneurysm are likewise at increased risk of complications. Early surgical intervention is advocated for type A pathology and this has been shown to improve prognosis.

**Treatment of type B (descending aortic):**

A conservative *medical* approach to type B aortic disease is considered acceptable when the lesion is stable. When there is evidence of disease progression, not be able to use medical treatment, if this would be:

- Persisting acute aortic pain despite medical management
- Increasing aortic wall thickness or diameter
- PAU greater than 20 mm in diameter or 10 mm in depth
- Increased volume or extent of haematoma
- Bulging haematoma
- Extra-adventitial blood
- Increasing pleural effusion
- IMH associated with PAU

*Surgery* is also complicated by serious morbidity. These include cardiac (10%) and respiratory complications (28%), renal failure (16%-17%), and paraplegia (7%-27%). Therefore although surgical treatment has been used in cases that are unsuitable for conservative management, it has failed to improve prognosis and is in fact no better than medical therapy.
Endovascular aortic repair (EVAR) (figure), involves the use of stent grafts which because of their size, are normally introduced through a femoral cut down. There are a number of potential benefits in the use of endovascular techniques to treat aortic pathology:

- Minimal access procedure
- Procedure undertaken in surgically unfit patients
- Short procedure time
- Minimal blood loss
- Shorter recovery time
- Financial savings
- Obliteration of false lumen in aortic dissection
- Occlusion of diseased segment of aorta
- Reversal of end organ ischaemia
- Reduced morbidity and mortality compared with surgery
Fig. 1: Schematic of aortic layers in typical aortic dissection shows a tear of the intimal layer, which has resulted in the formation of two lumina (one false, one true).

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**Fig. 23:** Penetrating ulcer scheme

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Fig. 25: Evolution of penetrating atherosclerotic ulcer. Based at Vilacosta I. Síndrome aórtico agudo.

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Conclusion

AAS is a medical and radiological emergency that requires early and accurate diagnosis.
CT is the imaging modality of choice.
DA is the most frequent and serious cause, and treatment varies depending on the entity and classification.
References


