Assessment of Acute Aortic Syndrome in the Emergency Department

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Learning objectives

- Review the basic concept of acute aortic syndrome (AAS), its pathophysiological mechanisms and clinical characteristics of the entities that comprise the AAS.

- Identify and describe the radiological findings of the entities included in the acute aortic syndrome.
Background

Acute aortic disease is a critical clinical entity which have a prognosis that often depends on an accurate diagnosis and prompt, as well as the early onset of the treatment.

This poster will focus on educational institutions that comprise acute aortic syndrome and its pathophysiological mechanisms, clinical features and assessment of the radiological findings at the emergency department.

Because of their similarities in clinical manifestations, risk factors and possible fatal consequences as rupture, no trauma acute emergencies affecting the thoracic aorta are included within the same entity called acute aortic syndrome (Fig. 1).

Acute Aortic Syndrome

Acute aortic syndrome (AAS) include:
1. Acute aortic dissection.
2. Intramural hematoma.
3. Penetrating atherosclerotic ulcer.

The aorta is a unique organ, in structure and functional behavior, is composed of the intima, media, and adventitia. The intima, the innermost layer, is thin, delicate, lined by endothelium, and easily traumatized.

The media is responsible for imparting strength to the aorta and consists of laminated but intertwining sheets of elastic tissue. The arrangement of these sheets in a spiral provides the aorta with its maximum allowable tensile strength. The aortic media contains very little smooth muscle and collagen between the elastic layers and thus has increased distensibility, elasticity, and tensile strength. This contrasts with peripheral arteries, which, in comparison, have more smooth muscle and collagen between the elastic layers.

The outermost layer of the aorta is adventitia. This largely consists of collagen. The vasa vasorum, which supplies blood to the outer half of the aortic wall, lies within the adventitia. The nervi vascularis, bundles of nerve fibers found in the aortic adventitia, are involved in the production of pain, which occurs with acute stretching of the aortic wall from a dissection. (Fig. 2).
Acute aortic syndrome is an acute entity of aortic wall that causes a weakening of the middle layer and increases the risk of aortic rupture and other complications, with high morbidity and mortality (Fig. 3).

The pathogenesis of AAS is often multifactorial and these conditions are often indistinguishable on clinical examination. Computed tomography (CT) is a highly accurate method for diagnosis at emergency departments and is useful in treatment planning.

Clinical presentation:

Aortic dissection is presented as:
Tearing pain, throbbing and migratory, located at precordium that radiates to the neck or interscapular region.

Sudden onset of severe hypotension.

Syncope without neurologic focality due to proximal aortic rupture into the pericardial cavity with tamponade or, less frequently, in the left pleural space.

Heart Failure due severe aortic insufficiency.

Signs suggestive organic vascular insufficiency or poor peripheral perfusion.

Intramural hematoma is indistinguishable from a clinic acute dissection.

Penetrating ulcer appears as dorsal chest pain at elderly patients with a history of hypertension and vascular atherosclerosis.
In a significant number of patients with the clinical semiology and physical examination may be normal at the time of presentation.

AAS:

Intramural hematoma:

Aortic IMH is considered a precursor of dissection, originating from ruptured vasa vasorum in medial wall layers (aortic wall apoplexy) potentially provoking secondary tear
and classic aortic dissection; IMH may, progress, dissect, regress, or resorb; two-thirds of cases are located in the descending aorta and are typically associated with hypertension. Similar to dissection, chest pain is more common with ascending (proximal) IMH, whereas back pain is more common with descending (distal) IMH. Nonetheless, the diagnosis of IMH cannot be made on clinically grounds, but by tomographic imaging in the appropriate clinical setting.

Penetrating atherosclerotic ulcer:

This is a lesion that penetrates the intima and progresses into the media. In the early stages the lesions just ulcerate the intima and are often asymptomatic. With further progression they ulcerate the media and lead to a hematoma of variable size within the media.

PAUs originate from atherosclerotic aortic segments and are localized in the descending thoracic aorta in over 90%. The typical patient is elderly (usually over 65 years of age), hypertensive with atherosclerosis, presenting with chest or back pain but no signs of aortic regurgitation or malperfusion; asymptomatic patients may also be found with aortic lesions indistinguishable from PAU by imaging criteria.

Acute aortic dissection:

An aortic dissection is a tear in the inner layer of the aortic wall, which allows blood to enter into the wall of the aorta, creating a new passage for blood, known as the "false lumen." Blood flow into the false lumen can cause several problems: It can rob crucial blood from the rest of the body, it can cause the dissection to spread and affect other arteries, and it can block blood flow in the true aortic channel ("true lumen"). These problems may cause decreased blood flow to vital organs. Aortic dissection also weakens the aortic wall and may lead to rupture, which may be fatal, or produce an aneurysm.

Classification:

Dissections of the thoracic aorta have been classified anatomically by 2 different methods. The more commonly used system is the Stanford classification, which is based on involvement of the ascending aorta and simplifies the DeBakey classification.

DeBakey classification. - (Fig. 4).

The DeBakey classification divides dissections into 3 types, as follows:

- Type I involves the ascending aorta, aortic arch, and descending aorta
- Type II is confined to the ascending aorta

- Type III is confined to the descending aorta distal to the left subclavian artery

Type III dissections are further divided into IIIa and IIIb. Type IIIa refers to dissections that originate distal to the left subclavian artery but extend proximally and distally, mostly above the diaphragm.

Type IIIb refers to dissections that originate distal to the left subclavian artery, extend only distally, and may extend below the diaphragm.

Stanford classification.- (Fig. 4).

The Stanford classification divides dissections into 2 types, type A (fig. 5) and type B (Fig. 6). Type A involves the ascending aorta (DeBakey types I and II); type B does not (DeBakey type III).

This system helps to delineate treatment. Usually, type A dissections require surgery, while type B dissections may be managed medically under most conditions.
Fig. 1: Shows the relationship between the three classical components of acute aortic syndrome.

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Fig. 2: Histology: concentric layers of the aorta

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Fig. 3: Pathophysiological mechanism of acute aortic syndrome.

© Haberman D, Gurfinkel E, Martínez A et al. Angiotomografía computada multicorte de 64 canales en la evaluación de la patología aórtica aguda.
Fig. 4: Classification of aortic dissection according to the nomenclature of De Bakey and Stanford.

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Fig. 5: Thoracic aortic aneurysm with acute aortic dissection Stanford type A. Dissection starts at the origin of the ascending aorta to the abdominal aorta. The intimal flap has less attenuation than blood opacified by the contrast. The green arrow indicates the false lumen and red arrow the true lumen.

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Fig. 6: Acute aortic dissection Stanford type B. Dissection begins immediately posterior to the output of the left subclavian artery to the abdominal aorta. The false lumen (green star) is thrombosed. The right renal artery arises from the true lumen (red star).

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Imaging findings OR Procedure details

Radiological findings:

Imaging techniques play a vital role in the management of acute aortic syndrome.

Chest radiography is widely used as a screening tool, but the sensitivity is very low and with a tendency to misinterpretation.

CT is the method of choice for diagnosis because of its high sensitivity and specificity, approaching 100%, and because it is a quick, easy and available in most hospital emergency departments.

Acute aortic dissection (Fig. 7).

Chest radiography:

The mediastinal widening or progressive changes in the configuration of the aorta on serial radiographs are suspected of dissection. Unexplained differences in the size of the ascending and descending thoracic aorta are also suggestive. The displacement of the trachea or esophagus. Signs suggestive of pleural effusion, the presence of an apical cap or paraspinal thickening suggest extravasation from dissection, growth of cardiopericardial silhouette, or radiological signs of acute heart failure.

Displacement of intimal calcium to the center of the aortic lumen (Fig. 8).

Hypodense line within the aortic lumen (flap).

Semilunar area of increased attenuation within the wall of the aorta (rupture of the vasa vasorum in the absence of intimal tear) in unenhanced CT.

Presence of two aortic lumen:

- True lumen (Fig. 9):

Smaller, more dense.
Compressed by the false lumen in 80% of cases.
Wall has calcium
The celiac, superior mesenteric artery and right renal artery usually arise from the true lumen

- False lumen (Fig. 9):
  Larger, less dense.
  Intimal flaps can have inside.
  Presents partial and irregular mural thrombosis.
  Presents acute angle between the wall and the flap.
  Intimal flap discontinuity.
  The false lumen usually thin linear zones corresponding to lower attenuation of the residual strands middle layer is not torn completely (sign of the web) (Fig. 10).

Hemopericardium.

Aortic intramural hematoma. (Fig. 11)
Internal displacement of calcium.

Crescentic thickening or, less frequently, concentric, hyperdensity of the aortic wall on CT without contrast and hypodense posterior to administration of contrast (Fig. 12).

Extension smaller than aortic dissection.
Smooth inner and outer margin (contained by the intima and adventitia respectively).
Small ulcers due to communications or avulsion of the intercostal and lumbar arteries.

Penetrating aortic ulcer. (Fig. 13).
Internal displacement of calcium mural.

Hypodense line in the center of the aortic lumen (intima displaced) very short length associated to a entrance gateway (Fig. 14).

Intimal calcifications around the entrance of the ulcer (broken atheromatous plaque).
Crescentic hyperdense on unenhanced CT above and below the ulcer corresponding to intramural hematoma.
Atheromatous signs (calcium and mural thrombosis and aneurysmal areas) in the remainder of the aortic segments.

The entities in the acute aortic syndrome are usually clinically indistinguishable but show different characteristics in radiological imaging studies. These are important conditions to identify and correctly diagnose these entities and allow prompt and appropriate management based on clinical and radiological findings.

Advances in treatment and imaging techniques have shown the importance of early diagnosis, to be crucial for survival. However, despite the above, the acute mortality remains high. The initial high clinical suspicion and improved experience in multidisciplinary teams are the only variables that have reduced mortality
**Fig. 7:** Acute aortic dissection Stanford type A. The green arrow indicates thrombosed of the false lumen at the distal part of the aortic arch, the red arrow indicates the true light. Pericardial effusion was also evident.

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**Fig. 8:** Thoracic aortic aneurysm with acute aortic dissection Stanford type A. the intimal flap can be seen in the aortic arch with some calcification. The green arrow indicates the false lumen and red the true lumen.

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**Fig. 9:** Thoracic aortic aneurysm with acute aortic dissection Stanford type A. the false lumen (green arrow) is opacified with lower density, looks more irregular and contains a thrombus, the true lumen (red arrow) is opacified with more density and is smaller.

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Fig. 10: The sign of the web (Cobweb sign) is exclusive of the false lumen. Represents strands or ribbons of media crossing the false lumen, and appearing as thin filiform filling defects.

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**Fig. 11:** Infrarenal abdominal aortic aneurysm with an extensive intramural hematoma with higher density areas inside.

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**Fig. 12:** Infrarenal abdominal aorta aneurysm which shows a concentric thickening of the aortic wall with a slight increase of the attenuation of the hematoma. It also presents a bad left posterolateral edge definition as a sign of imminent rupture.

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Fig. 13: Penetrating atherosclerotic ulcer: Contrast opacification of focal ulceration extending into the aortic wall

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**Fig. 14:** Penetrating atherosclerotic ulcer: The arrow denotes a focal dilatation of the right anterolateral wall of the infrarenal abdominal aorta.

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Conclusion

Acute aortic dissection is a clinical emergency, which prognosis usually depends on an accurate diagnosis and prompt, and the early onset of treatment.

Advances in computed tomography (CT) have made the diagnosis of acute aortic syndromes easier and faster.

The role of the radiologist is of utmost importance in the management of patients with suspected SAA. A better understanding of the natural history and predictors of the disease provides a great help the clinician to raise the most appropriate therapeutic strategy.
References


