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Successful Surgical Outcome and Short-Term Follow-Up of Type A Acute Aortic Dissection in a Young Patient: a Case Report

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ABSTRACT

We report the case of a 34-year-old male patient presenting with type A Stanford aortic dissection, sustained by transesophageal echocardiography and thoracic-abdominal CT with intravenous contrast; he had no previously known associated diseases, and the initial symptoms were relatively uncommon. We performed emergency surgical correction by Bentall procedure combined with coronary bypass, followed by good short-term results.

Keywords : aortic dissection, transesophageal echocardiography, thoracic CT-angiography, Bentall procedure

INTRODUCTION

Acute aortic dissection is the most common life-threatening disorder affecting the aorta. The immediate mortality rate in aortic dissection is as high as 1% per hour over the first several hours, making early diagnosis and treatment critical for survival [(1)].

Aortic dissection occurs from a rupture of the intimal layer, which allows blood to enter the media and dissect between the intimal and the adventitial layers. The estimated incidence of aortic dissection is 5 to 30 cases per million population per year. Chronic systemic hypertension is the most common factor predisposing to aortic dissection and is present in 62–78% of patients with aortic dissection [(2)]. The diagnosis is often difficult to make because not all dissections have the typical presentation of sudden severe chest pain radiating to the back. Symptoms often include abdominal pain, flu-like complaints, vomiting and diarrhea, low back pain, stroke syndromes and syncope. Strategies to reduce risk and improve outcome include staff education on various presentations and risk factors, rapid availability of diagnostic testing modalities such as chest CT scan or transesophageal echocardiogram, and protocols to ensure prompt transfer for cardiothoracic surgery [(3)].

THE CASE

Clinical Presentation

A 34-year-old male, without significant past medical history (except for occasional hypertension episodes which didn't impose the onset of medical therapy), suddenly presented intense diffuse chest pain accompanied by profound dyspnoea and immediately followed by syncope and coma. He was admitted in our service soon after these symptoms' appearance. On physical examination, the patient was unconscious, with a GCS of 5 pts, and presented respiratory distress caused by acute pulmonary edema, and demanding artificial ventilation through orotracheal intubation. The blood pressure was 130/80 mmHg in both arms and the cardiac rhythm was regular with a frequency of 65/min; the peripheral pulses were present bilaterally. Heart sounds were normal, but with a grade II diastolic murmur in the aortic auscultation area. The ECG showed normal sinus rhythm and elevated ST segment in the inferior territory. The laboratory tests revealed increased WBC count at 18.870/µl, otherwise the values of hemoglobin, serum ions and coagulation markers were normal.

Investigation

At presentation we performed transesophageal echocardiography which revealed a dilation of the ascending aorta (of about 65 mm), a second degree aortic regurgitation, with an intimal flap from 8 mm above the aortic valve involving the right coronary artery, and the extension of this flap to the aortic arch, the origin of carotid arteries and the descending aorta; there was no evidence of pericardial effusion, and the left ventricular ejection fraction was within normal limits (Figures 1 and 2).

Figure 1 - Transesophageal image of descending thoracic aorta. The intimal flap divides the aortic lumen in "true" lumen (arrow) and "false" lumen.

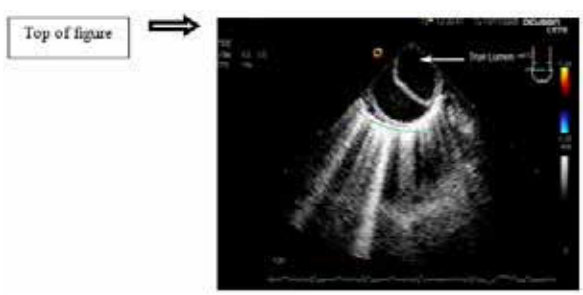
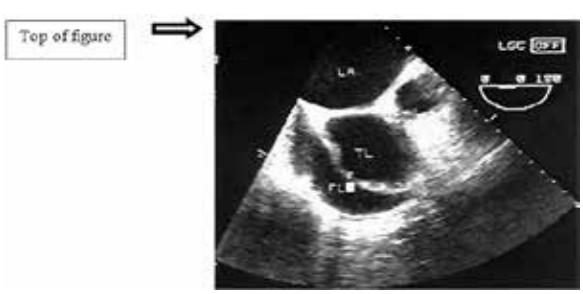


Figure 2 - High transesophageal image of ascending aorta and left atrium (LA), also showing the intimal flap (F) which divides the aortic lumen into true lumen (TL) and false lumen (FL).



The contrast CT scan of the chest and abdomen demonstrated the presence of two distinct lumens with a visible intimal

flap in the ascending aorta with extension to the aortic arch and its branches, and the descending thoracic aorta; also, the intimal dissection could be observed in the sub-renal aortic segment and common iliac arteries. Therefore, the diagnosis of Stanford type A aortic dissection was confirmed (Figures 3, 4 and 5).

Figure 3 – Axial slice of thoracic CT-angiography showing the dissection of ascending and descending aorta with collapsed true lumen (arrowheads) and large, circulated false lumen (arrow).

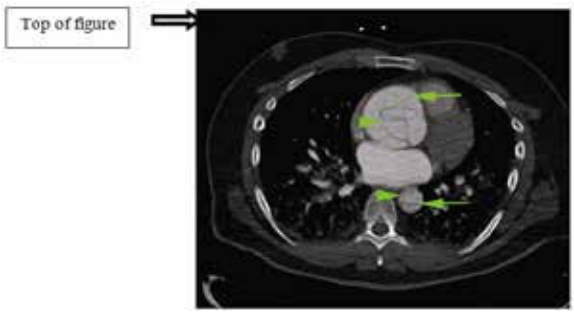


Figure 4 - Axial slice of thoracic CT-angiography showing the dissection flap extension on the supra-aortic vessels with large, circulated false lumen (arrows).

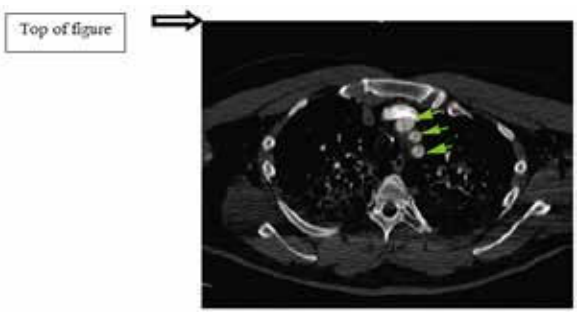
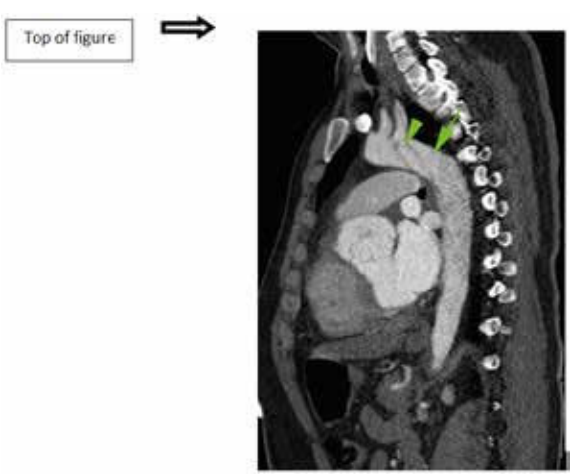


Figure 5 - MPR sagittal reconstruction of thoracic CT-angiography showing the extension of the dissection flap on the descending aorta, with both lumens circulated (false lumen – arrow, true lumen – arrowhead).



Management

The patient was immediately taken to the OR for the surgical repair of type A aortic dissection. Right femoral artery cannulation, median sternotomy, and right atrial cannulation for total cardiopulmonary bypass were carried out. At the opening of the pericardium there was a typical dilation of the ascending aorta (approximately 65 mm) extended to the aortic arch; after aortotomy we found an infiltrated aortic wall, with hematoma at the sinus of Valsalva level, and a circumferential dissection and intimal rupture, extended proximally to the aortic ring. There was also a medium aortic regurgitation and the intimal rupture circumferentially extended at the origin of right coronary artery. At the aortic arch level we found no intimal tear. Subsequently, the Bentall procedure was performed: the segment of the aorta containing the intimal tear was resected and replaced with a Dacron graft; teflon felt was attached to the aortic wall with continuous sutures on the outer bound; also, glue was applied to fill the entire space between the dissected layers; the aortic valve was replaced by a mechanical prosthesis; the reimplantation of the left coronary artery into the prosthetic graft and a coronary bypass graft with saphenous vein on the second segment of right coronary artery was accomplished. CBP and aortic cross-clamp times were 300 and 240 minutes, respectively. After long-run anesthesia and CBP, the patient was admitted to intensive care unit with inotropic support. Also we associated beta-blockers, angiotensin inhibitors, antiplatelet drugs, and anticoagulants.

Figure 6 – Aortic wall (obx10) tinted with alcian blue PAS. The arrows indicate the "pools" of proteoglycans.

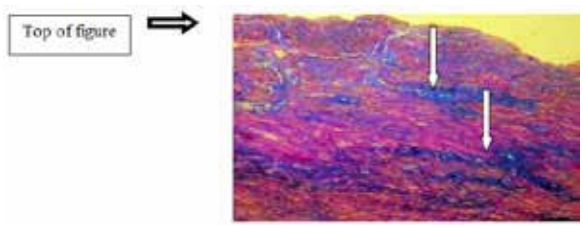
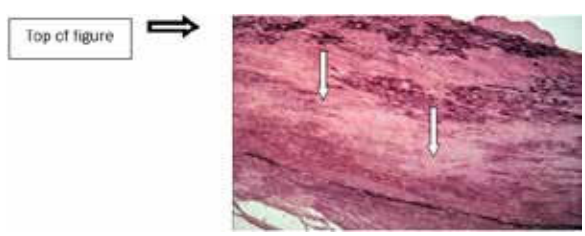


Figure 7 – Aortic wall (obx10) tinted witn orceine. The arrows indicate the areas in which the elastic fibers are absent.



In the early postoperative period the patient presented uncontrolled hemorrhage necessitating urgent reintervention for haemostasis (a source of bleeding at the level of proximal anastomosis of venous graft was found and resolved by supplemental sutures); also during the first 3 days after surgery he presented marked psycho-somatic agitation status (with the need for sedation) and disorientation, but the neurologic evaluation found no evidence of stroke.

In the late postoperative period, there were surgical wound complications, at the site of sternal incision (with remission after local antiseptic therapy), and at the site of femoral incision (for which surgical evacuation of lymphatic collection was performed). Also the patient presented prolonged fever (with a Tmax of 38.7°C), sustained by an inflammatory syndrome; this was assumed as a reaction to aortic graft, and remission was obtained after systemic anti-inflammatory therapy. The initial postoperative echocardiography and thoracic X-ray showed minimal pericardial and left pleural effusions, with no other pathologic findings.

The histopathologic exam of the resected aorta revealed the important alteration of the elastic structure of the aortic wall, with ruptured and tenuous elastic fibers; also the medial layer of the aortic wall was fulfilled with proteoglycans (Figures 6 and 7). The described aspects plead for the cystic degeneration of the aortic wall, this alteration being frequently associated with hypertension, bicuspid aortic valve, and the Marfan syndrome. In 20% of cases we find only an idiopathic dilation of the aortic root, without any associated disease. At discharge, the patient was hemodynamically and respiratory stable, with healed surgical wounds.

DISCUSSION

The young age of the patient, the absence of risk factors or associated diseases, and the lack of triggers for the sudden onset of symptoms made the clinical diagnosis in this case very challenging. Prompt diagnosis and management of aortic dissection are key to reduce patient morbidity and mortality; hence the need to have a high index of suspicion for this condition [4]. In this case the main diagnostic suspicion was for a cerebrovascular accident, but that may have catastrophic consequences in a clinical condition which, untreated, is associated with a high mortality during the first 48 hours after the onset of symptoms. The correct and definitive diagnosis was made by correlating a careful physical examination, echocardiography, and computed tomography. Non-invasive imaging modalities, such as spiral CT, multiplanar transesophageal echocardiogram (TEE), and magnetic resonance imaging (MRI) are replacing aortography for purpose of evaluating for aortic dissection [4]. TEE has the advantage that patients don't need to be transported for the procedure; it doesn't require the use of potentially nephrotoxic contrast agents and provides functional evaluation of aortic valve [4]. In our case this test was the first to establish the diagnosis; the hemodynamic and respiratory stability of the patient allowed us to perform the additional CT with intravenous contrast for more detailed information about the lesions extension.

In acute type A aortic dissection, the high risk of mortality with medical treatment and the more favorable surgical outcomes mandate urgent surgical intervention [5]. The surgical treatment in type A aortic dissection should alleviate symptoms, and to prevent complications and aortic wall rupture. Usually the procedure entails removing the intimal flap, obliterating the false lumen, and reconstruction of the aorta. In this case the need to perform a more complex procedure (Bentall procedure) was imposed by the associated lesions: the aortic regurgitation, and the intimal dissection extended to the origin of right coronary artery. However, despite the prolonged times of CBP and aortic cross-clamping, and the technical difficulties, the surgical outcome was good.

Regarding the immediate and late postoperative complications in the reported case, we consider them minor and transient; this is proved by the complete recovery of the patient with local or systemic treatment, and by the short-term follow-up.

One-month postoperatively, the surgical wounds appeared healed and the sternum was correctly consolidated; the blood pressure and the cardiac rhythm were within normal limits, but with a mild systolic murmur in the aortic auscultation area; laboratory findings showed no important abnormalities, with the exception of high levels of serum LDL-cholesterol.

The thoracic CT angiography taken six-months after the acute event revealed no arterial segments occluded in the examined region.

Fourteen-months after surgery, the patient was completely asymptomatic, and the clinical evaluation was within the normal limits. On the thoracic angio-CT there was evidence for dilated aortic arch (38 mm), with thrombotic deposits, with a diameter of circulated lumen of 7 mm, and an intimal dissection with extension to the descending thoracic aorta and to the origin of left carotid and subclavian arteries; also it showed dilated descending thoracic aorta (45 mm) with intimal dissection, with circulated true lumen of approximately 10 mm anteriorly situated, and the false lumen of 22 mm; the dissection extended inferiorly to the abdominal aorta and iliac arteries, with the celiac artery originating from the both lumens (Figures 8, 9, 10 and 11).

Figure 8 - Axial slice of thoracic CT-angiography showing the aortic valve and ascending aorta replacement without dissection flap, with false lumen thrombosis (arrow), and permeable, narrow true lumen (arrowhead), being compressed by the false lumen.

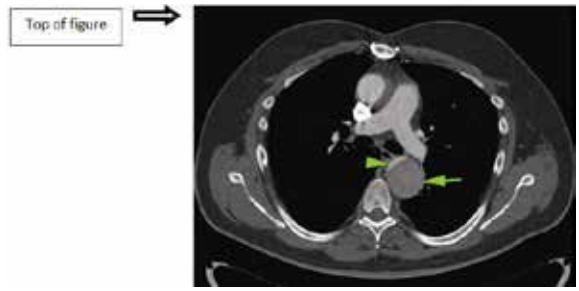


Figure 9 - Axial slice of thoracic CT-angiography at the supra-aortic branches level showing thrombosis of the false lumen (arrows).

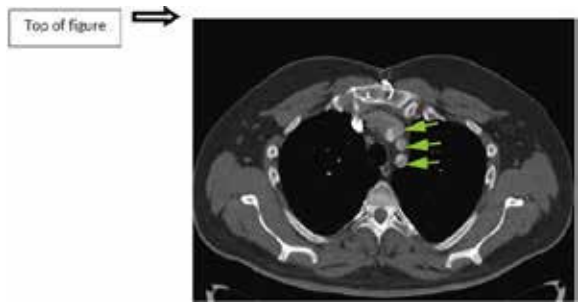


Figure 10 - Axial slice of thoracic CT-angiography showing the descending thoracic aorta with a narrow true lumen being compressed by the false lumen (arrow), which is circulated through a re-entry point (P).

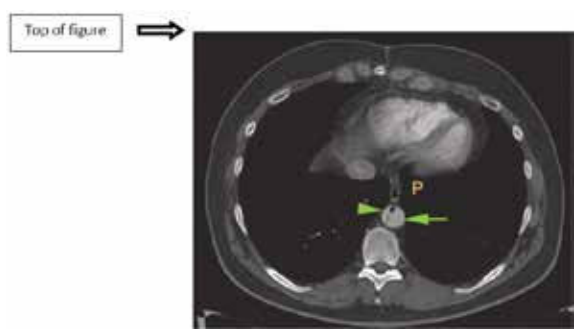
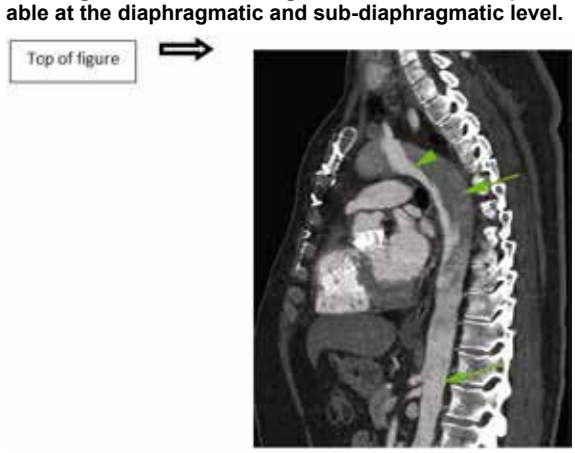


Figure 11 - MPR sagittal reconstruction of thoracic CT-angiography showing the narrow, circulated true lumen (arrowhead), being compressed by the false lumen (arrows); the false lumen is occluded (thrombosis) in the first segment of descending thoracic aorta, and permeable at the diaphragmatic and sub-diaphragmatic level.



Some studies have established that despite all actually possible techniques, about 70% of patients after surgery for acute type A preserve a patent false lumen and in about 50% subse-

quent aneurysm formation will follow [6]. If we want to improve the long-term results, we will have to aim at complications related to this. Other factors such as blood pressure control, may also play an important role and can easily be controlled [6].

CONCLUSION

Aortic dissection is the most lethal disease of the aorta. Its incidence is estimated to be 3 in a 1000 cases according to the international registry of aortic dissection (IRAD). If left untreated 33% of individuals will die within 24 hours of presentation, and 50% die in the initial 48 hours [7]. According to IRAD the in-hospital mortality for promptly surgically treated patients was 26.9% compared to 56.2% for those treated only medically. A study based on IRAD data suggests that for patients treated medically, long-term, ongoing medical treatment may provide some benefit [8]. The most common causes for death after acute aortic dissection type I include: rupture in the pericardial space followed by cardiac tamponade; occlusion of coronary arteries; rupture of the aorta in the thorax or abdomen. The reported case outlines the variety of clinical aspects, the importance of diagnostic imaging and the need for emergency treatment in the acute aortic dissection type I, in order to obtain a successful surgical outcome and to improve the quality of life.

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