

What hypertension can do – case report

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Abstract

Hypertension is one of the most frequent and ignored diseases of the adult worldwide. It usually does not hurt unless there is a peak of high blood pressure. Hypertension is often diagnosed in a screening measurement or at the hospital when the patient arrives with complications due to this hidden disease. One of the severe and deadliest complications can be aortic dissection. Some risk factors are described for aortic dissection, like hypertension, aortic aneurysm, bicuspid aortic valve, genetic disorders that affect the aortic wall (familial thoracic aortic aneurysm and dissection, Marfan syndrome, Ehlers-Danlos syndrome and others). Aortic dissection is a very unpredictable and deadly disease, especially if left untreated. Surgical treatment is indicated in all type A aortic dissections and must be done immediately. Surgical strategy can vary and it is done considering the lesion, patient characteristics, experience and possibilities. A heart team is absolutely necessary in order to improve outcomes and increase knowledge about this disease.

Keywords: hypertension, acute aortic dissection, cardiac surgery, deep hypothermic circulatory arrest.

Abbreviations: AD – aortic dissection; CAVGR – composite aortic valve graft replacement; CT – computed tomography; LVEF – left ventricle ejection fraction; NIRS – near-infrared spectroscopy; REDO – reoperation; STJ – sinotubular junction; TAAD – thoracic acute aortic dissection; TEE – transesophageal echocardiogram; TEM – type – entry – malperfusion; TTE – transthoracic echocardiogram; VSARR – valve-sparing aortic root replacement.

Introduction

Having a blood pressure of systolic/diastolic over 140/90 mmHg or more consistently can lead to the diagnosis of hypertension. It is estimated that worldwide 1,28 billion adults aged 30-79 have hypertension. Most of them are from low- or middle-income countries. Almost half of the hypertension population is not aware

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of their condition. They do not measure their blood pressure and probably do not visit their general practitioner unless they have a visible problem. The other almost half of patients (42%) are diagnosed and treated, but the third part of, around 12%, have a diagnosis but do not take their treatment due to different reasons like financial problems, refusal, medical system mistrust and other different causes. So, in conclusion, only 1 in 5 adults (21%) with hypertension have this condition under control [1].

Some risk factors for hypertension are described in the literature: older age, genetics, obesity or being overweight, sedentarism, high-salt diet and alcohol abuse. Usually, hypertension is asymptomatic, but sometimes patients describe headaches, blurred vision or chest pain. There are described hypertensive peaks when blood pressure is very high (around 200 mmHg systolic). Patients can feel this in different ways, such as severe headaches, chest pain, dizziness, difficulty breathing, nausea, vomiting, blurred vision or other vision changes, anxiety, confusion, buzzing in the ears, nosebleeds and abnormal heart rhythm [1].

The modifiable factors that can have an impact on high blood pressure consist of a healthy lifestyle. This includes eating a low salt, low saturated fat diet, rich in fruits and vegetables, losing weight (if necessary), exercising regularly and quitting tobacco.

Multiple complications are described in the literature, but one of the most important and one with the highest mortalities is aortic dissection (AD). It means that one or more tears appear in the aortic wall, separating the intima and media from the adventitia and forming another lumen, named the false lumen. Fortunately, this is an uncommon disease. Data shows that two-thirds of patients are men, and the average age is around 60. Young patients can also have aortic dissections, especially if they have genetic disorders that affect the aortic wall and/or valve. Seven genes were identified as more present in patients with AD: ACTA2, MYLK, PRKG1, SMAD3, TGFB2, TGFBR1 and TGFBR2 [2, 3, 4].

From review data until 2020, type A aortic disease (TAAD) incidence is 3/100 000 individuals/year and 1,6/100000 individuals/year for type B aortic dissection [5].

The most frequent symptoms described by patients presenting with AD are abrupt onset of severe pain in the chest, back, or abdomen, shortness of breath, pain in the arms or legs, weakness, or loss of consciousness [3].

A complete diagnosis usually starts with the suspicion of AD based on the onset of symptoms and clinical features. In the beginning, blood pressure measurement on both sides and listening to the heart and pulse measurements can indicate a high suspicion of AD. An electrocardiogram (ECG) may show complications from coronary malperfusion.

The most frequent diagnostic test includes computed tomography (CT), but also transesophageal

echocardiogram (TEE), and magnetic resonance imaging can be used. Transthoracic echocardiogram (TTE) can diagnose AD, but a CT scan should be done before surgery, if possible, in order to evaluate the extent of dissection, tears and malperfusion.

The Stanford classification use is very simple and defines TAAD as dissection involving the ascending aorta, type B involving only the descending aorta, and “non-A non-B” involving the aortic arch but not the ascending aorta [6]. Another more complete classification system that specifies Stanford type of dissection, entry and malperfusion (TEM) has evolved. Type includes the Stanford classification, and entry specifies the tear level, which can be “0” (not visible), “1” (ascending aorta), “2” (arch), or “3” (descending aorta). Another important feature in this classification tells us more about malperfusion, so there are 4 types: “0” (no malperfusion), “1” (coronary arterial), “2” (supra-aortic vessels), or “3” (visceral/renal or a lower extremity); a “+” sign is added if malperfusion is clinically evident or a “-” sign is added if malperfusion is a radiological finding. This is called TEM classification of aortic dissection, and it helps clinicians recognize each case faster and describe it more accurately [6].

It can be surprising and unpredictable for the onset, evolution and prognosis. Diagnosis can include myocardial infarction, which happens due to the rupture of one or both of the coronary ostia (usually the right coronary is involved), the false lumen can occlude the coronary ostia by covering it, or the thrombus within the false lumen can compress and occlude the coronary ostia. Pericardial effusion or tamponade can coexist if the adventitial rupture communicates with the pericardium. Stroke or lipotomy can occur as a result of aortic arch/cerebral vessels tear, dissection or vessel occlusion, intermittent or continuous. Retrograde dissection propagation can cause aortic valve regurgitation by prolapse of a commissure or by dilation of the sinotubular junction (STJ). Most type B aortic dissection complications are defined as malperfusion of different organs: spinal cord, gastrointestinal tract, kidneys, renal, or extremities [6].

All aortic dissection needs medical treatment as soon as possible and systolic blood pressure must be reduced to 100-120mmHg [6].

There are two options widely used and recommended for aortic root replacement in acute AD: valve-sparing aortic root replacement (VSARR) and composite aortic valve graft replacement (CAVGR). There is a debate on which is better or could provide better results, but multiple studies have similar results between the two procedures. Ultimately, it depends on each case and the experience and possibilities of each hospital [7].

Mortality depends on the patient’s general state, comorbidities and extent of the dissection at presentation, and malperfusion. It is classically described as 1%/hour since the onset of symptoms, so the emergent surgical treatment is well indicated [3].

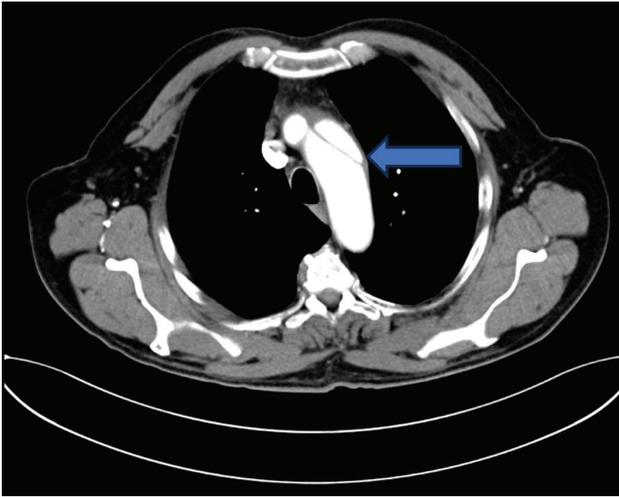


Figure 1. CT scan, Axial plane, aortic arch with visible aortic dissection, two circulated lumens at the level of proximal aortic arch (arrow).

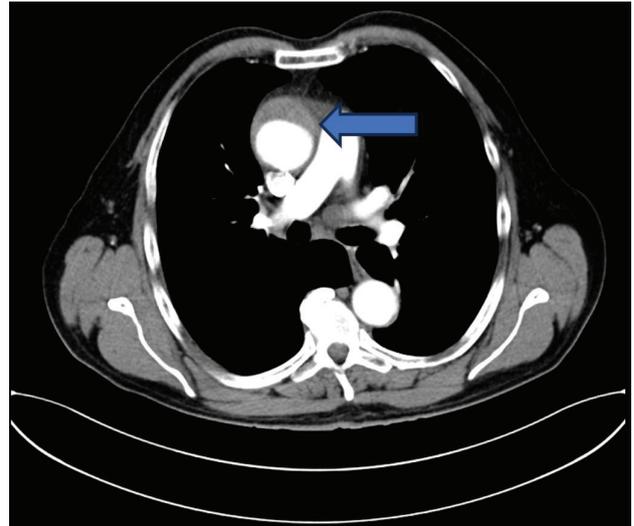


Figure 2. CT scan, Axial plane, ascending aorta with visible dissection and thrombosed false lumen (arrow).

Case report

We present a case of acute aortic dissection type A in a 58 years old male known for tobacco and alcohol abuse, presenting in the emergency department with severe headache, chest pain and unmeasurable blood pressure at home. The onset of symptoms started 36 hours before hospital arrival. He had echocardiography, which revealed an ascending aorta of 37mm, tricuspid aortic valve, mild aortic regurgitation with central jet, important concentric ventricular hypertrophy, preserved ventricular function, an echogenic image in the aortic arch suggesting aortic dissection at this level. On the computer

tomograph scan (CT), he was diagnosed with aortic dissection TEM A20. The false lumen was visible in the proximal part of the aortic arch and an ascending aorta hematoma, as seen in Figures 1–6. He was immediately sent to the operating room for surgical treatment, where the diagnosis was confirmed, as shown in Figure 7.

We cannulated the right femoral artery and right atrium for extracorporeal circulation and proceeded to fast deep hypothermia up to 18 degrees Celsius. After the cardioplegia, we reached the temperature and stopped the circulation to the body but maintained perfusion to the head with the antegrade flow on one carotid artery with monitored flow and pressure. We had near-infrared spectroscopy (NIRS)

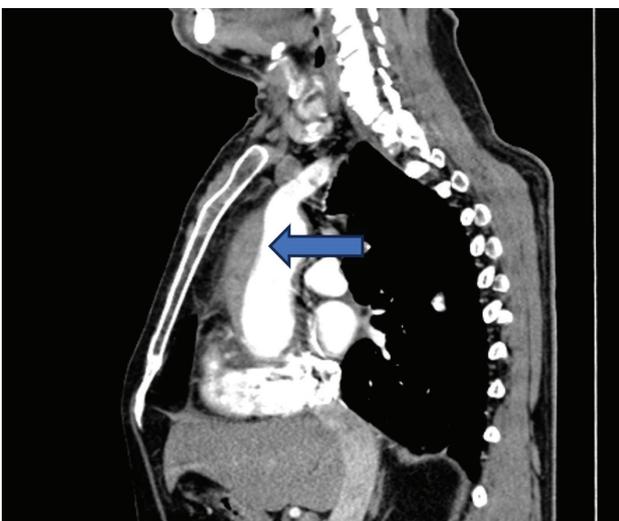


Figure 3. CT scan, Sagittal plane, ascending aorta with visible aortic dissection, thrombosed false lumen at the level of anterior wall (arrow).

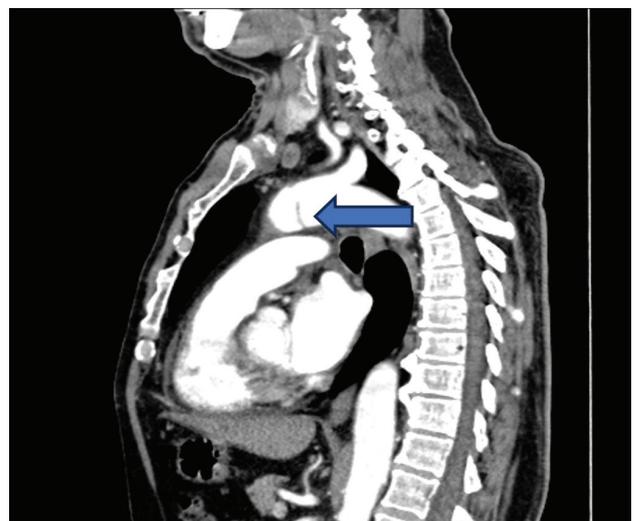


Figure 4. CT scan, Sagittal plane, aortic arch with visible aortic dissection, two circulated lumens at the level of proximal part (arrow).

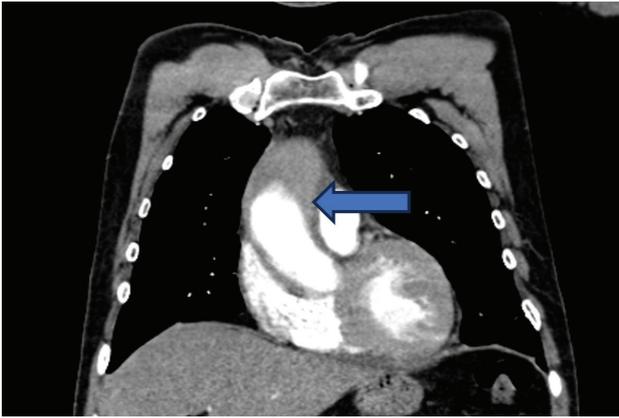


Figure 5. CT scan, Coronal plane, ascending aorta with visible aortic dissection, thrombosed false lumen at the level of anterior wall (arrow).

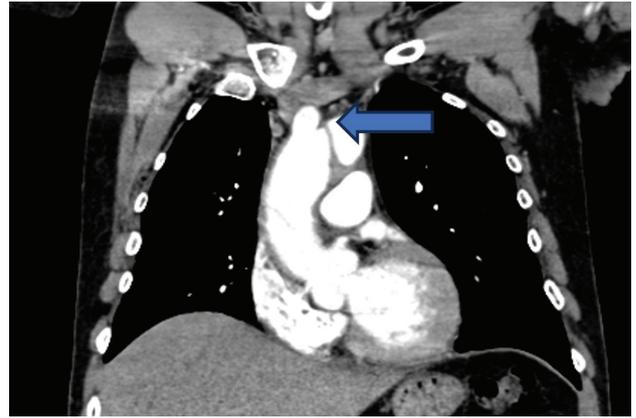


Figure 6. CT scan, Coronal plane, aortic arch with visible aortic dissection, two circulated lumens at the level of proximal part (arrow).

monitoring of the brain the whole procedure time with no incident. The ascending aorta was dissected and had a thrombosed false lumen. A tear was visible on the anterior part of the aortic arch, between the left common carotid artery and brachiocephalic arterial trunk, as seen in Figure 8. The distal aortic arch and descending aorta had no dissection, so we put a clamp in order to start rewarming and shorten the extracorporeal circulation time. We first replaced the ascending aorta from the sinotubular junction with a 28 mm vascular graft and then the aortic arch with reimplantation of the cerebral vessels patch into the prosthesis. After deairing the arch, a clamp was placed on the proximal part of the prosthesis.

Meanwhile, the two prostheses were sutured between them, followed by deairing the heart and removing the aortic clamp. The heart had a spontaneous sinus rhythm at 30 degrees Celsius. The circulatory arrest was 45 minutes, the aortic clamp was 117 minutes and extracorporeal circulation was 153 minutes.

The patient had an uneventful recovery, extubated 11 hours postoperatively and after three days in intensive care, he was transferred to the ward. After 7 days postoperatively, he was discharged.

Our institutional protocols for follow-up in cardiac surgery involve regular checkups at 1, 3, 6, and 12 months postoperatively and then yearly. Our patient missed his appointment at 1 month and reappeared

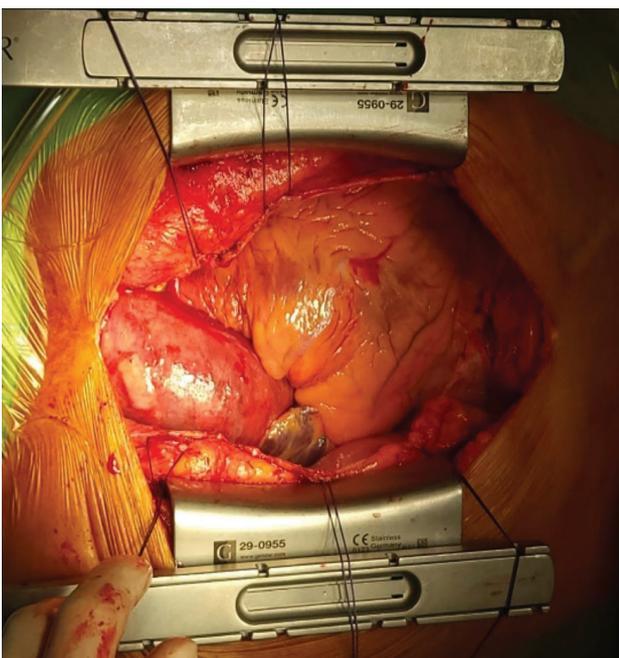


Figure 7. Intraoperative view – dissected ascending aorta.

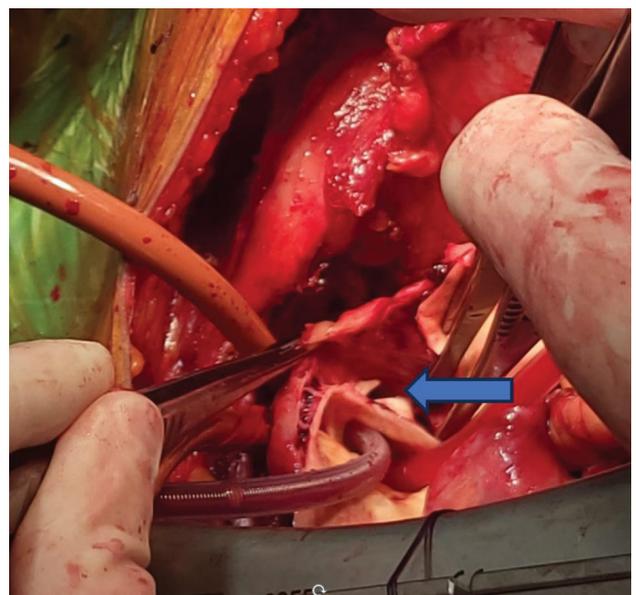


Figure 8. Intraoperative view – dissected aortic arch, visible tear between the brachiocephalic arterial trunk and left common carotid artery (arrow).

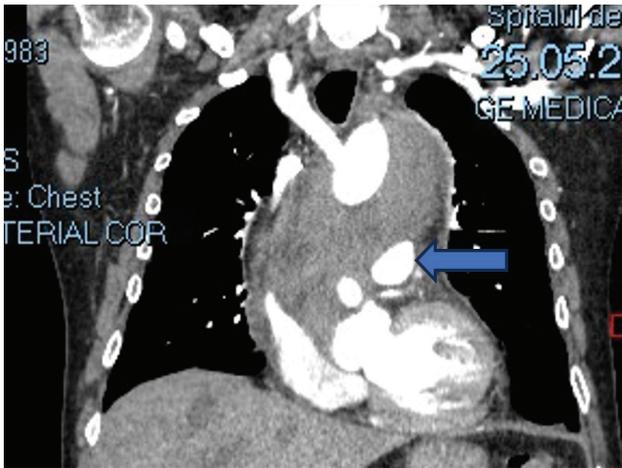


Figure 9. CT scan, Coronal plane – important ascending aorta hematoma and active bleeding visible above the left coronary artery (arrow).

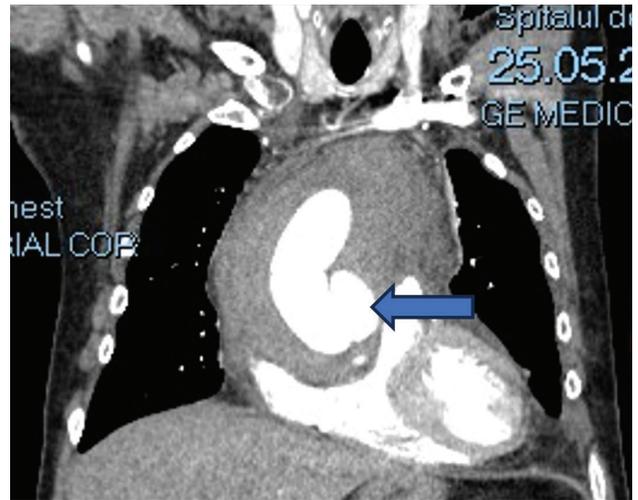


Figure 10. CT scan, Coronal plane – important ascending aorta hematoma and active bleeding visible (arrow).

at the emergency department at 1,5 months with shortness of breath and fatigue for the last one and a half weeks. He immediately had his blood tests, echocardiography and CT scan.

He had mild anemia, the echography showed an important hematoma and effusion around his aortic root with a diameter of 96 mm, minimal pericardial effusion, moderate left ventricle dysfunction (LVEF 45 %), basal inferior hypokinesia and mild aortic regurgitation. CT scan showed active bleeding from his aortic root, massive hematoma and blood around the ascending aortic prosthesis with a size of a maximum 44mm circumferential, and no aortic dissection in the aortic arch as seen in Figures 9-12.

The patient was immediately admitted for surgery.

Considering the REDO (reintervention) surgery, important ascending aortic prosthesis hematoma

and active bleeding from the root, we decided first to cannulate the patient, start deep hypothermia to 18 degrees Celsius and then REDO sternotomy. We cannulated the right axillary artery through a vascular prosthesis 8mm diameter and the right atrium through the right femoral vein. We had 18 degrees after 30 minutes. Meanwhile, we proceeded to sternotomy and cardiac structures dissection. When the temperature was reached, the heart stopped spontaneously. We entered the ascending aorta hematoma (Figure 13 and 14) and saw the bleeding in the aortic root, just proximal to the anastomosis at the level of the left-right commissure. Ascending aortic prosthesis was clamped and selective antegrade cardioplegia was given. The aortic arch was inspected, and after we confirmed everything was in order, we started rewarming the

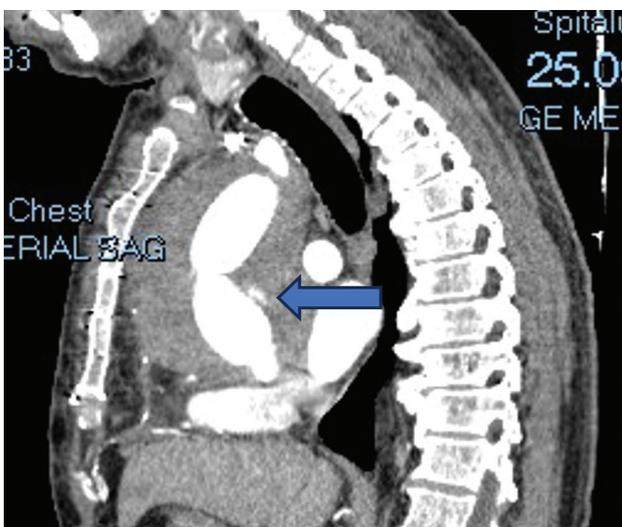


Figure 11. CT scan, Sagittal plane – important ascending aorta hematoma and active bleeding visible (arrow).



Figure 12. CT scan, Coronal plane – important ascending aorta hematoma and active bleeding visible between the ascending aorta and pulmonary artery (arrow).

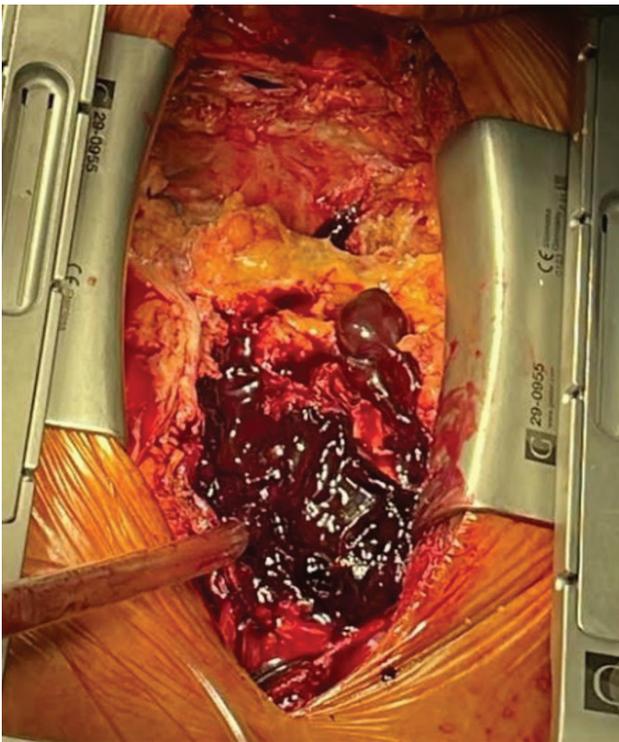


Figure 13. Intraoperative view - important ascending aorta hematoma.

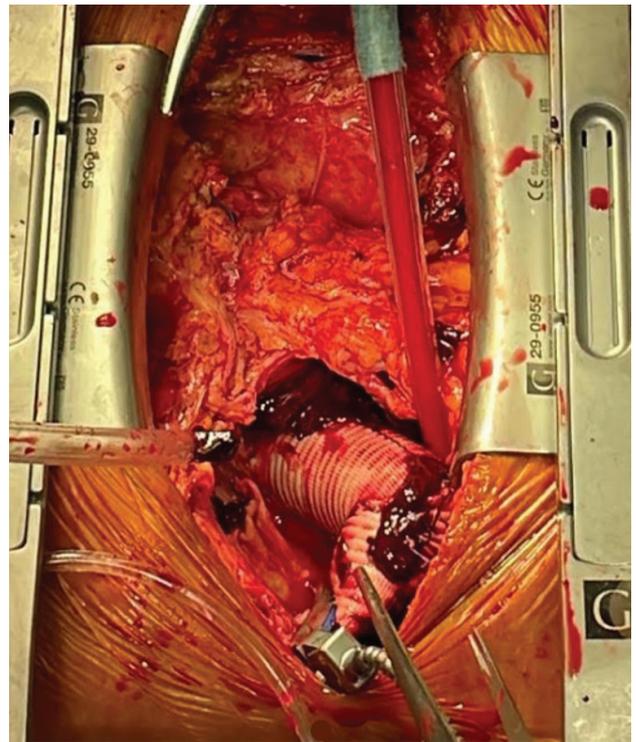


Figure 14. Intraoperative view - important ascending aorta hematoma.

patient to 36 degrees Celsius. We measured intraoperatively the aortic annulus, it was 25 mm, the cusps were normal, good coaptation. We proceeded with the Yacoub procedure, replaced the aortic sinuses with a 28-vascular prosthesis and reimplemented the coronary ostia in a standard fashion. We assessed the aortic cusps again, and no asymmetry was observed. Then we sutured the two vascular prostheses and con-

tinued with deairing the heart, and removed the aortic clamp. Sinus rhythm was spontaneously resumed. Intraoperative TEE showed minimal aortic regurgitation same moderate LV dysfunction. At the end of the operation, before transferring the patient to the intensive care ward, we had ventricular fibrillation. After one external electric shock, he regained sinus rhythm again. Considering his moderate ventricular

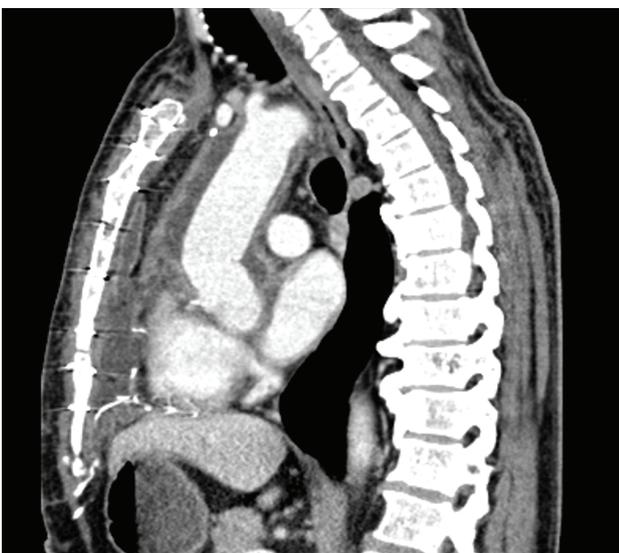


Figure 15. CT scan - sagittal view, reconstruction of the aortic root with visible right coronary.

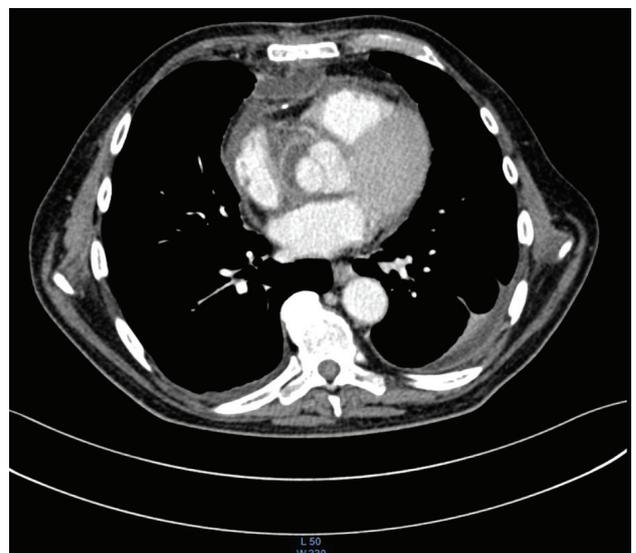


Figure 16. CT scan - axial view, reconstruction of the aortic root with visible right coronary artery.

dysfunction and inferior hypokinesia, we decided to do a coronary angiography. He had no coronary stenosis and their ostia were normal.

The patient had an uneventful recovery, extubated on day 2 postoperative, transferred to the ward on day 4 and discharged on day 14.

We performed a CT scan before the discharge to evaluate the repair and no false lumen was observed, as shown in Figures 15 and 16, aortic regurgitation remained minimal and ventricular function was preserved.

Discussion

Aortic dissection can have multiple causes, but the most frequent risk factor encountered is high blood pressure, most of the time diagnosed in the emergency department altogether with aortic dissection. More than half of patients diagnosed with aortic dissections also have hypertension. Unfortunately, as well as diabetes, it is not observed by patients, so they are not diagnosed on time and by the time of their diagnosis, they may have some complications.

Unfortunately, some patients are not compliant with the medical treatment or diet changes after the surgery, so they present with hypertension complications multiple times, even after surgery [8, 9].

For the second procedure, with the diagnosis of active root bleeding, minimal aortic regurgitation with central jet and normal aortic cusp structure, we thought that a VSARR procedure would be a good option. The two options are David's or Yacoub's procedures. The main difference between the two is that David's stabilizes the aortic annulus and could be a better option in patients with aortic annulus enlargement or connective tissue disorders where we expect a postoperative increase in the aortic annulus diameter over time. On the other hand, Yacoub's procedure is more physiologic for replacing the aortic sinuses and offers a better reconstruction of the aortic root, but could be an option only in patients with normal aortic annulus or it needs the use of an annulus stabilizer, such as a suture annuloplasty or an aortic annulus [10, 11, 12].

Conclusion

AD is an unpredictable disease with a high mortality. Blood pressure management is essential for cardiovascular risk patients to prevent associated complications. Especially for patients with AD treated successfully with surgery, it is essential to manage blood pressure so further complications can

be prevented. Recommendations are that systolic blood pressure is maintained at an upper limit of 120mmHg, but patient compliance to continuous medical treatment and regular follow-up can be difficult to accomplish.

Conflict of interests

The authors declare no conflict of interest.

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