Spontaneously Resolved Intramural Haematoma of the Thoracic Aorta: Case Report

Spontan Olarak Rezorbe Olan Torasik Aorta İntramural Hematomu

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Yazışma Adresi/Correspondence: Adem GÜLER, MD Gülhane Military Medical Academy, Department of Cardiac and Vascular Surgery, Ankara, TÜRKİYE/TURKEY drademguler@yahoo.com **ABSTRACT** In this case report we present a 46 year-old hypertensive male patient with a diagnosis of intramural hematoma of the descending thoracic aorta. He was admitted to emergency department with the complaints of both back pain and upper abdominal pain. Neither no specific positive findings on ECG and nor on direct X ray were observed. Following thoracic computerized tomography (CT) and transesophageal echocardiography intramural hematoma of descending thoracic aorta was confirmed. He was treated and followed with medical therapy and responded well to the treatment. After two years the intramural hematoma was spontaneously resolved on thoracic CT.

Key Words: Hematoma; aortic diseases; aorta, thoracic

ÖZET Bu yazımızda, inen torasik aortada intramural hematom tanısı konulan 46 yaşındaki hipertansif erkek hastayı sunduk. Hasta, ani başlangıçlı sırt ve üst abdominal ağrı yakınması ile acil servise başvurmuştu. Yapılan ilk değerlendirmede spesifik bir bulgu saptanmayan hastada, EKG ve direk grafilerde de anlamlı bir bulguya rastlanmadı. Bunun üzerine yapılan toraks tomografisi ve transözefageal ekokardiyografi ile inen torasik aortada intramural hematom olduğu saptandı. Medikal tedavi ile takip edilen hastanın yakınmaları kayboldu. İki yıl sonra yapılan toraks tomografisinde intramural hematomun spontan rezorbe olduğu gözlendi.

Anahtar Kelimeler: Hematom; aort hastalıkları; aort, torasik

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ortic intramural hematoma (AIH) is a type of aortic dissection having no intimal tear and caused by the rupture of vasa vasorum within the aortic wall. Although there is a localized wall thickness, there is no sign of intimal tear. It is not always possible to distinguish AIH from aortic dissection. This type of aortic dissection differs from the other types with having no connection between true and the false lumen. Natural course, incidence of mortality and morbidity and management of AIH is also totally different from aortic dissection. Differential diagnosis is easier with the use of advanced imaging methods. AIH can be diagnosed easily with transesophageal echocardiogarphy (TEE) and in doubtful cases CT and MRI can be useful to confirm the diagnosis.

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CASE REPORT

A 46-year-old male patient with a history of hypertension admitted to the emergency service with the complaints of sudden onset back and upper abdominal pain. First evaluation with telecardiography, electrocardiograhy and routine biochemical blood analysis revealed no specific finding. Also transthoracic echocardiography revealed no pathological finding except left ventricular hypertrophy that was associated with hypertension. But, on thoracic CT images aortic intramural hematoma that reflected high density images and aortic wall thickness were detected (Figure 1). Hematoma was beginning approximately 2 cm distal to subclavian artery and going 8 cm distal on the descending aorta. The widest diameter of the aorta with intramural hematoma was 36 mm. TEE was performed to distinguish aortic dissection from AIH. TEE showed aortic wall thickness but no intimal flap of dissection was seen (Figure 2).

As a result of the evaluation, patient was diagnosed as intramural hematoma of the descending thoracic aorta. After obtaining the informed consent from the patient, he was hospitalized and observed for a period of time. Within this period no complication came out, so medical treatment without surgical therapy was decided. During the hospitalization period he had complaints of angina pectoris. In order not to cause any dissection the patient was not thought suitable effort test and after consulting with cardiology department a coronary angiography was advised to exclude or detect any concomittant coronary artery disease. According to this, a coronary angiography was performed. It revelaed segmentary stenosis of right coronary artery (RCA). So, PTCA was performed to RCA and a stent was implanted. Patient was put on an antiplatelet treatment regim with clopidoprel 75 mg once dialy and this treatment regimen still continues until now on for two years. At the end of a follow up period of 2 weeks, a control TEE was performed. No increase of aortic wall thickness and no formation of intimal flap were seen. So, the patient was discharged. Control evaluation was performed 3 months later and he had no complaints and no increase of aortic wall thickness was detected. At two year follow up, aortic wall seemed completely normal at the control CT (Figure 3).

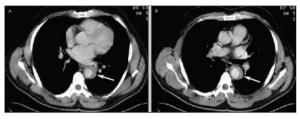


FIGURE 1: CT image showing haematoma as a high density reflection and wall thickness.

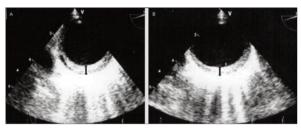


FIGURE 2: TEE examination showing the increase of the thickness of the aortic wall.

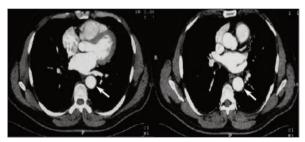


FIGURE 3: CT images showing completely resolved intramural hematoma at the two year follow-up.

DISCUSSION

AIH is a variant form of aortic dissection and can lead to aortic rupture.³ This syndrome is known as a form of acute aortic syndromes that has no intimomedial tear. Its incidence was reported as 12.8% among acute aortic syndromes.⁴ Hypertension is both the most significant known risk factor. However, elastic tissue disorders are the other risk factors.⁵ Although a lot of etiologic factors play role in the progression of this disease, rupture of the vaso vasorums within the aortic wall has been supposed as a basic pathology.¹ There are some literatures report that rupture of the aortic atherosclerotic plaques and its role as a intimal tear is another etiologic factor playing role in the formation of AIH.^{4,5} Nevertheless, the triggering mechanism re-

mains unclear.⁴ Although no intimal tear is pathogonomic for this disease, very small group of patients may have an inflow tear but, these patients have no outflow tear. So, a flow within the aortic wall is not in question.⁵

Two forms of disease has been determined one of which is traumatic type with good prognosis while the other one is non-traumatic type with poor prognosis. The latter is supposed to be the precursor of aortic dissection.⁶ Similar to thoracic aortic dissections, AIHs affect ascending aorta, arcus aorta or both (Type-A) or descending aorta (Type-B).² Likely to acute aortic dissection, the clearest symptom of the disease is chest pain.5 Typically, this pain does not respond to nitroglycerine and does not expand on extremities or chin.4 This is important in distinguishing it from acute coronary syndromes, but our case responded to nitroglycerine. We think that this is more related with the lesion in the RCA than the course of the disease. So, it should be kept in mind that acute coronary syndrome may accompany to AIH and further evaluations should be performed especially for the patients under risk factors. Presence of abdominal pain should bring to mind the disease of descending aorta.5

The disease is diagnosed via an imaging method showing the intimomedial tear or intimal separation. CT imaging figures out fresh hematoma as a high density image on aortic wall, while MR imaging may present some information about the age of the hematoma according to the methemoglobin within the hematoma.4 However, aortography has a limited diagnostic value.6 Although there are plenty of imaging methods, TEE has a very significant value in making the diagnosis of AIH.1 Intramural hematoma is seen as a reflection of blood that caused thrombus formation in the area between intima and adventitia.4 To diagnose AIH with TEE; there should be no membrane of dissection, doppler examination should reveal no flow relationship between false and true lumens and the wall thickness in the area of hematoma should be greater than 7 mm.^{1,4} Although presence of atherosclerotic plaques and transmural thickness can be confused with each other, repetitive examinations with TEE shows no difference in the transmural thickness and this is in favor of AIH.⁴

The most significant and still debatable subject about AIH is which patients should be treated with medical therapy and which ones with surgical treatment. Because AIH affecting ascending aorta has a high complication rate and has poor prognosis, surgical treatment is recommended at the time when it is diagnosed, while medical therapy and close follow up is recommended for AIH affecting descending aorta.1 In medical treatment control of hypertension and sedation are satisfactory. High risky patients for rupture of AIH are patient with penetrated aortic ulcer, uncontrolled hypertension and increase in the diameter of the hematoma.³ We followed our case with antihypertensive therapy and antiaggregating therapy due to stent implantation. Although medication antiagregating treatment may increase the diameter of hematoma, it was obligatory in our case to use such type of medicine in order to provide patency of the stent.

In AIH affecting descending aorta, if there is a pain not respond to medical treatment, hematoma continuing to expand or there are complications like dilatation, and pericardial effusion; surgical intervention should be the mainstay of the treatment in order to prevent the development of dissection or rupture. In these indications, as an alternative of surgery endovascular treatment modalities can be used at last decades. However, it requires further multicenter studies and large group of patients, and it cannot be recommended for routinely use at this time, except complications mentioned below.

As a conclusion, in cases with AIH, concomittant coronary artery stenosis can be observed. Although it is a risk to perform coronary angiography to delienate any coronary stenosis, it is the best option to perform this technique since it provides a chance to treat the stenosis in the same time without having time loss. Antiagregating medication may cause progression of the hematoma, but as in our case with a close follow-up period this risk may be undertaken. Finally this case presentation is a good example of medically resolved aortic intramural hematoma.

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