HEMODINAMICALLY SIGNIFICANT EXTRINSIC COMPRESSION OF THE LEFT ATRIUM BY NONANEURYSMATIC SEGMENT OF DESCENDING AORTA

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ÖZET

Anevrizmatik Olmayan İnen Aorta Segmenti Tarafından Sol Atriyumun Hemodinamik Olarak Önemli Kompresyonu

Sol atriyumun komşu yapılar ya da sindirim organları tarafından kompresyonu, klinik olarak önemli hemodinamik bozulmaya yol açabilir. Ekokardiyografinin genellikle klinisyenlere çoğu olguda ayırıcı tanı yapma olanağı sağlamasına rağmen tomografi ve manyetik rezonans görüntüleme kesin tanıda oldukça yardımcı araçlardır. Bu yazıda inen aortanın anevrizmatik olmayan bir segmentinin sol atriyuma bası yaparak atriyal fibrilasyon, hemodinamik bozulma ve pulmoner konjesyon oluşturduğu bir olgu sunulmaktadır.

Anahtar Kelimeler: Sol Atriyal Bası, İnen Aorta, Bilgisayarlı Tomografi, Ekokardiyografi.

SUMMARY

Left atrial compression by adjacent structures or digestive organs can be a clinically significant cause of hemodynamic compromise. Although echocardiography enables the clinicians to make a differential diagnosis successfully in most cases, computerized tomography or magnetic resonance imaging can provide valuable and complementary information to make the diagnosis clear. We present a case of non-aneurysmatic segment of descending aorta compressing the left atrium that could cause atrial fibrillation, hemodynamic compromise and pulmonary congestion.

Key Words: Left Atrial Compression; Descending Aorta; Computerized Tomography; Echocardiography.

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INTRODUCTION

An unusual source of hemodynamic compromise is extrinsic left atrial compression (1) that can be caused by mediastinal structures such as bronchogenic cysts (2), lymphoma (3), diaphragmatic hernia (4), and gastric structures (5, 6), especially stomach and esophagus. However, few cases with aortic aneurysm (7,8) and an infrequent and fatal form of type B aortic dissection (9,10) causing hemodynamically significant extrinsic left atrial compression were reported. Echocardiography enables the clinicians to make the differential diagnosis successfully in most cases. In this report, we describe a case of extrinsic left atrial compression by the calcified and displaced non-aneurysmatic segment of descending aorta that could cause paroxysmal atrial fibrillation, hemodynamic instability, and pulmonary congestion.

CASE REPORT

A 75-year-old woman was referred to our Intensive Care Unit for severe palpitation, which started 2 hours ago. Also, the patient was suffering from the dyspnea following the onset of palpitation. In her detailed past history, the palpitation has a paroxysmal characteristic and these paroxysmal attacks started to happen more frequently in recent weeks. The patient had had a history of hypertension for 20 years but no history of cardiovascular disease. She was on angiotensin converting enzyme inhibitor for hypertension. On admission, she was dyspneic and diaphoretic. Her systolic blood pressure was 80 mmHg and heart rate was 130 beats/min irregular. There was a systolic murmur with a grade of 2/6 on the left sternal border. Chest examination revealed rales over the lower two thirds of both lung fields. Her resting electrocardiography showed atrial fibrillation with a high ventricular rate. Heparin infusion fol-



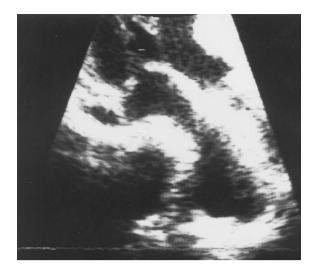


Figure 1: Transthoracic echocardiographic apical four chamber (a) and enlarged view (b) showing compression of the left atrium by a cystic mass.

lowing bolus injection was started for atrial fibrillation with a control of activated coagulation time. Echocardiographic examination revealed that a cystic mass, which could be aortic segment, compressing the left atrium externally and impairing the ventricular filling (Figure 1). Other echocardiographic findings were as follows: left ventricle enlargement due to severe aortic insufficiency, mild mitral insufficiency, and fibrotic mitral leaflets. Mitral valve area was within normal limits. Systolic function was lower than normal limits (EF=44%). Atrial fibrillation converted to sinus rhythm with a direct current electrical cardioversion of 200J. Fluid and dobutamine therapy was started. Hemodynamic stability was achieved on the basis of sinus rhythm and dobutamine infusion. Chest X-ray revealed a calcified and angulated aorta to the right side of thorax (Figure 2). Computerized tomography (CT) of the aorta showed a nonaneurysmatic segment of the descending aorta causing a compression of the left atrium (Figure 3). No dissection of the aorta or mass including gastric structures were observed in the CT. After clinical stabilization of the patient's status, we tried to perform transesophageal echocardiography. Unfortunately, the patient could not tolerate the procedure. Because we could not offer any surgical option for the patient, medical therapy had been the choice of therapy. The palpitation attacks were controlled with the drug therapy including amiodarone (200 mg a day). The patient has become well with the medical therapy.

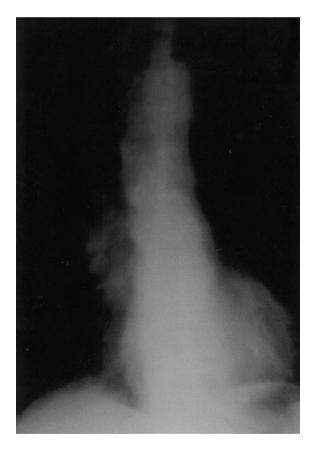


Figure 2: Chest X-ray demonstrates a calcified and angulated nonaneurysmatic segment of descending aorta to the right side of the patient's thorax.

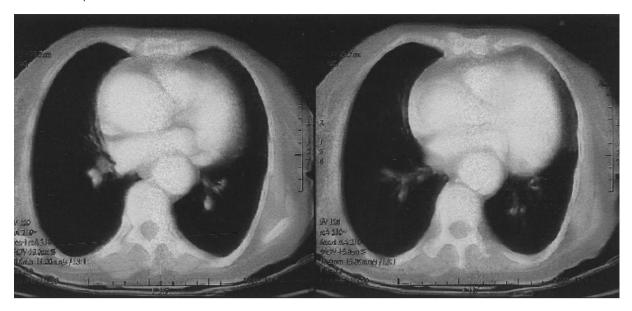


Figure 3: Computerized tomography showing the descending aorta compressing the left atrium.

DISCUSSION

Because the left atrium is highly susceptible to compression, extrinsic left atrial compression by the displacement of any organ can cause tachycardia, hypotension, and hypoxia, which are known as the findings of hemodynamic compromise. Left atrial compression can result in an obstruction of left atrial inflow, decreased left ventricular filling, and increased pulmonary venous pressure, leading to pulmonary edema and decreased cardiac output (5,10). Also, atrial arrhythmias, especially atrial fibrillation, can occur due to this kind of compression (2).

D'Cruz et al. (1) classified the relation between mediastinal masses and the heart in three categories: proximity (a contiguous or adjacent structure without chamber deformation), encroachment (distortion or partial displacement of one or more cardiac chambers by a mediastinal mass with no hemodynamic effect), and compression with hemodynamic compromise. Left atrial compression can resemble clinical scenarios caused by acute myocardial infarction complicated with cardiogenic shock, pericardial tamponade, and acute pulmonary embolus (2-5). All hemodynamically significant clinical scenarios can be easily differentiated by using transthoracic echocardiography, the test of choice for diagnosing left atrial compression (5,6,8). However, in the cases, which cannot be explained by echocardiography, CT and/or magnetic resonant imaging can provide complementary imaging to define the relation between the mediastinal masses and the heart,

especially left atrium. In the present case, echocardiography showed clearly the left atrial compression by the descending aorta. CT made it clearly possible to diagnose the descending aorta compressing the left atrium. In most of reported cases, surgical therapeutic options resulted in normal hemodynamic function. However, surgery could result in relief in cases with left atrial compression caused by the hematoma from the ruptured dissection complicated by pulmonary edema (9,10). In our case, we could not offer any surgical option, and the patient had been included in the follow-up program.

In conclusion, the clinical features in our patient were due to extrinsic left atrial compression caused by the calcified and angulated nonaneurysmatic segment of descending aorta. This type of shape of aorta should be considered in the differential diagnosis of hemodynamic compromise.

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