Huge Non-ruptured True Saccular Aneurysm of the Ascending Aorta

Dev Rüptüre Olmamış Çıkan Aortun Gerçek Sakküler Anevrizması

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ABSTRACT

Aortic aneurysm (AA), a disease characterized by dilatation of all aortic segments, causes fatal cardiovascular complications. Aortic aneurysms are usually related to hypertension or atherosclerosis. Generally, an AA is fusiform shaped, while the saccular type of AA is very rare. The risk of rupture and dissection in AA dramatically rises with increasing diameter. Risk of rupture is particularly higher in the female gender, Marfan syndrome and the presence of additional vascular involvement. In this case report, an asymptomatic, huge, non-ruptured, true saccular AA of an elderly female patient was presented. (Gazi Med J 2012; 23: 151-3)

Key Words: Aortic aneurysm, ascendant aorta, saccular, computed tomography, aortography

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ÖZET

Aort anevrizması (AA) ölümcül kardiyovasküler komplikasyonlara yol açan, aortun dilatasyonu ile karakterize, tüm aort segmentlerinin katıldığı bir hastalıktır. Aort anevizmaları genellikle hipertansiyon ve ateroskleroz ile ilişkilidir. Genellikle AA'ları fusiform şekillidir. AA'nın sakküler tipi nadiren görülür. AA'ında rüptür ve diseksiyon riski çap artışı ile dramatik bir şekilde artar. Rüptür riski kadınlarda, Marfan sendromlularda ve ek vasküler tutulumun varlığında özellikle daha yüksektir. Bu vaka sunumunda yaşlı bir kadın hastada asemptomatik, ileri boyutlarda ve rüptüre olmamış çıkan aortun gerçek sakküler anevrizması sunuldu. (Gazi Med J 2012; 23: 151-3)

Anahtar Sözcükler: Aort anevrizması, çıkan aort, sakküler, bilgisayarlı tomografi, aortografi

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INTRODUCTION	Fusiform aneurysm is the most common form and h a smooth uniform shape, with symmetrical dilatatic

Aortic aneurysm (AA) is characterized by permanent, localized, pathological dilatation of the aortic lumen. There are two morphological forms of true aortic aneurysm defined as fusiform and saccular type. Fusiform aneurysm is the most common form and has a smooth uniform shape, with symmetrical dilatation involving the full circumference of the aortic wall. In contrast, saccular aneurysms are very rare and have a more localized dilatation that appears as an outpouching of only a portion of the aortic wall.

Address for Correspondence / Yazışma Adresi: Dr. Sinan Altan Kocaman, Department of Cardiology, Rize Training and Research Hospital, Rize, Turkey Phone: +90 464 213 04 91 E-mail: sinanaltan@gmail.com

©Telif Hakkı 2012 Gazi Üniversitesi Tıp Fakültesi - Makale metnine www.medicaljournal.gazi.edu.tr web sayfasından ulaşılabilir. ©Copyright 2012 by Gazi University Medical Faculty - Available on-line at www.medicaljournal.gazi.edu.tr doi:10.5152/gmj.2012.35 If the aneurysm diameter is less than 5 cm, the risk of rupture and dissection is only 2% per year in AA; in diameters between 5 to 5.9 cm, rupture risk slightly increases (3% per year), but if the diameter is 6 cm or more, the risk increases dramatically (7% per year). Risk of rupture is particularly higher in females, Marfan syndrome and presence of additional vascular involvement (1).

In this case presentation, we aimed to demonstrate a non-ruptured, asymptomatic, true saccular AA of gigantic size in an elderly female patient. Although larger aneurysms are prone to rupture, we hereby present an interesting asymptomatic patient with a huge aneurysm seen on aortography.

CASE REPORT

A 76-year-old woman with the symptom of chest pain was admitted to the emergency department of Rize Education and Research Hospital in Rize, Turkey. She had a history of hypertension without previous coronary artery disease. On physical examination, her blood pressure was 160/90 mmHg, heart rate was regular at 80 beats/min. Except for a 2/6 grade mild apical systolic and mild-moderate diastolic murmur, her cardiac findings were normal. The lung fields were clear. Her ECG revealed a 1 mm ST-segment depression of the anterior leads (V1-V4) and left ventricular hypertrophy (LVH) voltage criteria.

On admission, the patient's creatinine kinase level was 170 U/L with an MB fraction of 23 U/L and the troponin T level was minimally increased. The patient was admitted to the coronary care unit with the diagnosis of acute coronary syndrome. After initial antiaggregant, anticoagulant and nitroglycerine treatment, her symptoms resolved.

Echocardiography revealed severe aortic insufficiency, normal left ventricular systolic function and slightly increased end-diastolic and end-systolic diameters. The diameter of the ascending aorta was 5.3 cm at the aortic annulus, left ventricle end systolic diameter was 4.1 cm and left ventricle end diastolic diameter was 5.9 cm.

The patient underwent coronary angiography and aortography which demonstrated a *high-heeled shoe* shaped saccular aneurysm of the ascending aorta (Figure 1). The diameter of the aneurysm measured by aortography was 8.3 cm. In addition, severe aortic insufficiency was confirmed by aortography. We failed to engage the coronary ostia and could not perform selective coronary angiography because of peripheral arterial tortuosity and extreme dilatation of the ascending aorta despite having both femoral and brachial access.

The computed tomography scan was consistent with aortography and confirmed the diagnosis and diameter of the large non-ruptured true saccular AA (Figure 2). The patient was then transferred to the vascular surgery department for aneurysm and aortic valve replacement surgery.

DISCUSSION

Aortic aneurysm is defined as the pathological dilatation of the aortic lumen involving one or several segments. Aneurysms are usually described according to location, size, and morphological appearance. Two morphological forms of true aortic aneurysm exist: fusiform and saccular shape. False aneurysms or pseudo-aneurysms that lack three-layer arterial vessel wall involvement are not actual aneurysms, and may result from a contained rupture of the aortic wall.

Aneurysms of the thoracic aorta most often result from cystic medial degeneration or cystic medial necrosis, which is the leading pathophysiological cause of aortic aneurysm development. Especially fusiform aneurysms occur due to elastic fiber degeneration with prominent cystic areas in the media. In contrast, this mechanism may not be relevant for true saccular aneurysms of the aorta.

True AA, which could be seen at all segments of the aorta, frequently tend to be confined to the infra-renal abdominal aorta (1-3). Saccular aortic aneurysms are less common than fusiform aneurysms of the aorta. Saccular aortic aneurysms occur predominantly due to trauma, active or healed focal aortic infections, penetrating aortic ulcers, after open heart surgery as well as atherosclerotic disease, cystic medial degeneration and syphilis (4, 5). Although saccular aneurysm may also develop after aortic surgery, this is rare after coronary artery by-pass grafting operation (6). In symptomatic saccular aneurysm, surgery should be considered independent of the aneurysm diameter (7, 8).

Fusiform aneurysms often involve the aortic root and thus may consequently result in aortic regurgitation. The term annuloaortic ectasia is often used to describe this condition. The presence of an aortic aneurysm may be an indicator of more diffuse aortic disease which is related to increased cardiovascular morbidity and mortality. Up to 13% of patients with aortic aneurysm might have multiple aneurysms, and 25%-28% of patients with thoracic aortic aneurysms might have concomitant abdominal aortic aneurysms. For this reason, a patient with an aortic aneurysm should undergo examination for the presence of other aneurysms in all segments of the aorta (9).

Although aortic aneurysms are generally asymptomatic, symptoms may occur due to either a vascular consequence of the aneurysm or a local mass effect. Aneurysms of the arch or descending aorta may compress the trachea or main stem bronchus and produce wheezing, cough, dyspnea, hemoptysis or recurrent pneumo-



Figure 1. On aortography, aortic root (white arrow) and huge nonruptured true saccular aneurysm of the ascending aorta with highheeled shoe shaped appearance was seen (red arrow). The diameter of aneurysm was measured as 8.3 cm



Figure 2. Computed tomography was consistent with aortography and confirmed the diagnosis and diameter of the large non-ruptured true saccular AscAA (white arrow)

nitis. Compression of the esophagus can produce dysphagia, and compression of the recurrent laryngeal nerve can cause hoarseness.

Chest pain or back pain occurs in 25% of non-dissecting aneurysms and results from direct compression of other intra-thoracic structures or from erosion into adjacent bone. Generally, such pain is permanent, deep, boring and sometimes severe.

The most valuable diagnostic tools for diagnosis of aortic aneurysm are chest radiograph, transthoracic echocardiography (TTE), transesophageal echocardiography (TEE), computed tomography (CT), magnetic resonance imaging (MRI) and aortography. TTE is an excellent modality for imaging of the aortic root and proximal ascending aorta, but it cannot visualize other segments of the aorta. TEE can image almost the entire thoracic aorta, as it is a semi-invasive procedure, CT and MRI are usually the preferred imaging techniques for evaluation of non-dissecting thoracic aneurysms. Coronary angiography is often useful for revealing patency of the coronary arteries and aortography would be helpful for evaluation of patients with clinical suspicion of an aortic aneurysm (9).

The goal of treating aortic aneurysms is to prolong life by preventing rupture. At the present time, medical or surgical treatment for AA is decided according to aneurysm size and dilatation rate of the aorta at follow up routine measurements. It is a reality that mortality rate increases with delay of vascular surgery in patients with increased rupture risk.

In some exceptional cases, as in our case, a true saccular aneurysm with huge dimensions can be asymptomatic clinically. An aneurysm with this dimension has a dissection and/or rupture risk of 7% per year. When saccular aneurysms are not treated surgically, rupture into the mediastinum, pleural space, esophagus or trachea does occur within 5 years which results in death in 80% of cases due to uncontrolled hemorrhage and rapid circulatory collapse (1). Therefore, clinical suspicion is a prerequisite of diagnosis for this lethal disease.

CONCLUSION

Routine diagnostic tests may overlook large sized saccular aortic aneurysms. For this reason, in patients with a clinical suspicion of aortic aneurysm, advanced diagnostic tests such as echocardiography, contrast computed tomography, magnetic resonance imaging and aortography should be performed.

Conflict of Interest

No conflict of interest is declared by the authors.

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