

The importance of bedside echocardiographic assessment in emergency admissions with probable acute coronary syndromes: A case of an unruptured giant ascending aorta aneurysm

Olası akut koroner sendromlu acil başvurularda yatak başı ekokardiyografik değerlendirmenin önemi: Yırtılmamış çıkan dev aort anevrizması olgusu

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Summary– Symptomatic aortic aneurysms can manifest in different clinical settings, such as acute coronary syndrome (ACS), acute heart failure, a shock that is mostly due to the complications related to dissection or rupture of the aneurysm. In these clinical settings, the diagnosis can be established with the help of medical history, physical examination, and promptly performed imaging tests. However, the diagnosis of an asymptomatic aortic aneurysm is usually incidental. Thus, it is very rare to find a case of ACS with intact aortic aneurysm and without obstructive coronary artery disease. In this paper, we report a successfully treated male patient aged 41 years with intact ascending aortic aneurysm who presented with ACS and was diagnosed with the help of bedside echocardiographic assessment.

Özet– Semptomatik aort anevrizmaları, akut koroner sendrom (AKS), akut kalp yetmezliği, çoğunlukla anevrizmanın diseksiyonu veya rüptürü ile ilgili komplikasyonlara bağlı bir şok gibi farklı klinik durumlarda ortaya çıkabilir. Bu klinik durumlarda tanı, tıbbi geçmiş, fizik muayene ve anında yapılan görüntüleme testleri yardımıyla konulabilir. Ancak, asemptomatik aort anevrizmasının teşhisi genellikle rastlantısalıdır. Bu nedenle, intakt aort anevrizması olan ve obstrüktif koroner arter hastalığı olmayan bir AKS vakası bulmak çok nadirdir. Bu yazıda, AKS ile başvuran ve yatak başı ekokardiyografik değerlendirme yardımı ile teşhis edilen, intakt çıkan aort anevrizması olan, başarılı bir şekilde tedavi edilmiş 41 yaşındaki bir erkek hasta sunulmaktadır.

Aortic aneurysm is a well-known vascular pathology and the second most frequent disease of the aorta after atherosclerosis.^[1] Most of the time, patients with aortic aneurysm are asymptomatic and are diagnosed incidentally during investigative imaging for other reasons. Chest pain, complaints related to the compression of surrounding structures, aortic murmur, and complications such as embolism, dissection, or rupture will be the clinical presentation in case of symptomatic aortic aneurysm.^[2–4]

Acute coronary syndrome (ACS) related to aortic aneurysm complicated with dissection has been reported in the literature.^[5,6] In addition, there are case reports of ACS due to mechanical compression of the aneurysmal section of the aorta on coronary arteries.^[7,8] However, ACS with intact aortic aneurysm and

without obstructive coronary artery disease (CAD) has not been probably reported. In this report, we present a successfully treated male patient aged 41 years with intact ascending aortic aneurysm who presented with ACS and was diagnosed with the help of bedside echocardiographic assessment.

Abbreviations:

¹⁸ F-FDG	Positron emission tomography
PET/CT	with 2-deoxy-2-[fluorine-18] fluoro-D-glucose integrated with computed tomography
ACS	Acute coronary syndrome
AI	Aortic insufficiency
ANA	Antinuclear antibody
ANCA	Antineutrophil cytoplasmic antibody
CAD	Coronary artery disease
CRP	C-reactive protein
CTCA	Computed tomography coronary angiography
ECG	Electrocardiography
LVEF	Left ventricular ejection fraction
MI	Myocardial infarction
POCUS	Point-of-care ultrasonography

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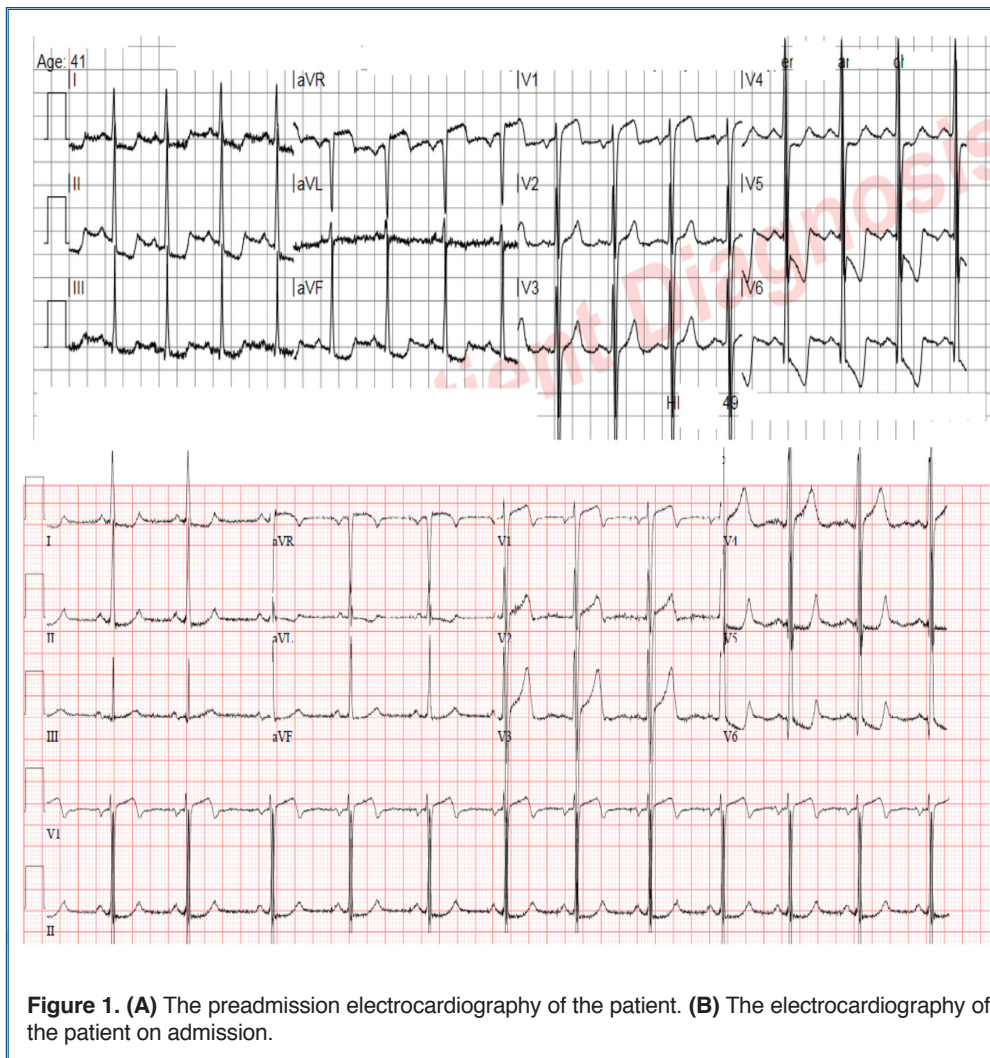


Figure 1. (A) The preadmission electrocardiography of the patient. **(B)** The electrocardiography of the patient on admission.

CASE REPORT

A male patient aged 41 years without past medical history was brought to the Emergency Department owing to a 1-hour duration of chest pain radiating toward the neck, which responded to sublingual 0.4 mg glyceryl trinitrate with a prenitrate hotline electrocardiography (ECG) showing ST depression over I, II, III, aVL, and V4-6 and 2-mm ST elevation over aVR and V1 (Fig. 1A).

On arrival, he had mild chest pain with significantly improved ST-segment changes (Fig. 1B) with left ventricular hypertrophy findings. Physical examination showed blood pressure of 162/53 mm Hg without significant difference between both arms; heart rate of 106 bpm with oxygen saturation of 98% at room air; and a mild, early diastolic murmur over the aortic region with widened, bounding peripheral pulses.

Bedside point-of-care ultrasonography (POCUS) showed mildly reduced left ventricular ejection fraction (LVEF) with a giant aneurysm confined to the ascending aorta along with significant aortic insufficiency (AI) but without regional wall motion abnormality. Chest X-ray showed widened mediastinum (Fig. 2). An urgent detailed echocardiographic examination revealed markedly dilated left ventricle with severe eccentric hypertrophy (LVEF of 42%, left ventricular end-diastolic diameter of 72 mm) and severe AI with tricuspid valve morphology (regurgitant velocity of 22.6 cm/s, regurgitant volume of 91.9 mL, and effective regurgitant orifice area of 0.85 cm²) along with a marked dilatation of the aortic annulus, aortic root, and ascending aorta (3.6 cm, 5.2 cm, and 6.4 cm, respectively). In laboratory findings, high-sensitivity troponin T levels were mildly elevated (19.7 ng/L on admission, 44.4 ng/L at a peak level after 4 hours, and 27.1 ng/L

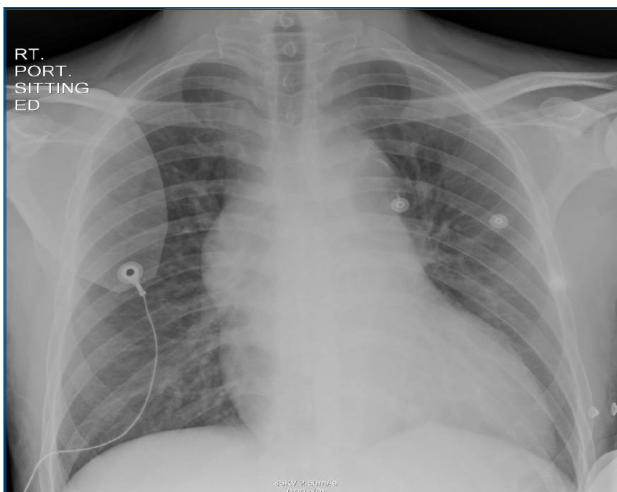


Figure 2. Portable anterior-posterior chest X-ray showing the widening of the upper mediastinum.



Figure 3. Axial computed tomography scan showing ascending aorta aneurysm.

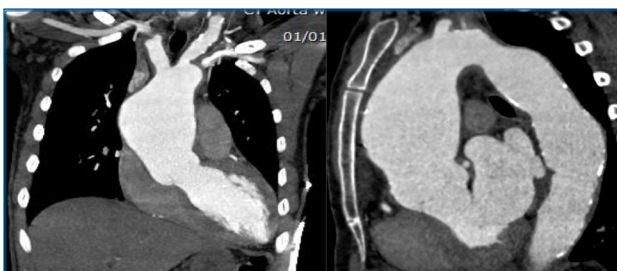


Figure 4. Coronal and sagittal computed tomography views showing ascending aorta aneurysm

after 24 hours from the arrival under normal renal function). The patient had a total cholesterol level of 4.6 mmol/L, low-density lipoprotein level of 2.8 mmol/L, triglyceride level of 1.4 mmol/L, and high-density lipoprotein level of 1.2 mmol/L. Serum C-reactive protein (CRP) level was <5 mg/L on admission.

Computed tomographic aortography confirmed the ascending aortic aneurysm of 7.5x7x9 cm in anterior-posterior, transverse, and caudocranial dimensions, respectively (Figs. 3 and 4) without evidence of aortic dissection. Computed tomography coronary angiography (CTCA) revealed zero coronary calcium without evidence of significant CAD (Fig. 5).

In addition, a nuclear study was performed to rule out vasculitis as a reason for the aneurysm. For this purpose, whole-body positron emission tomography with 2-deoxy-2-[fluorine-18] fluoro-D-glucose integrated with computed tomography (^{18}F -FDG PET/CT) was performed. There was no sign of active large vessel vasculitis on ^{18}F -FDG PET/CT. Test results of serum antineutrophil cytoplasmic antibodies (ANCA), antinuclear antibody (ANA), and *Treponema pallidum* antibody of the patient were negative. Doppler ultrasonography of the common carotid arteries showed a marked diffuse irregular increase in intima media thickness, with extensive irregular intima media calcification noted on both carotid arteries, which was causing a maximum luminal stenosis of 74% on the right and 79% on the left. Magnetic resonance angiography of both carotid arteries showed fusiform dilatation and subtle irregularities of the walls of the carotid arteries on both sides, which can also be noticed partially on computed tomographic aortography image (Fig. 4).

The patient remained clinically stable throughout the hospital course. Bentall procedure with a 29-mm mechanical valve was performed. Pathology report of the dissected material confirmed aortic root with atherosclerotic changes and degenerative valve disease. The patient was discharged safely on oral metoprolol and warfarin.

DISCUSSION

Ascending aortic aneurysm is mostly asymptomatic pathology, but it may have life-threatening complications, such as embolism, dissection, or rupture.^[1,4] The most common cause of aneurysm is atherosclerosis, and the disease is rare in individuals aged <50 years.^[9] Other rare causes of the disease are connective tissue disorders, such as Marfan disease and Ehlers-Danlos syndrome, syphilis, arteritis, and trauma.^[11]

In this report, we presented a case of atherosclerosis-related ascending aortic aneurysm that presented



Figure 5. Normal appearance of the main coronary arteries: the right coronary artery, the left anterior descending artery, and the left circumflex artery, respectively.

with ACS in contrast to the known complications. A negative result for *T. pallidum* antibody, ANCA, and ANA excluded the possibility of syphilis and vasculitis associated aneurysm. ^{18}F -FDG PET/CT also ruled out active large vessel vasculitis. In addition, the final pathologic examination of the specimen proved the atherosclerosis-related aneurysm with a degenerative aortic valve. Developing such a huge aneurysm at a younger age without secondary reason except atherosclerosis is very rare in the literature.

Concomitantly, the patient had significant AI. However, the patient did not have either bicuspid aortic valve disease or any other congenital valve disease that would be an explanation for the underlying pathology.^[10] Most likely, the reason for AI was atherosclerotic, degenerative valvular changes with dilation of aortic annulus owing to aneurysm. Pathologic examination supported our conclusion.

Overall, the development of widespread atherosclerosis-related thoracic aorta and its main branches in individuals of younger ages who are without cardinal cardiovascular risk factors is very rare. The findings indicate such systemic effects on the great arterial vasculature, such as genetic tendency, apart from the commonly known connective tissue disorders, such as Marfan disease, Ehlers-Danlos syndrome, and others. *ACTA2*, *COL3A1*, *TGFBR1*, *TGFBR2*, *SMAD3*, *TGFB2*, *MYLK*, *MYH11*, and *PRKG1* genes are frequently searched genes in academic settings.^[11] Genetically mediated thoracic aorta aneurysm consists

of approximately 5% of thoracic aorta aneurysm.^[12] Unfortunately, we were not able to perform genetic testing for the patient.

The patient presented with 1-hour chest pain, dynamic ST deviation, and elevated cardiac enzymes that all were indicating the diagnosis of acute non-ST-segment elevation myocardial infarction (MI) as per the fourth universal definition of MI.^[13] In the literature, there are examples of mechanical compression of coronary arteries by aneurysm presenting with ACS.^[7] However, our case did not have any obstructive CAD or mechanical compression on CTCA or regional wall motion abnormality on echocardiographic examination. Myocarditis can also mimic ACS at this age,^[14] but this was unlikely in our case considering low CRP, negative results for infective causes, and lack of previous respiratory infection. Probable explanations for the patient's ACS-like presentation may be coronary microembolism, dilated left ventricular chamber, or impaired diastolic coronary perfusion due to significant AI.

Ascending aorta aneurysms are mostly asymptomatic but may have dramatic outcomes in case of complications, such as embolism, aortic dissection, and rupture.^[5,6] However, as in our case, it may also present with chest pain and ECG changes mimicking acute MI.^[15] For differential diagnosis, bedside cardiac POCUS examination may be very helpful to rule out other life-threatening conditions, such as aortic dissection, aortic aneurysm, tamponade, or severe

valvular disease, and to avoid harmful invasive procedures and/or unnecessary medical treatments.^[15,16]

Another significant aspect of this case presentation was the importance of bedside echocardiography in patients with ACS presenting in an atypical clinical setting mimicking high-risk features (such as dynamic ST deviations over several leads with an absence of a typical pattern of ST-segment elevation MI). A quick look over the heart using POCUS can easily give an opinion on regional wall motion of the left ventricle, aortic root, pericardium, valvular structures, and their related pathologies (such as aortic dissection or aneurysm, pericardial tamponade, severe valvular insufficiency, or stenosis) that can easily mimic ACS.^[17,18] In our case, we performed the POCUS on arrival owing to physical examination and ECG findings, and POCUS findings changed the management accordingly from ACS-directed management to acute aortic aneurysm management.

Ascending aortic aneurysm with severe AI at a younger age is an extremely rare condition. It may mimic ACS, and bedside cardiac POCUS examination is very helpful in the initial differential diagnosis. In this paper, we reported a successfully diagnosed and treated giant ascending aortic aneurysm with severe AI.

Informed Consent: Written informed consent was obtained from the patient for the publication of the case report and the accompanying images.

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