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A case of aortic dissection presenting with a transient ischemic attack

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The authors stated that the written consent was obtained from the patient presented with images in the study.

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Abstract

Aortic dissection (AD) is the rupture of the aortic intima, separation of the tunica media and blood filling into the wall. It is an exceedingly rare life-threatening disease with a high mortality rate. The pathogenesis of AD is multifactorial and aortic diseases such as aortic dilatation, aneurysm, ectasia, arteritis, bicuspid aorta, aortic arch hypoplasia, coarctation, chromosomal abnormalities (Turner, Noonan), connective tissue disease (Marfan, Ehlers-Danlos) are well-defined risk factors for aortic dissection. Chest pain is the most common clinical presentation of AD. Cardiovascular and neurological systems are often affected. Patients with AD may also present with unexpected symptoms such as syncope, hemiparesis-hemiplegia, paraparesis-paraplegia, myocardial infarction, dysphagia, and side pain. In this article, we present a patient who presented to the emergency department with neck pain, amaurosis fugax, and hypotension, who was admitted to the neurology ward with suspicion of transient ischemic attack and diagnosed with aortic dissection.

Keywords: Aortic dissection, Transient ischemic attack, Hypotension

Introduction

Aortic dissection (AD) is the rupture of the aortic intima, separation of the tunica media and blood filling into the wall. AD, which has a high mortality rate, is a rare life-threatening disease [1]. The pathogenesis of AD is multifactorial and aortic diseases such as aortic dilatation, aneurysm, ectasia, arteritis, bicuspid aorta, aortic arch hypoplasia, coarctation, chromosomal abnormalities (Turner, Noonan), connective tissue disease (Marfan, Ehlers-Danlos) are well-defined risk factors for aortic dissection. The most widely used terminology in AD is DeBakey classification: Type I dissection starts from the ascending aorta and extends through the transverse arch to anywhere in the descending aorta. Type II starts from the ascending aorta and ends before the innominate artery. Type III starts from the left subclavian artery region and ends in the diaphragm (IIIa) or abdomen (IIIb) [2]. The Stanford classification, which is another classification used in AD, is more functional. Accordingly, aortic dissections are divided into two types. Regardless of where Type A primary tear is located, all dissections in which the ascending aorta is involved are called Type A, and distal involvement in the subclavian artery is called Type B [3].

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Chest pain is the most common clinical presentation of AD. The pain in the chest is severe, sometimes tearing, stabbing, or sharp, is sudden onset, and reaches the maximum in a brief time. Studies report that 10-55% of patients have painless aortic dissection [4]. More than one-third of the patients with AD present symptoms and signs of secondary organ involvement. Cardiovascular and neurological systems are the most involved [5]. Patients with AD may also present with unexpected symptoms such as syncope, hemiparesis-hemiplegia, paraparesis-paraplegia, myocardial infarction, dysphagia, and side pain. Studies showed that 31-39% of acute aortic dissections are initially misdiagnosed [6]. In this case report, we present a patient admitted to the emergency department with neck pain, amaurosis fugax, and hypotension, who was transferred to the neurology department with the preliminary diagnosis of a transient ischemic attack. The study aims to examine the patient diagnosed with aortic dissection by further investigations in the light of literature.

Case presentation

A 57-year-old male patient with no history of chronic diseases and trauma was admitted to the emergency department with complaints of neck pain, dizziness, hypotension, and transient visual loss in his right eye. Neurological examination of the patient in the emergency department was normal. The patient's vital signs were as follows: Blood pressure was 80/50 mmHg, fever was 36.2°C and pulse was 72/min. Electrocardiography (ECG) was compatible with early repolarization. There were no abnormal findings except for creatinine elevation in routine blood tests. The cardiac enzymes examined in the emergency department were normal. D-dimer value, chest X-ray (Figure 1), cranial tomography (Figure 2a), and cranial diffusion (Figure 2b, 2c) were normal in magnetic resonance imaging (MRI).

Figure 1: Normal chest X-ray

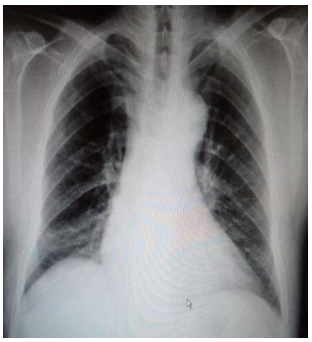


Figure 2a: Normal cranial tomography findings

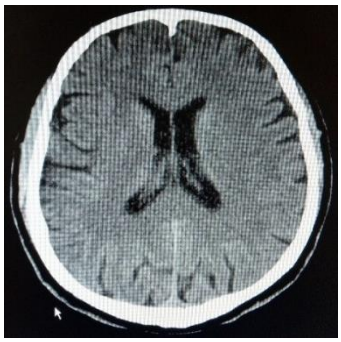


Figure 2b: Normal Diffusion-weighted magnetic resonance imaging

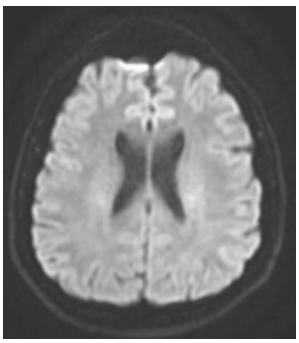
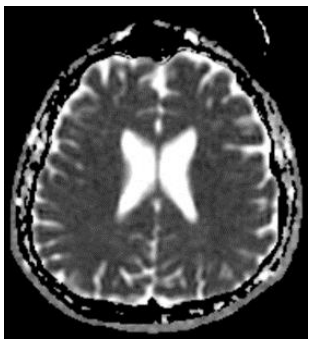
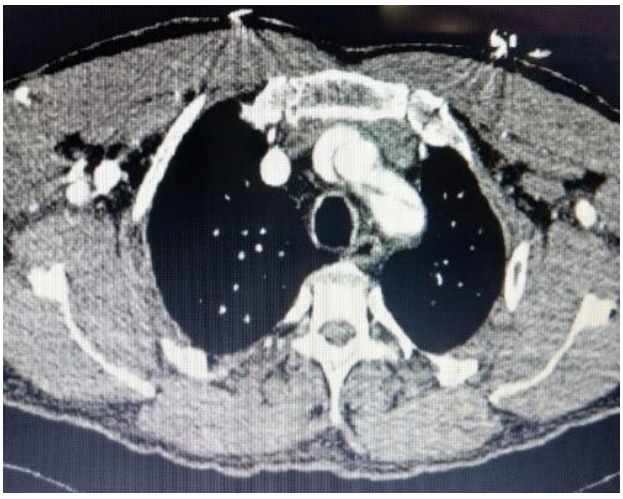


Figure 2c: Normal Apparent diffusion coefficient



The patient was hospitalized in the neurology department with a preliminary diagnosis of a transient ischemic attack. Forty milligrams of low molecular weight heparin (enoxaparin sodium) was administered twice a day, and 100 mg acetylsalicylic acid was administered once a day. The patient was hydrated with isotonic saline for hypotension. Carotid-vertebral artery Doppler ultrasonography (USG) was performed to investigate the etiology of the transient ischemic attack after admission to the neurology department. Carotid-vertebral artery Doppler USG revealed an intimal flap extending from the arcus aorta to the mid-section of the right common carotid artery (CCA), and that the thrombosed pharyngeal lumen was observed in the superior region of the intimal flap, which was compatible with a thrombosed dissection. The cardiology department was consulted to investigate the etiology of the transient ischemic attack and hypotension. The patient was then diagnosed with aortic dissection by transthoracic echocardiography (TTE). His ejection fraction was 60%. The ascending aorta was measured as 55 mm. A dissection flap was seen in the aortic arch. The patient was referred to the cardiovascular surgery department in a tertiary medical center for treatment. Thoracic computerized tomographic (CT) angiography and abdominal aortic angiography were performed at the center where the patient was referred for further examination and treatment, which revealed that the diameter of the ascending aorta was 51 mm and an intraarticular lumen dissection flap starting from proximal aorta extending to the aortic arch (Figure 3). The dissection flap was monitored along the right brachiocephalic trunk. No pathological finding was present in abdominal CT angiography. The patient was operated on by the cardiovascular surgery department with the diagnosis of aortic dissection, recovered uneventfully, and was discharged from the hospital.

Figure 3: Thoracic computed tomographic (CT) angiography: The diameter of the ascending aorta was 51 mm, and an intraarticular lumen dissection flap was observed starting from proximal aorta to the aortic arch.



Discussion

Aortic dissection is a life-threatening disease characterized by sudden chest and/or back pain. The disease is twice as common in males compared to females [7]. It should be kept in mind that aortic dissection may be painless in 10-55% of cases [5]. These cases are often associated with signs of stroke, coma or spinal cord ischemia, acute renal failure, myocardial infarction, and mesenteric ischemia [8].

Studies report that aortic dissection should be considered in patients with symptoms and findings such as back and chest pain before the stroke, syncope, hypotension, lack of pulse, and aortic regurgitation murmur [9]. The first and crucial point in the diagnosis of aortic dissection is to think of dissection. CT angiography is most used as the first diagnostic test in aortic dissection. MRI, CT angiography, and TTE have similar sensitivity and specificity in their diagnosis. Due to the high cost of these tests, access difficulties, high radiation exposure, the use of contrast material, and consequently the occurrence of adverse effects such as anaphylaxis and acute renal failure, more simple and useful biochemical tests were required in the case of suspicion of aortic dissections and the exclusion of this diagnosis [10]. Some studies indicate that serum D-dimer values may be high in aortic dissection, and D-dimer level may be a useful parameter in the exclusion of aortic dissection diagnosis [11]. Blood D-dimer levels should be evaluated within 6 hours after the onset of symptoms, especially in the exclusion of the diagnosis of aortic dissections presenting with painless, atypical clinical findings. Detection of elevated D-dimer allows patients to undergo faster imaging (CT, MRI) procedures and be referred to surgery faster, but this test has high sensitivity and low specificity. The literature review showed that CT, chest X-ray, and ECG were normal in a 66-year-old male patient who had transient ischemic attack findings in the form of weakness in the left arm during and after syncope. The patient's neurological examination was normal. Pulse rate was 45/min and arterial blood pressure was 80/60 mmHg. D-dimer value was > 4000 ng/ml (normal range <500 ng/ml). A thoracic CT angiography was performed on the patient at once and the patient was referred to surgery with the diagnosis of an ascending aortic dissection [12]. In another study involving 61 patients, blood D-dimer levels were not always high in patients with acute aortic dissection and there was no correlation between acute aortic dissection and D-dimer levels [13]. In our case, blood D-Dimer levels were normal.

There are reports of aortic dissection cases presenting with neurological symptoms and complications in the literature. In one of the case reports, an 84-year-old female patient with transient ischemic attack symptoms and hypotension in the form of recurrent aphasia episodes and right-sided weakness was reported to have aortic dissection in transesophageal echocardiography performed to investigate the etiology of transient ischemic attack and it was emphasized that acute painless aortic dissection may present with recurrent transient ischemic attack symptoms [14]. In another case report, a 73-year-old female patient presented with transient ischemic attack symptoms in the form of transient left hemiparesis and dysarthria. Carotid Doppler ultrasonography revealed CCA stenosis and mobile flaps on CCA origin and the patient was evaluated by thorax tomography and the diagnosis of thoracic aortic dissection was confirmed [15]. Like other published case reports, our case also presented with transient loss of vision in the right eye (amaurosis fugax), transient ischemic attack symptoms in the form of dizziness, and hypotension. It was highlighted that aortic dissections should be considered in patients with transient ischemic attack symptoms without chest or back pain.

Conclusion

Cerebral ischemic events can be observed in 5-10% of cases with aortic dissection. The most important factor in the diagnosis of acute aortic dissection is considering the possibility of dissection. In our case, there were no typical findings of aortic dissection. Therefore, it is important to keep in mind the diagnosis of aortic dissection in patients presenting with unexpected symptoms such as hypotension, neck pain, and focal neurological deficits to the emergency department and evaluate patients with appropriate imaging techniques. The mortality rate could be significantly reduced by providing early treatment with a rapid and accurate diagnosis process.

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