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Aortic Dissection Presenting as Paraplegia Complicated with Pulmonary Embolism

Pulmoner Emboli ile Komplike Parapleji Olarak Başvuran Aort Diseksiyonu

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Öz

Aort diseksiyonu ve pulmoner emboli ciddi ve potansiyel olarak yaşamı tehdit eden kardiyovasküler hastalıklardır. Akut aort diseksiyonu sonucu gelişen parapleji en ciddi komplikasyonlardan biridir. Venöz tromboembolizm riski paraplejili hastalarda artar. Aort diseksiyonu, parapleji, derin ven trombozu ve pulmoner embolinin birlikteliği çok nadirdir. Bu yazıda, akut tip I aort diseksiyonu sonucu spinal kord iskemisine bağlı parapleji gelişen, subakut fazda derin ven trombozu ve pulmoner emboli ile komplike olan 81 yaşında bir erkek hastayı sunduk.

Anahtar Kelimeler: Aort Diseksiyonu, Derin Ven Trombozu, Parapleji, Pulmoner Emboli

Abstract

Aortic dissection and pulmonary embolism are serious and potentially life-threatening cardiovascular diseases. Paraplegia is one of the most life-threatening complication of aortic dissection. The risk of venous thromboembolism increases in patients with paraplegia. The coexistence of aortic dissection, paraplegia, deep vein thrombosis and pulmonary embolism are very rare. This paper reports a case of an 81-year-old male patient, developing paraplegia related to spinal cord ischemia due to acute type I aortic dissection, in the subacute phase, complicated with deep vein thrombosis and pulmonary embolism.

Keywords: Aortic dissection, Deep Vein Thrombosis, Paraplegia, Pulmonary Embolism

Introduction

The incidence of acute aortic disection (AD) is 2.6–3.5 cases per 100 000 person-years, and it is characterized by the presence of an intimal flap separating the true from the false lumen. Acute AD occurs most commonly in males and typically presents with sudden, severe chest and back pain (1). There are several classifications of AD, but the most commonly used are DeBakey and Stanford. DeBakey's classification distinguishes 3 types: type I the most common type, dissection involves the entire aorta, type II; it involves only the ascending aorta and type III; dissection involves only the descending aorta. Stanford type A correspond to DeBakey types I and II, and Stanford type Bto type III (1,2). Paraplegia due to spinal cord injury in patients with AD is a rare complication that occurs in about 2-8 % (3).

Venous thromboembolism encompasses deep vein thrombosis (DVT) and pulmonary embolism (PE). DVT can be determined in approximately two-thirds of PE patients. AD and PE are common and life-threatening cardiovascular disease. Early diagnosis and appropriate treatment are important due to the high mortality rates of both diseases. The combination of these two conditions are exceptional and forms a therapeutic dilemma, between the anticoagulant treatment for pulmonary embolism that would negatively affect the dissection and the harmful therapeutic abstention due to the embolism (2).

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Case

An 81-year-old male patient admitted emergency unit with complaints of shortness of breath, weakness and inactivity in the legs, inability to stand and walk. Chest pain, which began 21 day ago, lasted half day, hit the back of patients and descendingly stopped, was reported. Moreover, inactivity of the legs began on the same day. The swelling initiated in the patient's left leg a week earlier than the admittance. Another complaint was sudden onset of shortness of breath in the last 2 days. The family did not have a history of hypertension and AD. The patient did not have any history of coagulation disorders, previous PE or DVT.

In physical examination, fever was 36.9 °C, blood pressure was 100/60 in the right arm and 90/60 mmHg in the left arm, radial pulse was 103/min, oxygen saturation was 89% at room air and respiratory rate was 24/min. In blood gas evaluations, pH was determined as 7.37, PaCO2 was 42 mmHg, and PaO2 was 82 mmHg. His height was 172 cm and his weight was 86 kg. 1/4 diastolic murmur was heard in the aortic focus. All peripheral arterial pulses could be measured. There was a significant increase in diameter in the left lower extremity and Homan's test result was positive.

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Simplified version of Geneva clinical prediction score for pulmonary embolism was 5 (PE-likely≥3).

Patient could not stand with or without support. There was hypoesthesia starting from the bottom of the nine (9) thoracic vertebrae. Bilateral Babinski reflexes were positive. Paralysis was observed in both lower limbs, more apparently in the left lower limb. Muscle strength in right lower extremity was 3/5 in proximal and distal, in left lower extremity was 2/5 in distal and 3/5 in proximal.

The patient had no significant abnormalities in the cervical and lumbar spinal magnetic resonance imaging (MRI) examinations. De Bakey type I AD that started from the ascending aorta to the descending aorta was detected in the thoracic spine MRI (Figure 1). Moreover, the primary pathological image was not identified in vertebrae except spondylarthrosis. The sinus tachycardia (127/min), S1Q3T3 pattern, V1-3 T wave negativity and DII, III, aVF, V4-6 ST segment depression were observed in electrocardiography (Figure 2).

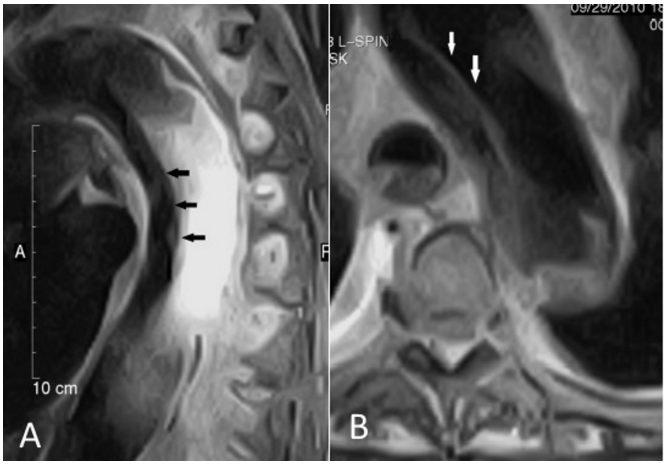

Figure 1. (A) Dissecting flap was observed in the aortic arch and descending aorta in axial MRI *(black arrows)*; **(B)** sagittal FSE T2 sequences MRI *(white arrows).*

In posteroanterior chest radiography, both hilar and mediastinal was found to be wide, oligemic areas in the middle zone of the right lung, thoracic aortic aneurysm and cardiothoracic index were found to be increased (Figure 3A). In transthoracic echocardiography, right heart dilatation, flattening of the interventricular septum and with depressed contractility of the right ventricular free wall compared to the echocardiographic right ventricular apex (McConnell sign) was observed. Pulmonary artery pressure was measured as 50 mmHg in the patient with moderate tricuspid valve insufficiency (Figure 3B). Moreover, the diameter of ascending aorta was observed as 5.8 cm, and dissection flap that started from the descending aorta lying abdominal aorta was seen (Figure 3C). In addition, moderate regurgitation was observed in aortic valve. Any pathological finding other than the image of the dissection extending beyond abdominal aorta was revealed in abdominal ultrasonography. Thrombus that was in distal part of the left superficial femoral vein and in popliteal vein in subacute period was observed in lower extremity venous Doppler ultrasound.

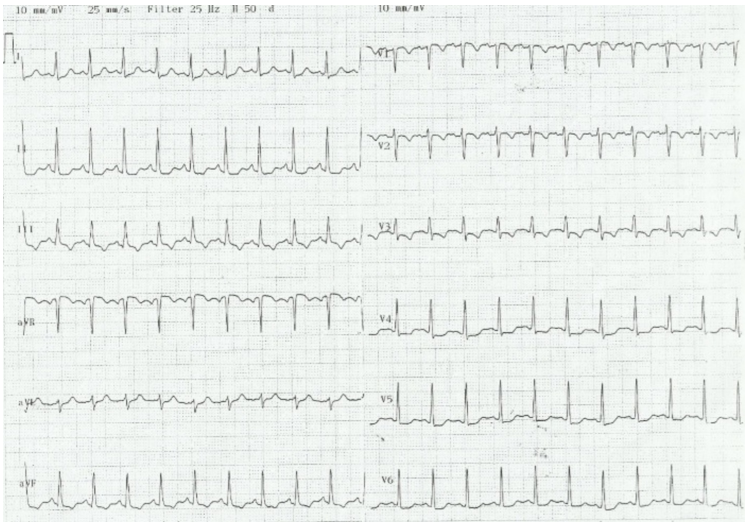

Figure 2. 12-lead electrocardiogram during admission.

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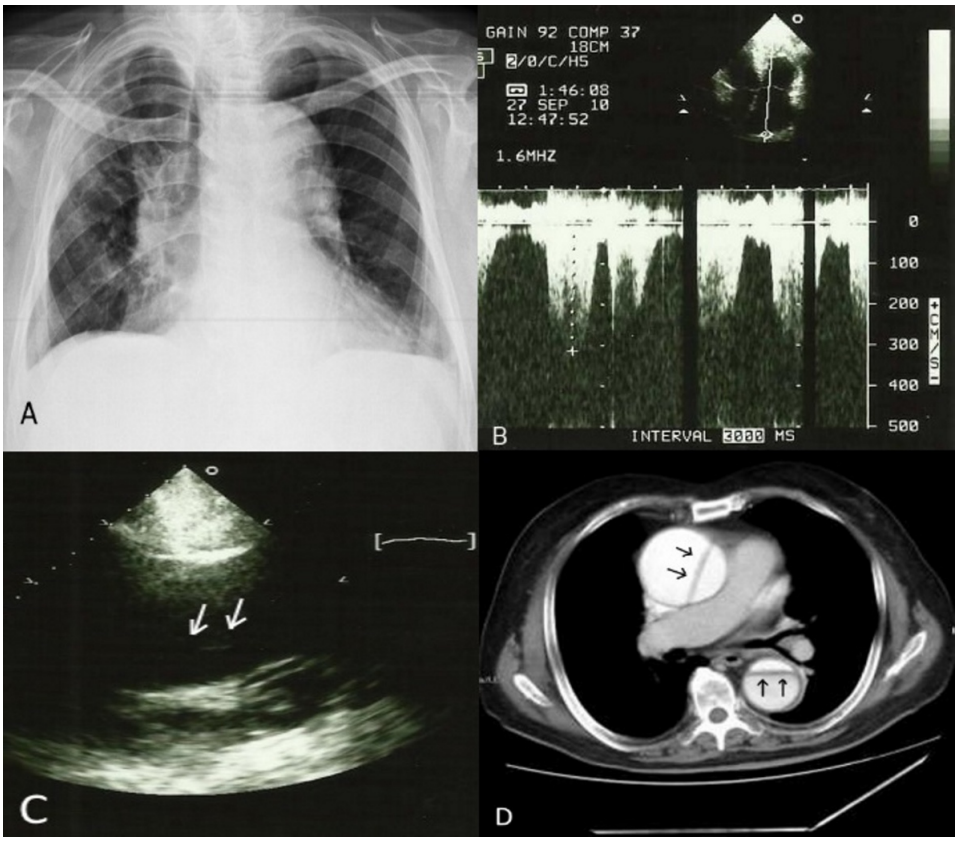

Figure 3. (A) Cardiomegaly and broad view in mediast were monitored on posteroanterior chest radiograph; **(B)** Transthoracic echocardiography revealed right heart dilatation and moderate pulmonary artery hypertension (50 mmHg); **(C)** 5.8 mm diameter of the ascending aorta with aneurysm view in the parasternal long-axis and dissection flap were observed in transthoracic echocardiography *(white arrows)*; **(D)** Flap of chronic type I dissection in both ascending and descending aorta and true and false lumen images were observed in the thoracic CT *(black arrows)*.

The results of blood biochemical examination were as follows; D-dimer >5.00 ug/ml (0.063-0.704), Troponin T 0.062 ng/ml (<0.014), proBNP 984 ng/mL (0-125), White Blood Counts 10.0 103/uL (4-11), Hb 11.6 gr/dL (11-18.8), Thrombocyte Counts 189 103/uL (150-400), Blood Urea Nitrogen 74 mg/dL, Creatinine 1.48 mg/dL (0.4-1.4), Glomerular Filtration Rate 48.4 mL/min/1.73 m2, Alanine Aminotransaminase 24 UL (0-50), Lactate Dehidrogenase 442 IU/L (0-450), Erythrocyte Sedimentation Rate 80 mm/s (0-20).

Contrast-enhanced pulmonary computed tomography (CT) angiography was not performed decreased glomerular filtration rate. Acute pulmonary embolism was diagnosed based on clinical findings, presence of deep venous thrombosis, electrocardiography and transthoracic echocardiography findings. Simplified pulmonary embolism severity index (sPESI) score was 3 (≥1). The patient was evaluated in intermediate-high risk cathegory. Cardiovasvascular surgery was consulted for an opinion. Medical management and close monitoring were continued for the patient. The intravenous unfractionated heparin was applied to the patient and this lasted for seven days. The warfarin sodium was initiated on the second day and treatment was continued for six months through keeping the international normalized ratio (INR) values between 2-3. Metoprolol succinate tablets 50 mg/day was subsequently started. In echocardiographical examination that was performed fifteen days later, the right heart chambers were measured as normal width, right ventricular wall motion as normal, and pulmonary artery systolic pressure was measured as 25 mmHg. Medical follow-up was decided for AD considering the patient's age, stage of subacute AD accompanying the complications and the patient's preference. Physical therapy rehabilitation program was started after fifteen days of hospital admission; a total of 30 physical therapy sessions was applied to the patient. He began to walk with only a cane 2 months later, and normally walked six months later. The physical examination findings were normal after sixth month and there was no neurological sequela. Lower extremity venous Doppler ultrasound performed at 6 months after discharge did not detect any residual thrombi in the left superficial femoral vein or popliteal vein. The diameter of ascending aorta was observed as 6.0 cm, and chronic type I dissection with aneurysm that started from the ascending aorta lying abdominal aorta was seen in

thoracic CT for control purpose two years later (Figure 3D). In the seven year of follow-up, ascending aortic diameter was 6.2 cm in transthoracic echocardiography and event-free survival continued. However, the patient was died due to urinary tract infection and progressive sepsis 10 years after suffering acute aortic dissection.

Discussion

AD is characterized with an intimal tear that suddenly occurs and separates the false lumen from true lumen of the aorta. Dissection can involve the side branches by spreading as the antegrade or retrograde, and it may cause the complications such as Malperfusion syndrome depending dynamic or static obstruction (3). AD is mostly seen in fifty or sixty years of age and often seen in males as twice than in females. Most cases have hypertension and the most common symptom is a sudden onset of severe chest or back pain. Some patients may not have pain. The mortality and morbidity rate of ascending AD is about 75% including especially in the first 24-48 hours at first two weeks. The prognosis of chronic dissection is better. The therapeutic approach towards AD depends on the location of involvement. Emergency surgery should be required in ascending AD while uncomplicated descending AD can be medically monitored at the beginning. Neurological sequelae after AD occur in approximately one fifth of patients. Paraplegia showing the spinal cord ischemia is mostly seen in descending ADs and may also occur in the ascending and abdominal ADs. The paraplegia can be also seen as a complication of thoracic and abdominal aortic surgery or in patients undergoing endovascular aortic graft (4). Paraplegia developed after dissection may be temporary or permanent. In acute phase of therapy, patients who had paraplegia receive the treatments such as cerebrospinal fluid drainage, anti-edema therapy, ensuring the volume balance and fluid-electrolyte, and prevention of hypotension (4,5). Following this, the patient may benefit from rehabilitation programs. In our case, paraplegia was completely resolved only after physical therapy and rehabilitation programs.

The anterior spinal artery that forms by the merger of the two branches of the vertebral artery nourishes the two third of anterior segment, two posterior spinal artery that is separated from the vertebral artery branch nourishes the one third of posterior segment of spinal cord. Other arteries that nourish the spinal cord through supplying it are the radicular arteries and primarily arise from the spinal branches of the lateral sacral, ascending cervical, deep cervical, vertebral, posterior intercostal arteries. Large anterior radicular artery, also known as the artery of Adamkiewicz, provides a large amount of blood to the two third of inferior part of spinal cord in low thoracal and lumbar region, and this artery is the most important artery of thoracolumbar region. This artery most commonly originates from the left side, between T9-12, inferior intercostal or superior lumbar arteries. Anterior spinal artery especially provides the blood supply to radicular arteries in the T10-12 level. Spinal cord blood flow is lower than other regions in T10-12 level. The nutrition of spinal cord in this region largely depends on anterior spinal artery blood stream and it is very sensitive to a decrease in blood stream. Therefore, the serious symptoms of spinal ischemia such as paraplegia may easily develop (6).

It may be difficult to distinguish spinal cord injury developing as a result of AD from acute transverse myelitis, the vascular diseases of the spinal cord, and other spinal cord diseases such as spinal cord malformations. Paraplegia in AD can suddenly develop or within minutes or hours. Acute transverse myelitis is a relatively indolent and there is no involvement of other organs. There are sudden onset of paraplegia, severe chest and abdominal pain in hemorrhage caused by spinal cord vascular disease. However, there is no other symptoms of AD in hemorrhage and be made distinct by imaging techniques such as magnetic resonance imaging. Intermittent claudication that suddenly starts and affects the legs is the main finding in patients with spinal vascular malformations. In this case, magnetic resonance imaging is useful in the differential diagnosis and spinal angiography confirms diagnosis (6).

The underlying mechanism simultaneous development of AD and PE remains unclear. Various factors may contribute to such comorbidity. Firstly, the likely cause of this simultaneous occurrence may be the close anatomical relationship between them, where compression of the right pulmonary artery induced by AD can cause the stagnation of blood flow that may lead to PE. Secondly, another possible cause may be DVT occurring in the lower extremities which travels up to the pulmonary artery and cause PE. Thirdly, AD can trigger a widespread coagulation response leading to venous thromboembolism (7).

It remains controversial how to treat PE patients concomitant with AD whether to choose fibrinolytic therapy, surgical embolectomy, percutaneous catheter directed treatment, pharmaco-mechanical approach, or inferor vena cava filter (7-10). Acute aortic dissection is a contraindication for thrombolytic and anticoagulant therapy.

Chen et al (7), successfully treated patients with descending AD combined with low-risk PE with a nonsurgical, non-anticoagulant treatment regimen. Nakamura et al (8), successfully treated a patient with aortic dissection who developed massive pulmonary embolism in the second postoperative week with anticoagulant therapy and urokinase. Tudaron et al (9), treated a patient with acute pulmonary embolism and dissected ascending aortic

aneurysm with unfractionated heparin and acenocoumarol and performed a successful operation after postponing surgical treatment for dissection for one month. Kagawa et al (10), detected a Stanford type A dissection, DVT, and PE in a 71-year-old woman. They successfully treated the PE in this patient with low-dose heparin, retrievable inferior vena cava filter, and warfarin-based anticoagulation.

In the moderate-high-risk group, thrombolytic therapy should be considered in case of clinical deterioration. Thrombolytic therapy was not considered for our patient because of the rapid improvement in his clinical condition within a few days after treatment, improvement in electrocardiographic findings, and complete normalization of right ventricular loading on echocardiography. In addition, the patient's excellent response to medical treatment and significant improvement in clinical and laboratory findings supported our diagnosis of pulmonary embolism.

Our case was a DeBakey type I AD in the subacute phase on the 21th day, who was admitted with acute PE. We started treatment of unfractionated heparin and continued warfarin-based anticoagulation for six months. We successfully treated venous thromboembolism and paraplegia without developing any adverse event in this case. We followed up the AD in this patient medically because the patient was older, the dissection was stable in the subacute phase, AD was complicated by paraplegia and PE and the patient did not want surgical treatment.

Conclusions

Consequently, the paraplegia may occur as a rare complication of AD. AD should be also considered in patients with paraplegia in the etiology. On the other hand, it should be taken into consideration that the risk of venous thromboembolism significantly increases in patients with paraplegia. The treatment of PE in patients with AD complicated with paraplegia is controversial. The treatment options should be considered based on clinical characteristics of each patient for following up such incidents.

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Conflict of interest statement

The authors certify that there is no conflict of interest with any financial organization regarding the material discussed in the manuscript.

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