


Case Report

Repetitive transient paraplegia caused by painless acute aortic dissection

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Case: Making a precise diagnosis of type A acute aortic dissection (AAD) presenting with atypical symptoms might be challenging for clinicians. Misdiagnosis and misuse of thrombolytic therapy can have devastating consequences.

Outcome: Herein, we report a case of painless type A AAD complicated by transient leg paresthesia, which was successfully treated with surgery. On admission, sudden onset of right leg numbness and muscle weakness was the only clue toward the correct final diagnosis.

Conclusion: When patients present with vague neurological symptoms, physicians should not rule out the possibility of AAD until proven otherwise.

Key words: Painless acute aortic dissection, paraplegia, surgical treatment

INTRODUCTION

ACUTE AORTIC DISSECTION (AAD) is an acute aortic syndrome leading to catastrophic outcomes in the absence of early diagnosis and treatment. The initial symptoms vary and can include chest and back pain, syncope, loss of consciousness, and cardiopulmonary arrest, whereas some patients are asymptomatic. Painless AAD is uncommon, with a reported incidence of 6.4–17%.^{1–3} It can be difficult to establish a final diagnosis in patients with atypical presentations, including those with painless AAD, especially as neurologic symptoms, which are well-known initial symptoms of AAD,^{3–9} can mimic a stroke and leave patients unable to describe their symptoms. As misdiagnosis and misuse of thrombolytic therapy can have devastating consequences, it is essential to clearly differentiate AAD from disorders with similar symptoms. Herein, we report a case of

painless AAD complicated by transient leg paresthesia, which was successfully treated with surgery. Patient consent for publication of the case details was obtained.

CASE REPORT

A 62-year-old man with a history of gastric ulcers was transferred to the emergency department of our hospital. He was complaining of sudden onset of right leg numbness and muscle weakness that had lasted for approximately 1 h. The patient denied any pain, and his initial symptoms resolved just before admission. The patient was a smoker and reported having smoked 15 cigarettes per day for 42 years. Bowel and bladder function were normal, and there was no significant medical family history. The patient was alert and oriented with no signs of acute distress. Vital signs on admission were normal (temperature, 36.4°C; heart rate, 80 b.p.m.; blood pressure, 128/44 mmHg in the left arm; and oxygen saturation, 96% on room air).

Examination was normal except for increased reflexes in both lower extremities. The pulsation in both the dorsalis pedis and popliteal arteries were palpable bilaterally.

Electrocardiography revealed normal sinus rhythm without ST-T changes. Chest radiography revealed no

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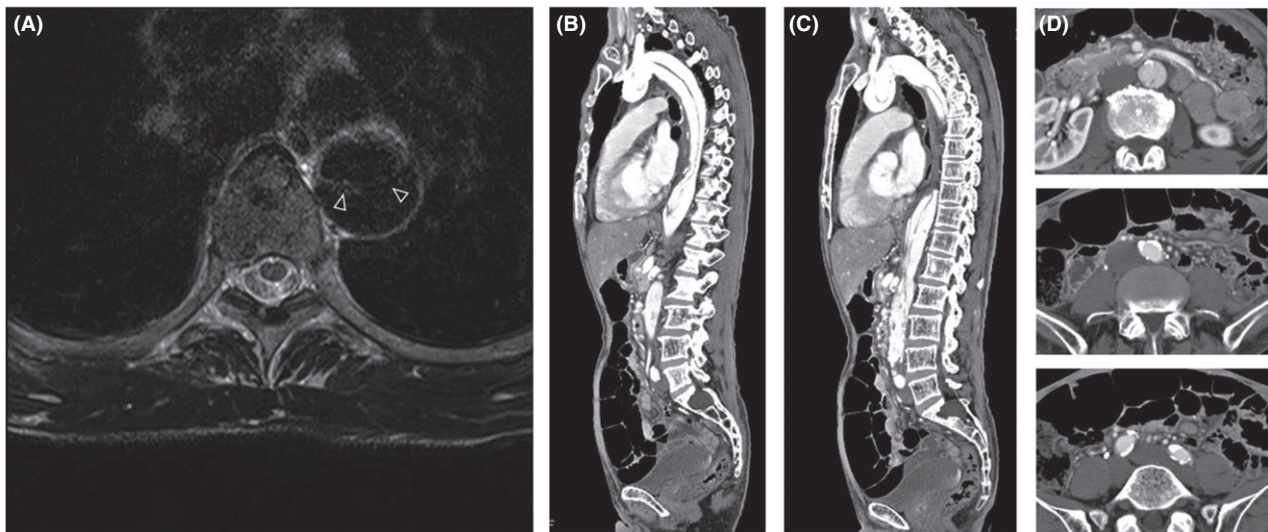


Fig. 1. Magnetic resonance imaging (A) and enhanced computed tomography (B, C) of the intimal flap of a 62-year-old man with painless type A acute aortic dissection. A, An intimal flap is seen in the descending aorta (arrow). B, The intimal flap extended from the ascending aorta to the descending aorta. The enhanced false lumen showed no thrombus formation. C, The abdominal aorta was intricately dissected without any findings of flow limitation in the visceral arteries. D, Aortic dissection ended at the proximal site of the aortic bifurcation. Blood flow to the common iliac arteries was not obstructed.

cardiomegaly, pulmonary congestion, or mediastinal enlargement, and the costophrenic angles were sharp.

Laboratory results revealed the following: white blood cell count, 10.9×10^3 cells/ μ L; red blood cell count,

389×10^4 cells/ μ L; hemoglobin, 13.4 g/dL; platelet count, 18.0×10^4 cells/ μ L; creatinine kinase, 243 IU/L; aspartate aminotransferase, 30 IU/L; alanine aminotransferase, 14 IU/L; lactate dehydrogenase, 365 IU/L; and C-reactive protein,

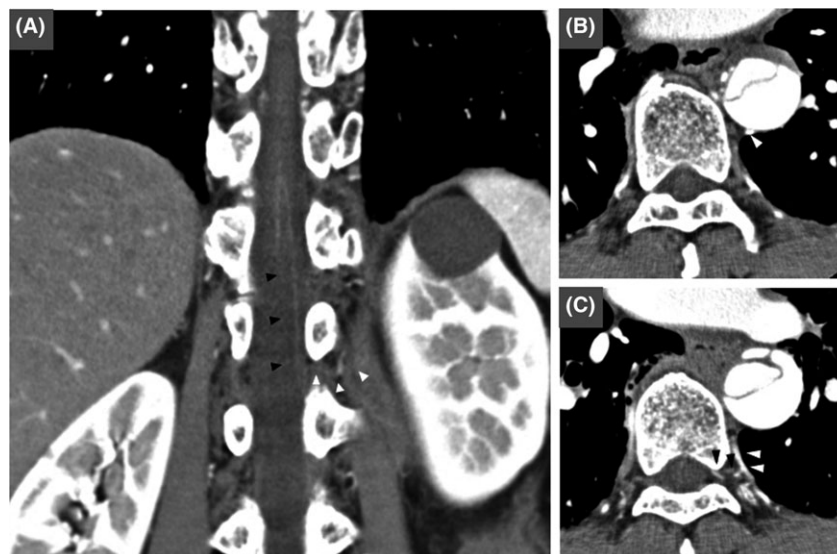


Fig. 2. Computed tomography evaluation of the artery of Adamkiewicz and intercostal artery of a 62-year-old man with painless type A acute aortic dissection. The artery of Adamkiewicz (black arrowheads) and the intercostal artery (white arrowheads) were enhanced on coronal and axial views. A, Coronal view showing the communication between the artery of Adamkiewicz and the intercostal artery running between two vertebrae. B, The intercostal artery originated in the false lumen. C, The connection between the artery of Adamkiewicz and the intercostal artery had no flow limitation.

0.06 mg/dL. Diffusion-weighted brain magnetic resonance imaging (MRI) did not reveal acute cerebral infarction; thus, transient ischemic attack was suspected.

Soon after the examination, paraplegia suddenly occurred, lasting for several minutes. Spinal cord infarction was suspected, but a thoracolumbar cord MRI did not indicate thoracolumbar infarction. However, the MRI revealed an intimal flap in the descending aorta (Fig. 1A). Thus, acute descending aortic dissection was considered a possible cause of paraplegia. However, vital signs remained stable, with no difference in right and left arm blood pressures (right, 105/60 mmHg; left, 110/83 mmHg), and the patient continued to deny pain.

D-dimer levels were elevated (75.1 $\mu\text{g/mL}$). Enhanced computed tomography (CT) revealed an intimal flap from the ascending aorta to the common iliac arteries (Fig. 1B–D), compatible with Stanford type A AAD, with blood flow in the artery of Adamkiewicz (Fig. 2, black arrowheads) and intercostal artery (Fig. 2, white arrowheads), both originating from the false lumen. The dissection extended into the left common carotid, left subclavian, brachiocephalic, and right common carotid arteries. An emergent operation was carried out. Neither aortic regurgitation nor wall motion abnormalities were seen on intraoperative transesophageal echocardiography. Intraoperatively, we found the false lumen entry in the ascending aorta and a massive bloody pericardial effusion that had not been noted on preoperative CT. Because the dissection did not reach the coronary artery ostia or sinus of Valsalva, ascending aorta replacement was carried out. Postoperatively, the patient developed respiratory complications that required intensive treatment.

On hospitalization day 17, the patient was extubated and started rehabilitation. Paraplegia did not recur. On hospitalization day 35, CT revealed that the descending aortic false lumen was patent, without progressive dilation, and there was blood flow in the artery of Adamkiewicz and intercostal artery (Fig. 3). On hospitalization day 39, the patient was discharged without residual complications, and remains asymptomatic at 18 months postoperatively.

DISCUSSION

IN A REVIEW of 1,805 patients, the rate of acute onset paraplegia or paraparesis in aortic dissection was reported at 4.2% (range, 2–8%).⁴ Transient paraplegia or paraparesis due to painless AAD has rarely been reported.⁵ These critical complications are due to anterior spinal artery ischemia. In the thoracolumbar region, the great anterior radiculomedullary artery, or artery of Adamkiewicz, supplies blood to the anterior spinal artery, which perfuses the



Fig. 3. Postoperative enhanced computed tomography (CT) evaluation of the false lumen of a 62-year-old man with painless type A acute aortic dissection. Enhanced CT on hospital day 35 revealed that the false lumen was patent and there was blood flow in both the artery of Adamkiewicz and intercostal artery postoperatively.

anterior two-thirds of the spinal cord. However, the artery of Adamkiewicz can be difficult to evaluate using conventional selective intercostal or lumbar angiography because of its small diameter, branching variation, and possible complications during angiography.¹⁰ Though highly variable, the artery of Adamkiewicz usually originates from the posterior aorta at the level of the T8 to L1 neural foramina. Most of the spinal cord receives additional blood supply from collateral vessels.^{4,11} In our patient, enhanced CT revealed that the artery of Adamkiewicz supplied blood to the anterior spinal artery.

In the present case, transient paraplegia might have been caused by an unstable dissected intima that obstructed the blood supply to the spinal cord or legs.^{5,12} Furthermore, the false lumen was well-enhanced through the other entry even after ascending aorta replacement and the closure of the primary entry by patch repair. As a result, blood flow to the artery of Adamkiewicz was maintained, which prevented paraplegia progression postoperatively.

The mechanism of painless AAD remains unclear.^{3–5,8,9,13} Several hypotheses have been proposed, which mention the speed of the dissection, lack of pain perception due to cerebral ischemic complications, and blockage of the normal pain perception neuropathways due to previous spinal ischemia.^{4,5,8,9}

CONCLUSION

PAINLESS AAD WITH acute neurological symptoms is rare and hard to diagnose emergently. The present case suggests that a high level of suspicion for AAD is warranted in patients complaining of neurological symptoms, even in the absence of pain.

DISCLOSURE

Approval of the research protocol: N/A.

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

Registry and the registration no. of the study/trial: N/A.

Animal studies: N/A.

Conflict of interest: None.

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