Case Report

Acute Postoperative Paraplegia Complicating with Emergency Graft Replacement of the Ascending Aorta for the Type A Dissection

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Postoperative paraplegia complicating with type A dissection is extremely rare. We describe a case of acute paraplegia after emergency graft replacement of the ascending aorta for type A dissection. A 63 year-old hypertensive man presented to hospital with chest pain. A chest computed tomography demonstrated an aortic dissection of the ascending aorta with mild pericardial effusion. Under deep hypothermic circulatory arrest with concomitant antegrade selective cerebral perfusion, the ascending aorta was replaced. The patient regained consciousness six hours after operation, however, he was complicated with paraplegia. All sensation was lost below the level of Th12. The cause of the paraplegia was assumed to be a thrombotic occlusion of costal arteries, which originated from the false lumen. Spinal fluid was drained for three days. Hyperbaric oxygenation therapy was started four days after onset of paraplegia. Fortunately, our patient recovered gradually, and he was doing well and was walking independently at one-year follow-up. (Ann Thorac Cardiovasc Surg 2003; 9: 330–3)

Key words: paraplegia, type A dissection, cerebrospinal fluid drainage, hyperbaric oxygenation therapy

Introduction

Paraplegia is a known complication after repair of descending thoracic and thoracoabdominal dissections of the aorta.[1] Usually, paraplegia in association with type A dissection is present preoperatively. Postoperative paraplegia that was not present preoperatively, complicating a repair of type A dissection of the aorta, is extremely rare.[2,3] We describe a case of acute paraplegia after emergency graft replacement of the ascending aorta for type A dissection.

Case Report

A 63 year-old hypertensive man presented to a hospital with chest pain radiating to the back, and consciousness disturbance. A chest roentgenogram revealed a widened mediastinum, and computed tomography (CT) demonstrated an aortic dissection of the ascending aorta with mild pericardial effusion (Fig. 1). He transferred to our hospital for operation 24 hours after the onset of initial symptoms. Physical findings indicated a temperature of 36.7˚C, blood pressure 110/68 mmHg, and pulse 80 per minute and regular. The 12-lead electrocardiogram were normal and equal femoral pulses. Neurological examinations were normal. Laboratory examinations were within normal limits. Pain had disappeared at our hospital with antihypertensive and negative inotropic drug medication. The echocardiography confirmed the aortic dissection of the ascending aorta with mild pericardial effusion with no evidence of aortic valve insufficiency. The exploration was through a median sternotomy, and the patient was placed on cardiopulmonary bypass (CPB) by way of the right femoral and right axillary artery and bicaval cannulation. Under deep hypothermic circulatory arrest with concomitant antegrade selective cerebral perfusion (SCP),

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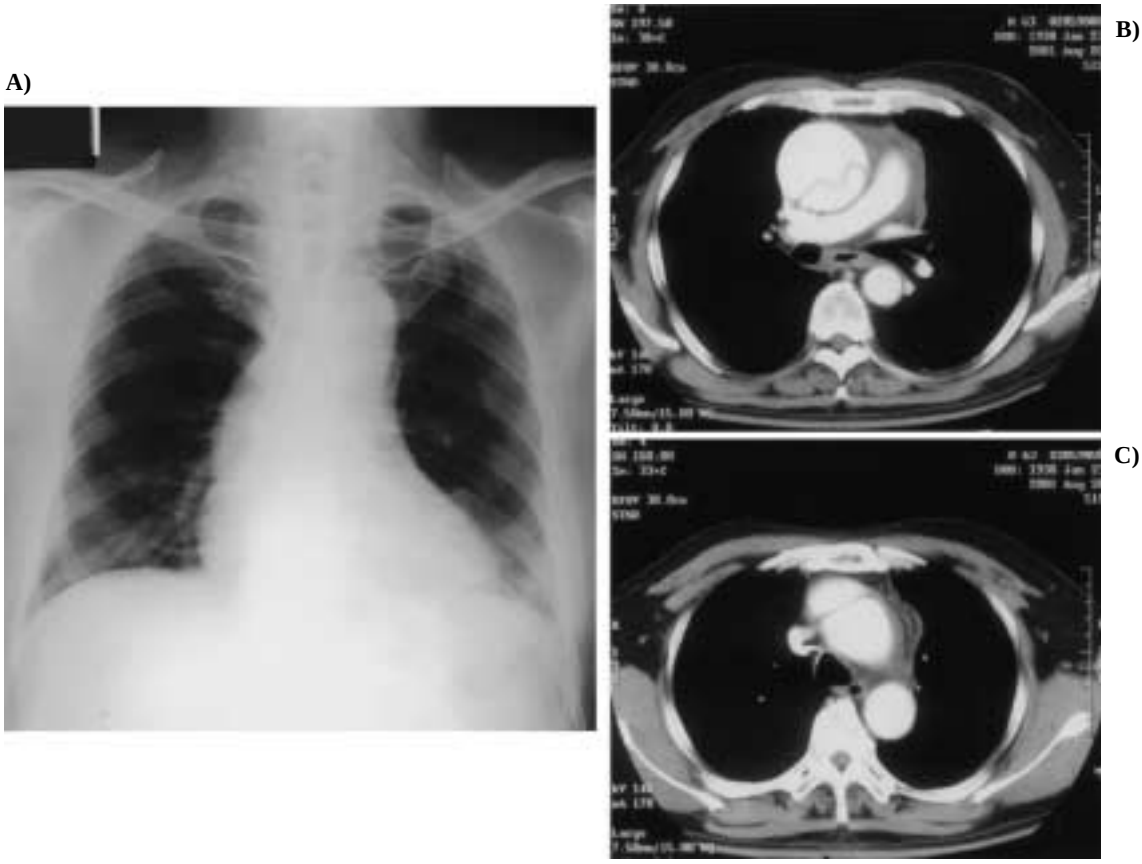

Fig. 1. A chest roentgenogram revealed a widened mediastinum (A), and CT demonstrated an aortic dissection of the ascending aorta (B) without dissection of arch (C).

the ascending aorta was replaced from just above the right coronary ostium to the level of the innominate artery with a 24-mm Hemashield (Meadox Medicals Inc., Oakland, NJ, USA) aortic graft. When the markedly dilated ascending aorta (65 mm) was incised, it was observed that the dissection extended close to the right coronary artery ostium to the descending aorta (type I dissection) with an intimal tear at the proximal portion of ascending aorta. Graft replacement of the ascending aorta was performed. After thrombotic material in the false lumen was removed, dissected layers of the aortic wall were approximated together with fibrin glue and the graft was sewn in place with continuous 3-0 Prolene sutures (Ethicon Inc., Somerville, NJ, USA) with reinforcing Teflon strips. Open distal anastomosis was performed under deep hypothermic circulatory arrest with rectal temperature at 20˚C. After the distal anastomosis was completed, retrograde perfusion was immediately started. Circulatory arrest was 41 minutes, myocardial ischemia 112 minutes, SCP 39 minutes, and CPB 145 minutes. Perfusion pressure was kept at 60 mmHg at the left femoral and/or left radial artery. The patient regained consciousness six hours after the operation, however, his lower limbs were found to be paralysed, and all sensation was lost below the level of Th12. The brain CT showed normal findings without any sign of infarction and hemorrhage. Paraplegia was diagnosed by disappearance of somatosensory evoked potential (SEP) and motor evoked potential (MEP), though responses of anterior columns were preserved. Magnetic resonance imaging (MRI) revealed partial spinal cord infarction at the level of Th12 (Fig. 2A, B). In the absence of any abnormalities, a diagnosis of spinal cord ischemia was considered most likely. The spinal pressure was measured, which showed 17 mmHg, therefore, spinal drainage to maintain pressure between 10-12 mmHg was continued for three days with the use of mannitol and steroids. However, this modality was not effective. We decided to treat with hyperbaric oxygenation (HBO) therapy. HBO was started four days after the onset of paraplegia and continued during the hospital stay, with the patient breathing 100% oxygen for 90 minutes at a pressure of 2.4 atmospheres absolute. The patient recovered gradu-

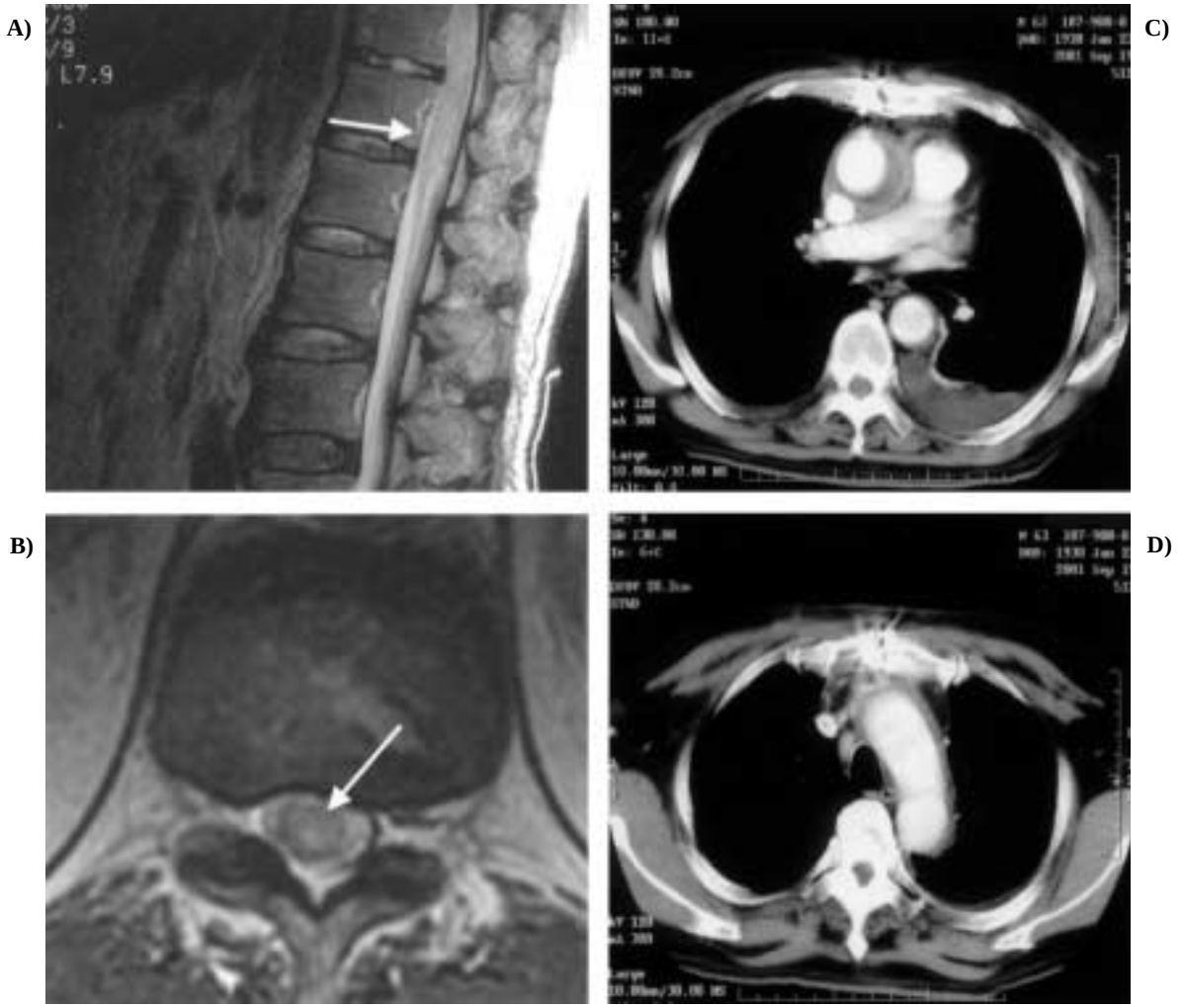

Fig. 2. T2-weighted MRI showing irregular high signals in the spinal cord of the Th12 level, meant spinal infarction (A, B). Postoperative CT demonstrating no residual dissection with a thrombosed false lumen on the descending aorta (C, D).

ally, and started to move his right leg two days after starting HBO. CT performed postoperatively demonstrated no residual dissection with a thrombosed false lumen on the descending aorta (Fig. 2C, D). He underwent extensive physiotherapy and was discharged 36 days after the operation to an other hospital for continuation of HBO therapy and rehabilitation. At discharge, he had regained the sensory function of his legs and could move his right leg well, but his left leg only slightly. At one-year follow-up, the patient was doing well and was able to walk by himself, although his left leg was weaker than the right leg.

Discussion

Ischemic spinal cord injury by aortic aneurysm surgery is a most serious complication.[4] Despite recent advances in operative techniques, anesthetic management, and postoperative care still remains unresolved. At present, the incidence of perioperative paraplegia ranges from 5% to 40%.[4,5] However, postoperative paraplegia complicating during the repair of type A dissection of the aorta is extremely rare.[2,3]

Postoperative paraplegia may be associated with a precipitating episode, like hypotension or hypovolemia leading to spinal cord ischemia.[3] Other factors, such as intraoperative proximal hypertension, elevation of cerebrospinal fluid (CSF) pressure, and postoperative hypotension, also have implications.[5] However, our patient did not suffer from any of these problems. The cause of acute paraplegia was thought to be insufficient radicular arterial flow caused by interruption of critical intercostals arteries, or extended aortic cross-clamp time.[4] In our case,

however, the duration of hypothermic circulatory arrest was 41 minutes. The vertebral and cervical arterial circulation were continued during the operation for SCP via left carotid and left subculavian arterial cannulation. Moreover, after the distal anastomosis was completed, retrograde distal aortic perfusion was immediately started. Therefore, we thought that spinal cord ischemia was ruled out.

The features of human spinal cord blood supply can markedly affect the strategies that should be adopted to prevent paraplegia. If anterior spiral artery (ASA) is continuity, the ASA could provide some blood supply to the thoracolumbar spinal cord from distant sources like the vertebral and cervical arteries, even if all intercostals arteries are occluded.[6] On the other hand, recently, it was stated that the ASA was segmental, via vertebral, intercostals, and lumbar arteries, with additional components from the cervical and iliac arteries.[7] Therefore, we thought that the cause of the paraplegia was assumed to be a thrombotic occlusion of costal arteries, which originated from the false lumen. Sakurada et al.[2] also reported the same case of paraplegia after graft replacement of the ascending aorta and total aortic arch. They described that the cause of the paraplegia might be thrombotic occlusion of costal arteries. It has also been described that the cause of paraplegia is likely to be related to the dissection in the descending aorta, whether it is secondary to an embolic event, ischemia, or flap closure of the origin of spinal artery.[3] Two main pathophysiological mechanisms have been hypothesized for this rare complication in our case. The first suggests an aortic dissection or adventitial hematoma leading to occlusion of important radicular arteries to the spinal cord with resulting spinal infarction. The second, is local occlusion of the artery of Adamkiewitz with thrombosed material without the presence of localized aortic dissection or hematoma. Actually, CT obtained postoperatively showed no residual dissection with a thrombosed false lumen on the descending aorta (Fig. 2C, D). There was no possibility of showering atherosclerotic emboli by retrogradely perfusing to the descending aorta because intraluminal atheromatic findings were not observed in the abdominal and descending aorta.

A variety of methods for intraoperative spinal cord protection, including CSF drainage, cord hypothermia, adjunctive pharmacology, reimplantation of intercostal arteries, and distal aortic perfusion, have been advocated.[3,4,8,9] However, the effectiveness of these prophylactic modalities cannot be conclusively determined. Once injury has occurred, treatment options are limited. The value of CSF drainage and HBO in the resolution of paraplegia is critically related to its implementation before irreversible ischemic damage occurs.[5,9,10] Therefore, there is no definitive postinjury treatment that guarantees improvement or resolution. This case demonstrates our lack of understanding of the unpredictable outcome of injury to the spinal cord.

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