Case report

Delayed paraplegia associated with vertebral necrosis after type A dissection surgery

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Abstract

Paraplegia is a rare complication of surgery for acute type A aortic dissection. We report a case of delayed postoperative paraplegia associated with necrosis of the thoracic vertebral bodies and soft tissue. The pathogenesis of delayed postoperative paraplegia is unknown, but our case report would strongly suggest ischemia of the descending thoracic intercostal arteries as the causative mechanism. The precipitating episode (respiratory distress syndrome with hemodynamic instability) might have promoted the compromised spinal circulation to become clinically evident in our patient. Treatment for this serious complication is mostly supportive, although CSF drainage may be helpful in the acute phase.

Keywords: Type A dissection; Paraplegia; Cerebrospinal fluid drainage

1. Introduction

Paraplegia after surgical treatment of acute aortic dissection is a rare complication. When it occurs, however, it has dramatic consequences for the immediate postoperative period as well as for long-term prognosis. There are very few case reports of paraplegia after surgical treatment of type A dissection [1—4]. We report a case of delayed postoperative paraplegia associated with necrosis of the thoracic vertebral bodies and soft tissue.

2. Case report

A 53-year-old male patient presented 7 h after initial chest pain, along with typical symptoms of aortic dissection. A CT-scan showed a dilated aortic root and ascending aorta, with dissection involving all aortic arch branches, and the superior mesenteric and left renal arteries. The dissection extended into both iliac arteries and the left leg was found to be pale and without a pulse.

The patient was operated on emergently. Cardiopulmonary bypass was established via right axillary perfusion and right atrial venous drainage. The entry tear was localized in close proximity to the right coronary ostium with concomitant dissection of the proximal right coronary artery (RCA). The RCA ostium was oversewn and a saphenous vein bypass graft was performed. The aortic root was replaced with a composite valve graft prosthesis. Circulatory arrest was instituted at a rectal temperature of 19 °C. The innominate artery was clamped and the left carotid and subclavian arteries were occluded by a balloon catheter. Antegrade cerebral perfusion through the right axillary artery was instituted. Twenty-four minutes were needed to perform distal repair (i.e. hemiarch replacement). The remainder of the procedure was uneventful. The patient was successfully weaned from CPB without need for inotropic support.

The immediate postoperative course was complicated by excessive bleeding. A redo sternotomy was performed without any identifiable bleeding source. The patient awoke the following day without any neurological deficit, and was extubated and discharged from the ICU. All peripheral pulses were palpable, and the left leg was perfused normally. No reintervention for peripheral malperfusion was required. Subsequently, readmission to ICU was required 2 days later because of a progressive deterioration of the patient’s pulmonary function. The CT-scan (Fig. 1) showed no pulmonary perfusion deficits, and slightly delayed opacification of the false channel in the descending aorta. The patient was reintubated and controlled mechanical ventilation with prone positioning was instituted. The oxygenation improved over the next few days. After discontinuing sedation on the eighth postoperative day, the patient was noted to have complete paralysis of the lower extremities. Cerebrospinal fluid (CSF) drainage was immediately performed and the pressure maintained constantly below 10 cmH₂O. The motor function of the right leg improved thereafter. The left leg, however, remained completely paretic. An MRI (Fig. 2)
showed an extensive myelopathy of the thoracic spinal cord (from 3rd to 8th thoracic segments), and early necrosis of the 6th–8th thoracic vertebral bodies with involvement of the adjacent soft tissue. An orthopedic consultation was obtained and it was decided that the patient did not have an indication for stabilization surgery. The left-dominant paraplegia slightly improved over the following 2 weeks and the patient progressed with mobilization. The soft tissue necrosis progressed to the skin and subcutaneous tissue over the left dorsal hemithorax, which was treated by

![Image of CT-scan](image1)

**Fig. 1.** Postoperative CT-scan. Postoperative CT-scan (A and C: arterial phase; B: venous phase) showing delayed perfusion of the false channel in the descending aorta. Both channels are simultaneously perfused in the abdominal aorta (part C).

![Image of MRI](image2)

**Fig. 2.** Spinal MRT. (A and B) MRT images showing the early necrosis of the 6th–8th thoracic vertebral bodies (white arrows) with involvement of the adjacent soft tissue (dotted arrow) in part (C).
debridement, vacuum drainage and full-thickness skin graft. The patient underwent extensive physiotherapy and was discharged to the rehabilitation center 38 days after the initial operation. At that time, he was able to walk a distance of 10 m with some support. At 1-year follow-up, the patient is able to walk distances of 500 m with a crutch. His left leg continues to be considerably weaker than the right, however, and he continues to complain of chronic neuropathic pain.

3. Discussion

Paraplegia after aortic surgery is a devastating complication. The exact mechanism of paraplegia after repair of an acute type A dissection is unknown. The proposed causes are insufficient radicular arterial flow because of the interruption of critical intercostal arteries (e.g. thrombosis of intercostals originating from the false lumen) or extended circulatory arrest times with subsequent spinal cord ischemia [5]. It has been postulated that a precipitating episode (i.e. hypotension, hypovolemia) could be associated with delayed paraplegia [6]. In the current case report our patient had respiratory distress syndrome with intravascular volume depletion and hypotensive episodes associated with the prone positioning, that could have additionally limited the blood flow to the jeopardized spinal cord. We suppose that occurrence of the respiratory distress syndrome was the underlying reason for delayed manifestation of paraplegia.

To the best of our knowledge, this is the first case report of extensive spinal injury and myelopathy accompanied by necrosis of the adjacent structures (i.e. thoracic vertebral bodies and surrounding soft tissue) following type A aortic dissection surgery. The segmental pattern of ischemic lesions, corresponding to the area supplied by the posterior intercostal arteries, strongly supports the hypothesis that the paraplegia was caused by occlusion of the intercostal arteries at the mid-descending thoracic level. The occlusion was likely related to the dissection in the descending aorta and could have been caused by embolism, thrombosis, or flap closure [2]. It is also interesting to note that the paraplegia in our patient was left-dominant, similar to other published case reports [1,2]. The left side of descending aorta is more frequently involved in the dissection process and serves as further argument for the critical role of the intercostals in the pathogenesis of this event.

The treatment options of overt paraplegia are limited. CSF drainage has been proven to reduce the incidence of paraplegia following surgery for thoracoabdominal aneurysms in randomized controlled trials [7,8]. In addition, there are case reports and small observational studies describing more complete recovery of delayed paraplegia after initiating CSF drainage [9]. In our case there was a 5-day interval between insertion of the CSF drainage and marked neurological improvement. It is therefore difficult to state that recovery was directly related to this intervention. Some authors [3] have described successful percutaneous fenestration procedure to relieve pressure differences between the true and false lumen in patients with stagnant blood flow in the false lumen of the descending aorta. The interventional fenestration has to be performed during a limited time interval after the onset of spinal ischemia and, therefore, was not appropriate in our patient. This treatment modality is still experimental and long-term sequelae in the setting of dissected thoracoabdominal aorta are unknown. Another group from Japan [1] has reported the successful use of hyperbaric oxygenation therapy for treatment of the post-operative paraplegia.

Some technical modifications in surgical and circulatory management of patients undergoing repair for acute type A aortic dissection may decrease the risk of intraoperative spinal cord damage, but the risk of delayed paraplegia in the setting of hypoperfused or even thrombosed false channel remains problematic. Therefore, focus should be placed on the augmentation of spinal cord perfusion and prevention of hypovolemia/hypotension events during the early post-operative course in the patients at seemingly increased risk of paraplegia (with early postoperative false channel hypoperfusion or thrombosis).

Our case report would strongly suggest ischemia of the descending thoracic intercostals arteries as causative mechanism for the paraplegia after type A dissection surgery, which became clinically overt with occurrence of respiratory distress syndrome.

References