



Case Report

Late onset posttraumatic vascular myelopathy without spinal chord injury – a case report

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Abstract

Background: Yet, we couldn't find any reported case of a trauma patient, that describes a late onset complete paraplegia of the lower limbs caused by spinal chord ischaemia without a spinal (chord) injury. Leading causes are dissections of the aorta and their reconstructions.

Case presentation: Here, we present a case of a 43 year old male patient with traumatic dissection of the external iliac artery and demolition of the iliac vein on the left side. Moreover, he was in hypovolemic shock. Injuries of the spine or the spinal chord were not apparent. He was able to move both legs on demand. After performance of damage control surgery at the day of the accident, the patient presented a complete sensoric and motoric deficit 7 days after surgery, the MRI showed a vascular myelopathy.

Conclusion: The myelopathy could be caused by impaired arterial perfusion due to hypovolaemia and disrupted arterial flow on the Adamkiewicz artery. A venous congestion as well as fibrocartilary embolisation are causes that also need to be discussed.

Keywords: Vascular myelopathy; late onset; posttraumatic; case report

Background

Spinal ischemia underlies 6% of acute myelopathies [1]. Spinal ischemia in traumatized patients is rarely described, and when it occurs, it is usually associated with direct trauma to the spine and consecutive vascular injury. More common causes are aneurysms of the aorta or ischemia after aortic surgery, compression of vascular structures by tumors, thrombosis or embolism and arterio-venous malformations.

Fibrocartilaginous emboli are described in up to 5.5% of cases for unexplained and sudden onset spinal ischemia [2]. Here, it is assumed, that with comparatively marginal loads on the intervertebral discs, nucleus pulposus material in the area of the Schmorl nodules embolizes into the spinal vessels.

The resulting sensory-motor deficits are often misdiagnosed as transverse myelitis in this context, as the group led by A AbdelRazek showed in their 2017 review. Accordingly, it can be assumed that fibrocartilaginous embolization occurs more frequently than previously thought [3].

MRI diagnostic showed an anterior hyperintense area in the region of the anterior spinal cord in the sagittal T2- weight, correspondingly hyperintense areas are often found in the axial layers [4, 5].

Whereas arterial occlusion in the spinal region quickly becomes symptomatic by neurological deficits, venous outflow obstruction may be

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clinically unnoticed for much longer and usually manifests as a slowly progressive neurological deficit. Depending on the cause, this may be the case for months. Treatment of vascular myelopathy or spinal ischemia primarily involves improving spinal perfusion. This includes the maintenance of adequate systemic blood pressure, if necessary by means of appropriate catecholamine therapy [6]. In addition, the pressure in the spinal canal can be reduced by means of a lumbar drain. Draining cerebrospinal fluid improves the microcirculation here [5]. In the recently published case report by Haynes et al, intraarticular thrombolysis with tissue plasminogen activator (t-PA) in combination with verapamil and an antiplatelet agent (Eptifibatide) was performed for acute occlusion of the anterior spinal artery in a 52-year-old patient. The sensorimotor deficits showed regression immediately after the intervention and after 8 months [7]. The prognosis of spinal ischemia depends on the severity of the neurologic deficit that presents at the beginning [5].

To date, no case has been described in the literature in which traumatized patients developed vascular myelopathy with a time delay without the presence of direct spinal trauma or direct vertebral vascular injury. This is the subject of our case report.

Case presentation

We present the case of a 43-year-old patient, who suffered a fall of an approx. 1-ton formwork element onto his body up to the level of his navel.

After the technical rescue by means of a crane, the patient showed shock symptoms from the beginning with initially existing tachycardia and in the course of time also hypotension. The polytrauma diagnostic of the patient, who was awake and breathing spontaneously on arrival in the emergency room, by means of CT showed an instable anterior and posterior pelvic ring fracture, a femoral shaft fracture on the right, a dissection of the external iliac artery on the left in the distal part, and a rupture of the iliac vein on the left. Injuries of the spine did not appear, the patient had no disturbances of sensibility on admission, all muscles of the lower extremity could be moved spontaneously and purposefully.

As part of the initial surgical treatment, the patient was treated on the same day with a symphyseal plate, an external fixator of the right femur and an arterial vessel reconstruction. Consecutively, a compartment syndrome had developed in the left upper and lower leg, which was split. Venous vessel reconstruction was performed the following day. Postoperatively, the patient remained ventilated for a total of 6 days with initial hemodynamic instability, unstable coagulation situation, and CVVHDF therapy due to a crush kidney with anuric renal failure. After extubation, the patient developed delirium, and a neurological examination was not possible immediately. After improvement of the delirium on the following day, a sensory-motor deficit of both legs from

the level of the navel was noticed. An MRI of the skull as well as of the entire spine showed a prolonged distension of the myelon from the conus medullaris to about the level of HWK 6 (Fig. 1-3) most likely corresponding to myelon edema, as caused by ischemia in the area of the A. radicularis magna. We consulted our colleagues in neurology and neurosurgery. A lumbar drain was placed, open decompression was not indicated. We decided not to administer high-dose methylprednisolone according to the second National Acute Spinal Cord Injury Study dosing protocol (NASCIS-2-regimen) because the time of onset of ischemia was uncertain but was probably present for longer than eight hours. In the meantime, the patient underwent further surgical treatment with a right femoral intramedullary nail, a right ISG screw, and secondary wound closure of the compartment syndrome. After six days, the lumbar drain was removed. Renal function improved and intermittent hemofiltration was no longer necessary. Finally, the patient was transferred to an early neurological rehabilitation. On transfer, the patient showed persistent complete paraplegia sub- TH10 with intermittent tingling paresthesias in both thighs and fecal and urinary incontinence.

Conclusion

Vascular myelopathies are usually associated with severe neurological sequelae. As described above, the most common cause is infarction.

We discussed possible causes for the delayed onset of paraplegic symptoms in an interdisciplinary manner. We most likely considered ischemia due to hypovolemia. Howard described prolonged hypotension combined with advanced vascular disease as one cause of spinal cord infarction [5]. Similarly, ischemia could underlie impaired inflow from the Adamkiewicz artery due to dissection. However, since this has a variable aortic outlet between Th9 and L1, this is rather unlikely. Although ischemia due to crush trauma from the concrete shuttering itself is possible, nevertheless, the sensory-motor deficit would presumably have been evident here by the time the patient arrived in the emergency room, making this hypothesis unlikely as well. Venous outflow was disturbed over a longer period of time, which could have led to venous congestion. This mechanism may be considered as another component of inferior perfusion. The patient described in our case showed smoldering nodules in the thoracic spine and lumbar spine as a possible cause for fibrocartilaginous embolization in the course of MRI diagnostics. Whether an embolization took place after the trauma, remains for this reason only to be assumed. Interventional radiological spinal angiography to verify the cause in combination with the possibility of spinal thrombolysis as described by Haynes can be discussed as an individual attempt at healing. However, current data on the lysis window are not available at present. However, in the case of lysis, local anticoagulation (by t-PA) and subsequent systemic anticoagulation required in a polytraumatized patient must be considered critically.

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