

New concept of pathogenesis of impaired circulation in traumatic cervical spinal cord injury and its impact on disease severity: case series of four patients

M. Salkov¹ · V. Tsymbaliuk² · L. Dzyak³ · A. Rodinsky⁴ · Y. Cherednichenko⁵ · G. Titov⁶

Received: 25 October 2014/Revised: 6 May 2015/Accepted: 6 May 2015
© Springer-Verlag Berlin Heidelberg 2015

Abstract

Purpose The purpose of this study is to justify a new concept of the pathogenesis of secondary changes in the cervical spinal cord, and its correlation with the depth of development of neurological disorders in spinal injury.

Methods Standard magnetic resonance imaging examination and angiography of the cervical and vertebral arteries of four patients were performed to diagnose the prevalence rate of ischemia and edema, and examine the spinal cord vasculature. Correlation of the data obtained with the neurological status was performed.

Results Collateral circulation is most apparent in the upper-cervical region, above the C4 vertebra. Following occlusion of the vertebral artery, the circulation above the C4 vertebra is performed by collaterals of the ascending

cervical artery. With extensive damage to the spinal cord, the intensity of edema and ischemia can be regarded as the effect of damage to radicular medullary arteries, which are injured in the intervertebral foramen. Secondary changes of the spinal cord are most apparent by impaired circulation in the artery of cervical enlargement.

Conclusions Collateral circulation is a significant factor that limits the damage to the cervical spinal cord. Impaired circulation in the artery of cervical enlargement is significant in extension of perifocal ischemia. The appearance of early arteriovenous shunting in the region of a primary spinal cord injury (contusion focus) by angiography is pathognomonic. The data obtained open a perspective for the endovascular treatment of spinal cord injury.

✉ M. Salkov
salkov@ua.fm
V. Tsymbaliuk
v.tsymbaliuk@i.ua
L. Dzyak
dsma@dsma.dp.ua
A. Rodinsky
a.rodinsky@gmail.com
Y. Cherednichenko
yuritch@ua.fm
G. Titov
natawaa@yandex.ru

¹ Dnepropetrovsk Medical Academy; Neurosurgeon of Spinal Department of Communal Institution, Dnepropetrovsk Regional Clinical Hospital named after I.I. Mechnikov, Dzerzhinsky str., 9, Dnepropetrovsk 49044, Ukraine

² Restorative Neurosurgery Department, The Institute of Neurosurgery named after A.P. Romodanov, 32 Platona Mayborody St, Kiev 04050, Ukraine

³ Department of the Neurology with Neurosurgery, Dnepropetrovsk Medical Academy, Dzerzhinsky str., 9, Dnepropetrovsk 49044, Ukraine

⁴ Department of Physiology, Dnepropetrovsk Medical Academy, Dzerzhinsky str., 9, Dnepropetrovsk 49044, Ukraine

⁵ Neuroradiology Department of Communal Institution, Dnepropetrovsk Regional Clinical Hospital named after I.I. Mechnikov, Govtneva sq. 14, Dnepropetrovsk 49005, Ukraine

⁶ Department of Communal Institution, Dnepropetrovsk Regional Clinical Hospital named after I.I. Mechnikov, Dzerzhinsky str., 9, Dnepropetrovsk 49044, Ukraine

Keywords Spinal injury · Spinal circulation · Angiography · MRI examination

Introduction

According to previously published studies, the main factors in the pathogenesis of spinal cord injury are attributed to primary and secondary mechanism. Primary spinal cord injury occurs at the time of the initial trauma or damage, forming a zone of spinal cord contusion. Disorders can be found in the local vasculature, causing edema and hemorrhage. In the damaged area, paralysis of motor, sensory and autonomic neuronal functions can occur. Vascular dysfunction, ischemia, glutamatergic excitotoxicity, inflammation and apoptosis also develop in the perifocal zone [1–5].

In our opinion, one of the leading mechanisms in the formation of secondary injury and its extensiveness is vascular dysfunction, which occurs not in the area of the spinal cord injury, but in blood vessels supplying the spinal cord, i.e., in the branches of the vertebral, ascending, and deep cervical arteries.

Through a worldwide literature search, we did not identify one detailed clinical description of the features of impaired circulation in spinal injury. Based on the fundamental experimental studies of G. Lazort et al. on vascularization and hemodynamics of the spinal cord, we have conducted a clinical study of impaired circulation in the cervical spine and spinal cord as a result of injury, and we have defined the role of collateral circulation in this type of pathology [6–8]. Based on the results obtained, correlation of vascular disorders with the severity of spinal cord

ischemia and the severity of the patients has also been conducted.

Materials and methods

From May to August of 2014, we examined four patients with cervical spinal trauma. All patients were men, aged 20–62. We performed standard magnetic resonance imaging (MRI) of the affected spinal area and selective angiography of the cervical and vertebral arteries to determine the extent of damage to the medullary region, spinal cord compression, the prevalence rate of ischemia and edema, and vascularization of the spinal cord. The studies were conducted from day 1 to day 30 post-injury. Neurological impairment on the American Spinal Injury Association (ASIA) scale corresponded to A in two patients and D in remaining two patients.

Case reports

Case 1

We conducted a study of the collateral circulation of the vertebral arteries in a patient with spinal trauma, spinal cord injury, and dislocation fracture of C4–C5. The uniqueness of the study is in the visualization of collateral circulation following occlusion of both vertebral arteries. Clinically, the patient did not show any stem disorders with impaired vital functions, and verte-brobasilar insufficiency was not evident. Collateral

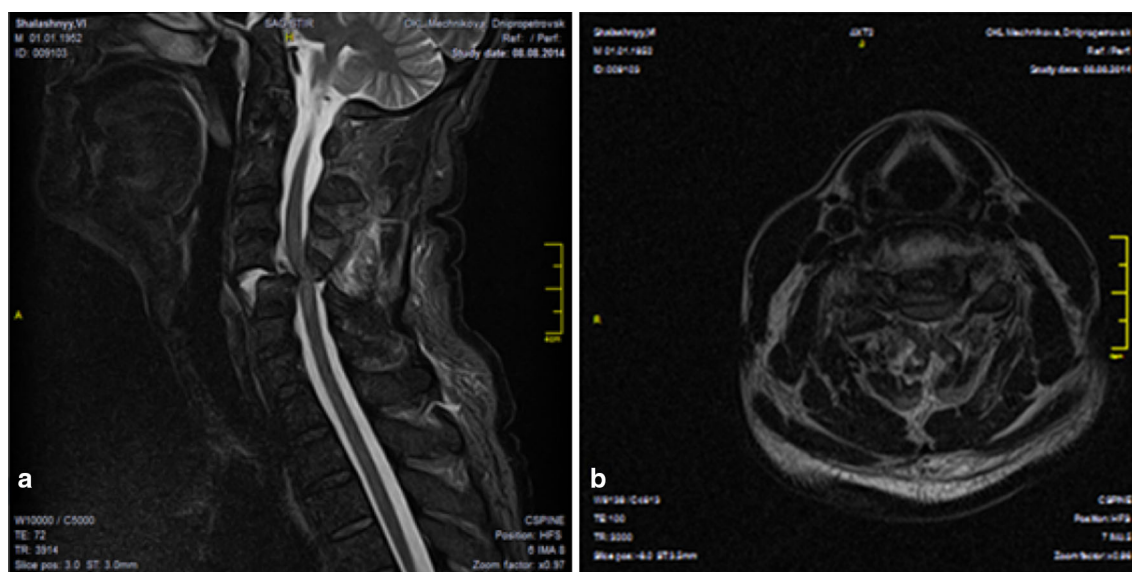


Fig. 1 Magnetic resonance imaging of Case 1. A dislocation fracture of C4–C5 can be observed, along with myelopathy at the level of C4–C5. **a** Sagittal view. **b** Axial view

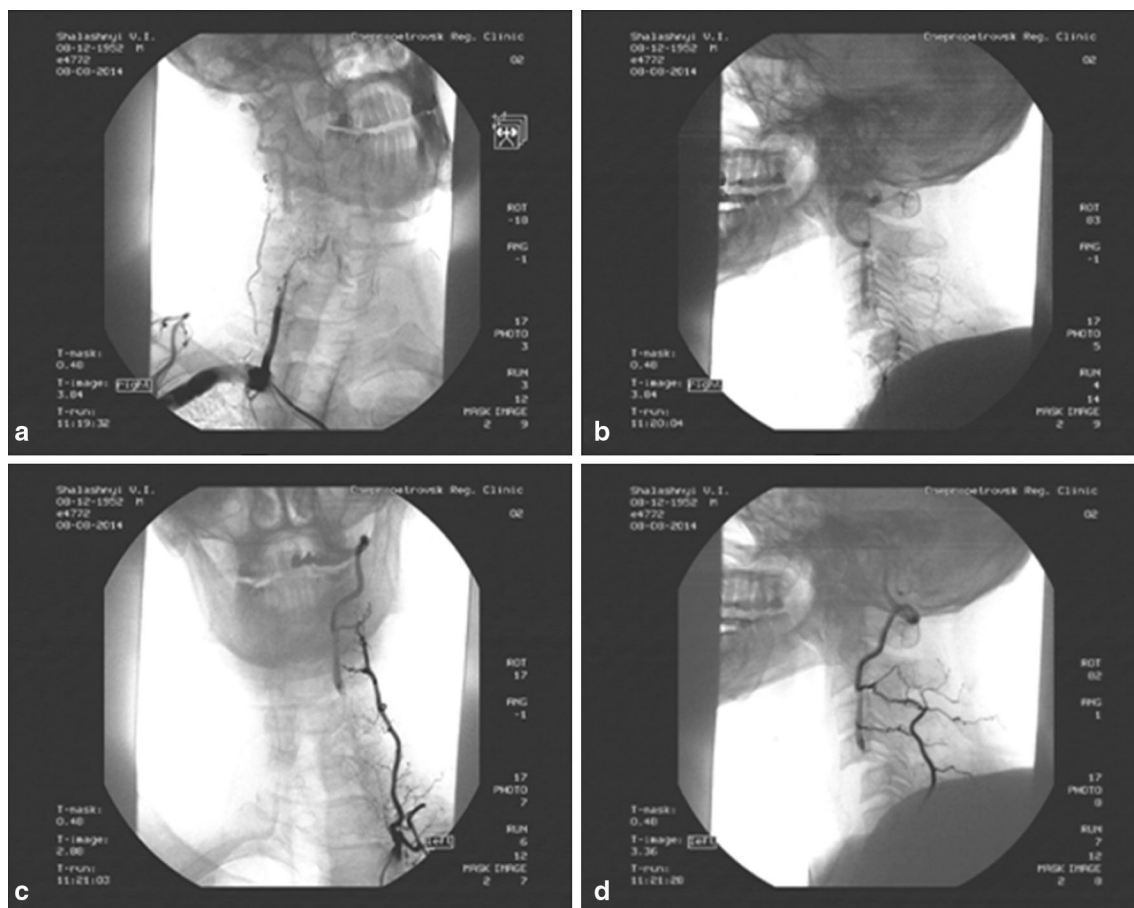


Fig. 2 Angiography of the vertebral artery. Dislocation fracture of C4–C5, posttraumatic occlusion of the vertebral artery. **a, b** System of the right vertebral artery. **c, d** System of the left vertebral artery

circulation was performed along both branches of the ascending cervical arteries that anastomosed with the vertebral arteries above the occlusion caused by the dislocation fracture, which prevented the progression of vertebrobasilar insufficiency. Collaterals progressed immediately after the injury, as evidenced by the clinical data (vertebrobasilar insufficiency is not noticeable during the first hours after injury).

Patient X, male, 62 years old. Diagnosis: spinal injury, with contusion of the spinal cord in the cervical region, dislocation fracture of C4–C5, and tetraparesis and ASIA scale D were observed at admission. No sensory disorders or disorders of pelvic organs were observed. On day 5 post-injury, magnetic resonance imaging (MRI) was performed, which identified a dislocation fracture of C4–C5, and myelopathy at the level of C4–C5 (Fig. 1).

On day 7, a total angiography was conducted, which revealed occlusion of both vertebral arteries: in the V2 segment on the right at the level of C6–C5 and from the V1–V2 segment up to the level of C4, with contrast enhancement above the occlusion on anastomoses of both



Fig. 3 X-ray of the spine. Dislocation fracture of C4–C5; an open reduction of dislocation and stabilization with the cage and plate were performed by means of front access; nitinol shape memory staples were placed at the C4 arc and C5 acanthi by means of posterior access (*lateral view*)

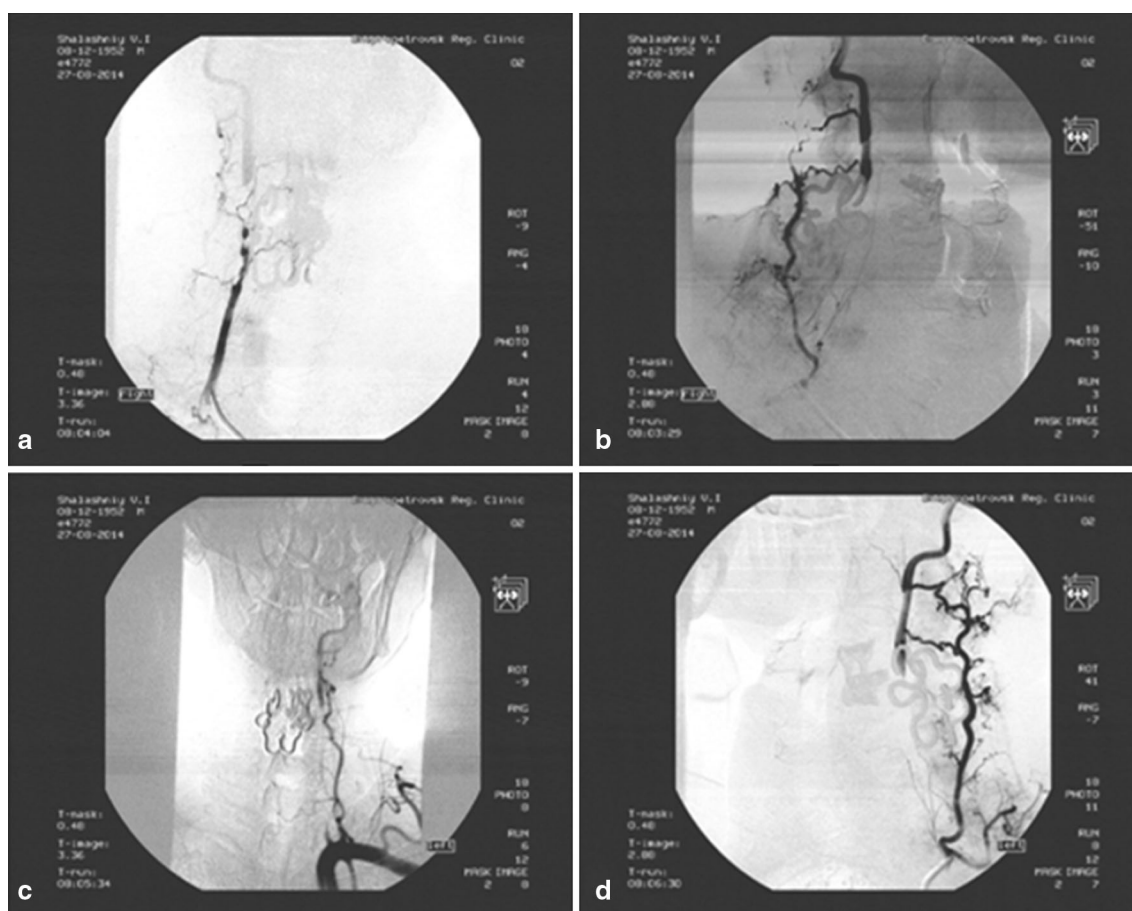


Fig. 4 Angiography of the vertebral artery. Dislocation fracture of C4–C5, with posttraumatic occlusion of the vertebral artery. **a, b** Right vertebral arterial system. **c, d** Left vertebral arterial system

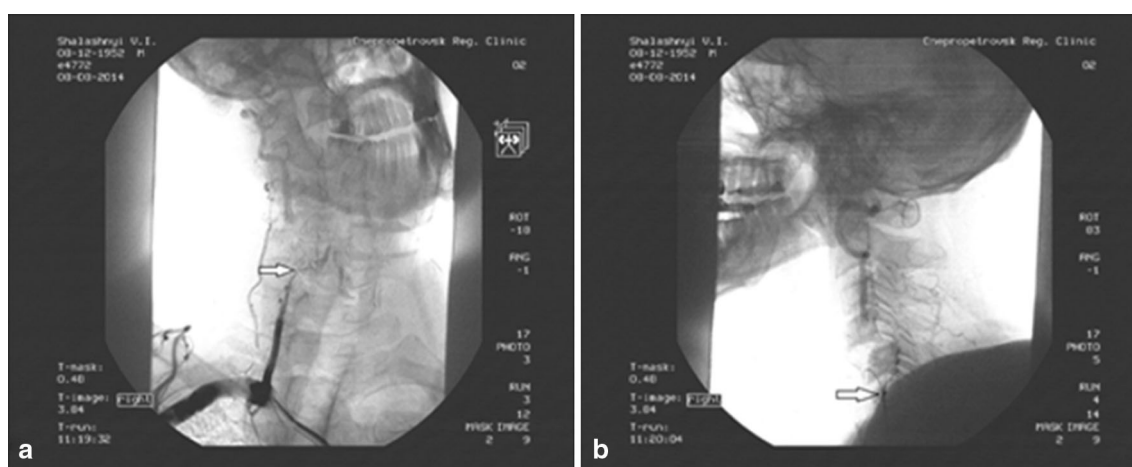


Fig. 5 Angiography of the vertebral artery. Dislocation fracture of C4–C5, with posttraumatic occlusion of the vertebral artery. **a, b** Right vertebral arterial system. The artery of cervical enlargement is marked with an *arrow*

ascending cervical arteries. The flow of blood was antegrade (Fig. 2).

On day 10, surgery was performed to correct the dislocation fracture of the C4–C5 vertebra by means of combined

access. Interbody corporodesis of C4–C5 was performed by a titanium cage transplant and plate by means of front access. Nitinol shape memory staples were placed at the C4 arc and the C5 acanthi by means of posterior access (Fig. 3).

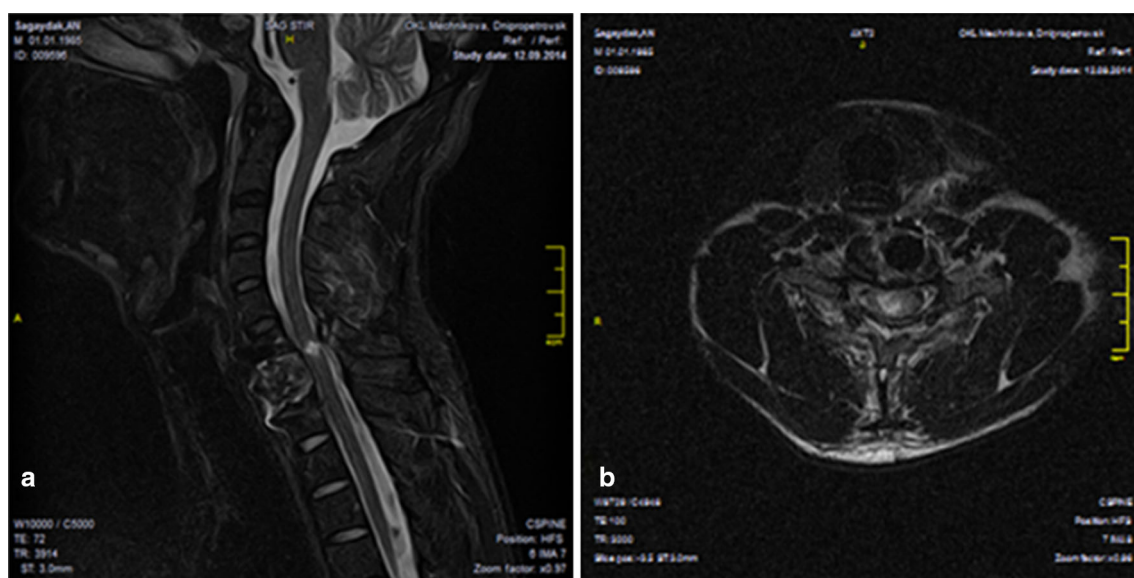


Fig. 6 Magnetic resonance images. Compression and comminuted fracture of C7, with myelopathy at the same level. **a** Sagittal view. **b** Axial view

Twelve days after surgery, a repeated angiography was conducted of the vertebral and cervical arteries on both sides. There was partial recanalization of the lumen of the right vertebral artery in the V2 segment at the level of C6–C5, spinal branches began to contrast at this level, and short occlusion of the right vertebral artery was seen above this level. Occlusion of the left vertebral artery occurred from the artery mouth, and good flows were observed at the anastomoses in the vertebral arteries of both ascending arteries. The flow of blood was anterograde (Fig. 4).

In addition, we established a pattern in the intensity of the spread of secondary spinal cord injury in the case of the damage to the artery of cervical enlargement. The patient experienced severe spinal cord compression, with a myelopathy focus (contusion) at the level of C4–C5, with sufficiently high neurological undamaged condition. During angiography, the artery of cervical enlargement, which was described by A. Lazort et al. as a major arterial branch in the form of a pin, which in this case extends from the proximal region of the occluded right vertebral artery, was revealed [6–8]. The presence of blood circulation in the artery of cervical enlargement allowed maintenance of blood flow in the spinal cord, which limited neurological impairment and extension of edema/ischemia in the regions of spinal cord located above and below the contusion focus (Fig. 5).

In Case 2, as well as Case 1, the artery of cervical enlargement is undamaged. MRI showed minimal extension of the zone of edema and ischemia. Neurological examination revealed tetraparesis (ASIA scale D). The



Fig. 7 Angiography of the vertebral artery. Compression and comminuted fracture of C7; corporodesis with a mesh cage and a plate. Left vertebral arterial system. The artery of cervical enlargement is marked with an arrow

patients had no changes in the neurological status after of treatment.

Case 2

Patient S, male, 29 years old. Diagnosis: spinal injury. Compression and comminuted fracture of the C7 body. Contusion of the spinal cord in the cervical region. Following injury, neurological function corresponded to ASIA scale D, which did not change throughout the study.

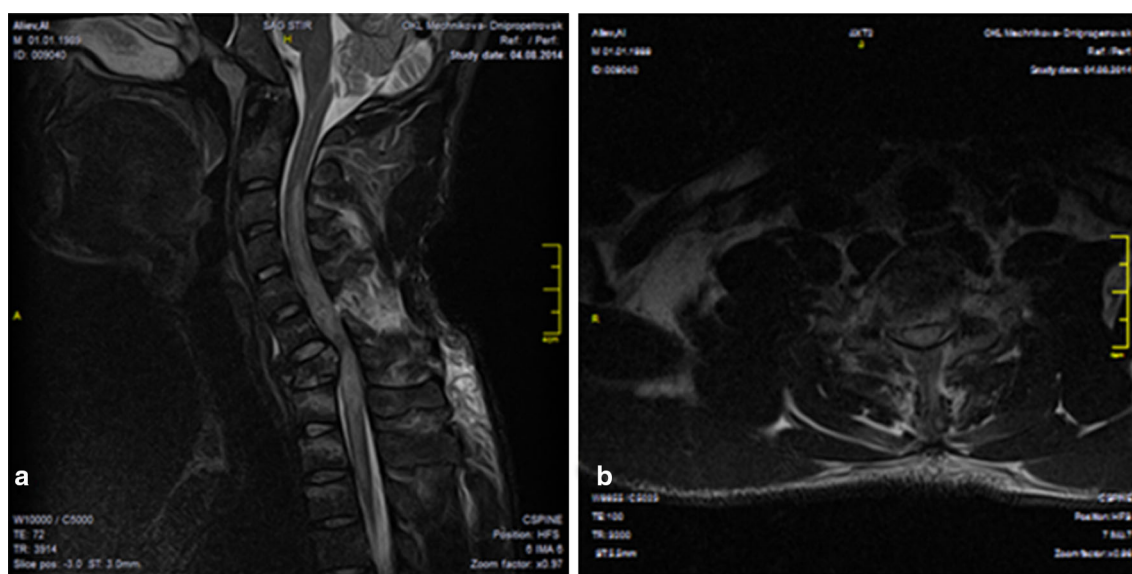


Fig. 8 Magnetic resonance imaging of Case 3. Compression and comminuted fracture of C7; myelopathy at the level of C7; an apparent zone of edema and ischemia. **a** Sagittal view. **b** Axial view

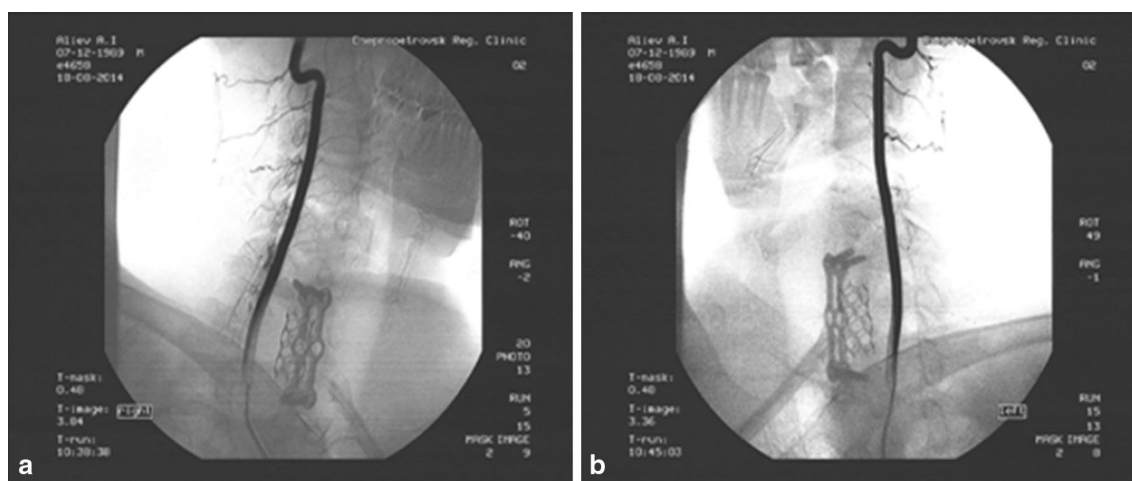


Fig. 9 Angiography of the vertebral artery. Compression and comminuted fracture of C7; corporodesis with a mesh cage and a plate. **a** Right vertebral arterial system. **b** Left vertebral arterial system. No artery of cervical enlargement

Surgery was performed on day 25 as follows: corpectomy of C7, followed by corporodesis with a mesh cage and plate. MRI of the cervical spinal cord and angiography of the vertebral arteries was performed during the postoperative period (Fig. 6).

The circulation of blood in vertebral arteries was maintained. The blood circulation in the artery of cervical enlargement maintained blood flow in the spinal cord, which limited neurological impairment and extension of edema-ischemia. The flow of blood was anterograde (Fig. 7).

Cases 3 and 4 show damage to the artery of cervical enlargement, following rupture or spasm of the branches of vertebral and ascending arteries, which is shown by the lack

of contrast enhancement of the artery of cervical enlargement and restriction of the vascular pattern in the branches of vertebral, ascending and deep cervical arteries. MRI showed an extensive area of ischemic edema of the spinal cord in the perifocal area of primary damage. There is persistent neurological impairment, with an ASIA scale score A. The patients had no changes in the neurological status after of treatment.

Case 3

Patient A, male, 25 years old. Diagnosis: spinal injury. Compression and comminuted fracture of the C7 body. Contusion of the spinal cord in the cervical region. ASIA scale A.

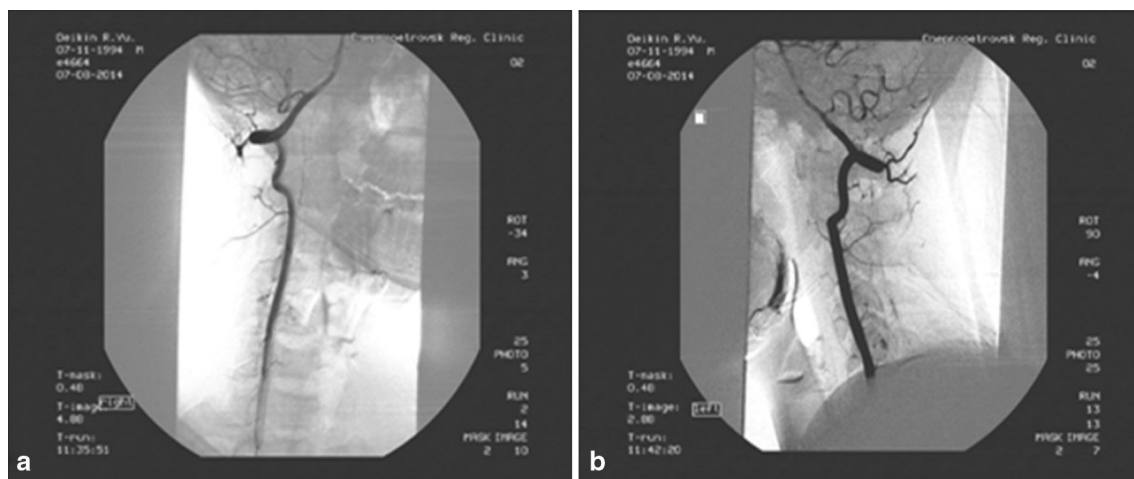


Fig. 10 Angiography of the vertebral artery. Compression and comminuted fracture of C5. **a** Right vertebral arterial system. **b** Left vertebral arterial system. No artery of cervical enlargement

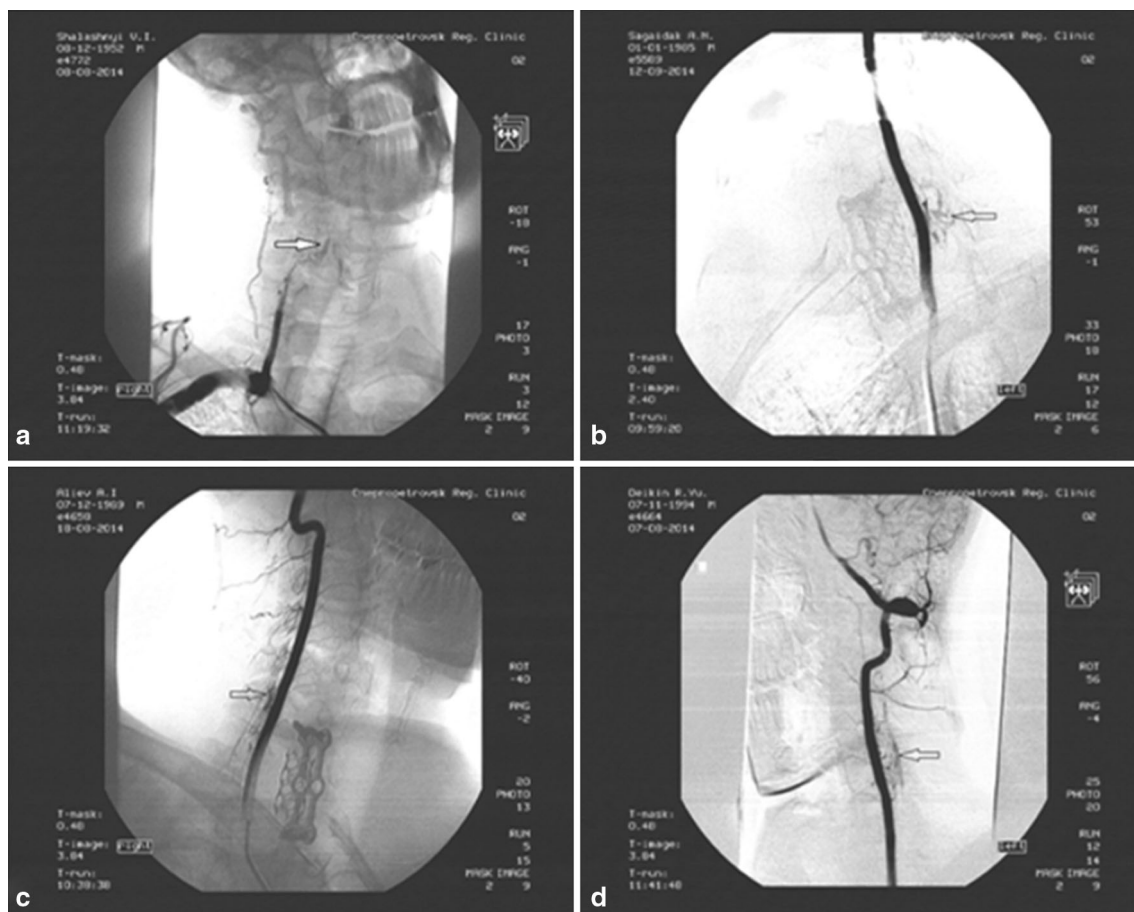


Fig. 11 a–d Cases 1–4. Early arteriovenous shunting is marked with an arrow

MRI of the cervical spinal cord was performed at day 4. An apparent zone of edema and ischemia was revealed (Fig. 8).

Surgery was performed on day 12 as follows: corpectomy of C7, followed by corporodesis with a mesh cage and a plate.

A total angiography was conducted on day 17. The circulation of blood in vertebral arteries was maintained. The artery of cervical enlargement is not visible; there is an accelerated arteriovenous shunting from the spinal branches of the vertebral and ascending cervical arteries on the right; and spinal branches are not enhanced on the left. The flow of blood was anterograde (Fig. 9).

Case 4

Patient D, male, 20 years old. Diagnosis: spinal injury. Compression and comminuted fracture of the C5 body. Contusion of the spinal cord in the cervical region. ASIA scale A.

Angiography was conducted on day 10 post-injury. The circulation of blood in vertebral arteries was maintained. The study revealed the absence of the artery of cervical enlargement; spinal branches were not enhanced. The flow of blood was anterograde (Fig. 10).

Surgery was performed on day 11: corporectomy of C5, followed by corporodesis with a mesh cage and plate.

We also noticed another phenomenon, which was the appearance of early arteriovenous shunting in the area of injury focus. As a result of reduction of perfusion in spinal cord injury, blood circulation in arterioles is disturbed. We believe that this is due to the compensatory inclusion of small anastomotic branches in the spinal cord.

This phenomenon was observed in all four cases (Fig. 11).

Conclusions

Collateral circulation is a significant factor in spinal cord injury, which limits the damage to the cervical spinal cord. Impaired circulation in the artery of cervical enlargement is

significant in the extension of perifocal ischemia. The appearance of early arteriovenous shunting in the region of a primary spinal cord injury (contusion focus) by angiography is pathognomonic. The data obtained in this study open a perspective for the endovascular treatment of spinal cord injury.

Conflict of interest None of the authors has any potential conflict of interest.

References

1. Anderson DK, Hall ED (1989) Pathophysiology of spinal cord trauma. *Ann Emerg Med* 22:987–992
2. Fehlings MG, Vaccaro AR, Boakye M et al (2013) Essentials of spinal cord injury: basic research to clinical practice. Thieme, New York
3. Sandler AN, Tator CH (1976) Review of the effect of spinal cord trauma on the vessels and blood flow in the spinal cord. *J Neurosurg* 45:638–646
4. Tator CH, Fehlings MG (1991) Review of the secondary injury theory of acute spinal cord trauma with emphasis on vascular mechanisms. *J Neurosurg* 75:15–26
5. Tator CH (1995) Update on pathophysiology and pathology of acute spinal cord injury. *Brain Pathol* 5(4):407–413
6. Lazorthes G et al (1962) La vascularisation de la moelle epiniere (etude anatomique et physiologique). *Rev Neurol* 106(6):535–557
7. Lazorthes G, Gouaze A, Zadeh JO, Santini JJ, Lazorthes Y, Burdin P (1971) Arterial vascularization of the spinal cord. *J Neurosurg* 35(September):253–262
8. Lazorthes G, Gouaze A, Djindjian R (1973) Vascularisation et circulation de la moelle epiniere, anatomie, physiologie, pathologie, angiographie. Masson & Cie, Paris