

# Cervical disc herniation as a trigger for temporary cervical cord ischemia

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**Background:** Disc herniations are only reported in few case reports as a rare cause of acute spinal ischemia. A surgical treatment has not been described so far in these reports with analysis of diffusion weighted magnetic resonance imaging (DWI/MRI) before and after surgery. The aim of our study is to report a case of cervical spinal cord ischemia caused by cervical disc herniation and discuss the literature concerning diagnostic and treatment options.

**Methods:** A 72-year-old female patient developed an acute progressive tetraparesis with emphasis on the upper extremities. MRI showed a disc herniation at the cervical segment 5/6 (C5/6) with consecutive spinal canal stenosis and additional signs of spinal cord ischemia in T2-weighted imaging (T2WI) and DWI reaching from C3 to C5 level. With the MRI being highly suggestive for anterior spinal cord ischemia, we hypothesized that this might be caused by compression of the anterior spinal artery through the significant disc herniation. Therefore, we decided to perform an anterior discectomy and fusion at C5/6 level.

**Results:** Following surgery, the patient's symptoms showed immediate regression with complete recovery after two months in correspondence with the normalization in the control MRI scan of cervical cord.

**Conclusions:** Assumedly our patient suffered from a partial anterior spinal artery syndrome, possibly caused by a disc herniation-related compression that was reversible following surgery. This was accompanied by a complete resolution of spinal cord signal abnormalities in T2WI and DWI.

**Keywords:** Spinal cord ischemia; diffusion weighted imaging (DWI); disc herniation; anterior cervical discectomy and fusion (ACDF)

Submitted Jun 15, 2016. Accepted for publication Jun 20, 2016.

doi: 10.21037/jss.2016.06.04

View this article at: <http://dx.doi.org/10.21037/jss.2016.06.04>

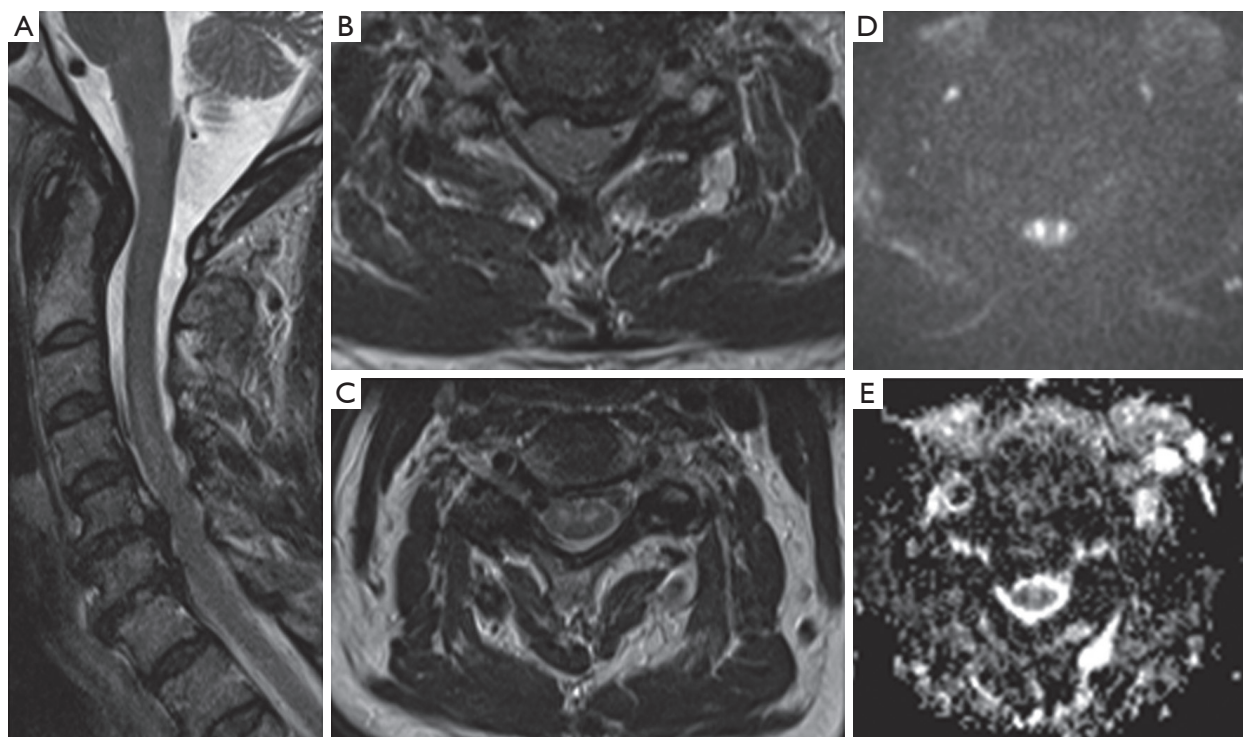
## Introduction

Cervical spinal cord ischemia is a rare disease that can be caused by various pathologies including vertebral artery dissection, epidural anesthesia and vasculitides (1). Compression by cervical disc herniation as a trigger for ischemia has rarely been reported in the literature (2,3). We report a case of temporary cervical cord ischemia presumably caused by cervical disc herniation. Although cervical disc herniations are frequently observed, there are only a few cases reported so far in which herniations were linked to consecutive spinal cord ischemia. We discuss the

relation between disc herniation and cervical spinal cord ischemia.

## Case presentation

A 72 year-old female patient presented to our hospital with acute progressive tetraparesis for five hours with emphasis on the upper extremities. The patient reported that she had suffered a sudden lancinating interscapular pain with radiation in both arms after a long period of kitchen work with flexed head. Her symptoms then progressed to asymmetrical weakness and sensory disturbance in both arms, so that



**Figure 1** Magnetic resonance imaging (MRI) study of the cervical spine performed about 12 hours after symptom onset. (A) Sagittal T2-weighted imaging (T2WI) shows linear hyperintensity within cord centered around C4 level and C5-6 disc extrusion impinging upon cord; (B) axial T2WI shows a left paramedian disc extrusion at C5-6 level; (C) axial T2WI shows increased signal involving gray matter of cord; (D) axial diffusion weighted imaging (DWI) shows restricted diffusion from central gray matter of cord at C4/5 level; (E) corresponding apparent diffusion coefficient (ADC) hypointensity from central gray matter of cord at C4/5 level.

she was admitted to our department. Her previous history includes hypertension and arthrosis.

Neurological examination revealed mild weakness of the upper arms (4/5 Medical Research Council Scale) associated with bilateral dysesthesia as well as left-sided hyperalgesia and disturbed thermal perception. The gait was slightly ataxic. In the course of diagnostics she developed mild hyperreflexia on the upper limbs and Babinsky sign on both sides.

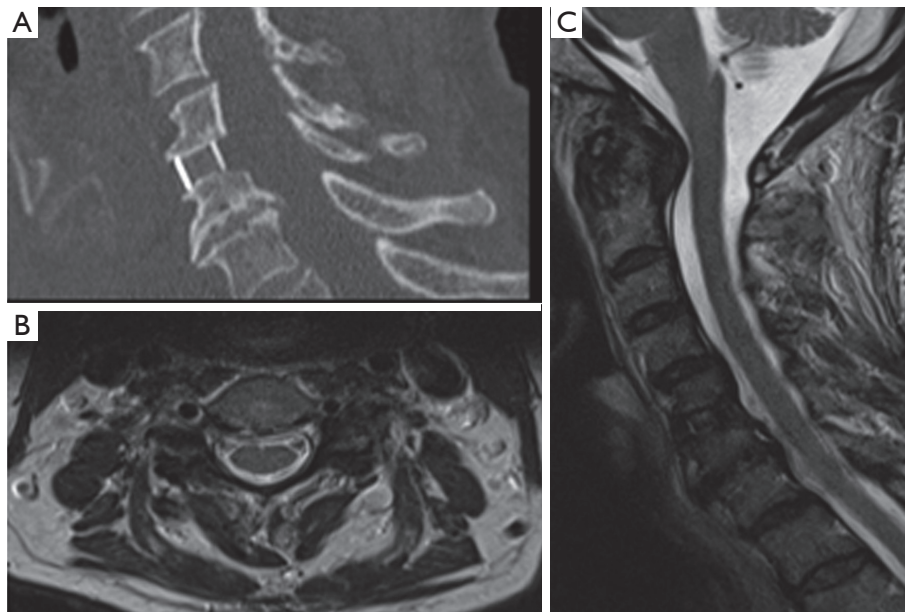
Magnetic resonance imaging (MRI) revealed a significant disc herniation at C5/6 with spinal canal stenosis (*Figure 1A,B*). T2WI showed an area of hyperintensity in the gray matter of the anterior cervical cord (*Figure 1C*). DWI revealed a corresponding diffusion restriction in respective sequences reaching from C3 to C5, indicating ischemia (*Figure 1D,E*). Computed tomography angiography (CTA) showed both vertebral arteries and the anterior spinal artery with proper continuity. Laboratory investigation showed no significant evidence of infection or inflammation. Although very rarely described, we assumed an anterior spinal cord ischemia by

compression through the disc herniation. As a rationale, we decided to perform an anterior discectomy and fusion at C5/6 level (*Figure 2A*). Additionally, we initiated steroid therapy (8 mg TID). Following surgery, the patient's symptoms showed immediate regression. Two months after surgery, the patient had recovered completely. A control MRI scan showed complete regression of signal abnormalities of the cervical spinal cord on T2WI and DWI (*Figure 2B,C*).

We hypothesized that our patient developed an anterior spinal artery syndrome which was likely caused by compression due to a disc herniation.

## Discussion

Spinal cord ischemia is a rare disease and usually located in the anterior spinal artery territory (4). The diagnostic modalities of this condition are still not standardized. Common MRI changes are described as "pencil-like"



**Figure 2** Magnetic resonance imaging (MRI) and computed tomography (CT) studies after surgery. (A) Postoperative CT imaging after anterior cervical discectomy and fusion (ACDF) at level C5/6; (B,C) follow-up MRI performed approximately 2 months after surgery. Axial (B) and sagittal (C) T2WI show complete resolution of signal abnormalities.

hyperintensity in T2WI with cord enlargement as well as contrast enhancement (1). However, in the acute phase these abnormalities might be missing (4). Weidauer *et al.* reported only 45% of the patients with acute spinal cord ischemia to have signal intensity changes on T2W images (5). Thus, the role of DWI and time course of the changes are still of major interest in spinal cord ischemia. Spinal DWI is technically more demanding than cranial DWI and is therefore not routinely used. As a consequence, the available literature includes one case report from Gass *et al.* [2000] and few small series of patients (1). A recent case report from Reynolds *et al.* also confirmed the usefulness of DWI in detecting ischemia of the thoracic spinal (6).

DWI changes in case of spinal ischemia have been reported to be detectable after a minimum of 3 to 4 hours after onset of symptoms; normalizing period has been described heterogeneously after approximately 7 to 28 days (7). Interestingly, apparent diffusion coefficient (ADC) values tend to be significantly lower in comparison to inflammatory myelopathies (8), so that ischemia can be differentiated from inflammation by MRI. It has been reported that after DWI normalization chronic spinal ischemia shows mostly atrophy of spinal cord and persisting T2WI hyperintensity (7).

In our case the neurological symptoms of our patient fit the anterior spinal artery territory with tetraparesis and

dissociated sensation deficits with a loss of temperature and pain sensation. MRI (12 hours after symptom onset) demonstrated “pencil-like” hyperintensity in T2WI with contrast enhancement. DWI showed restricted diffusion of the central gray matter as reported in the literature. In the CT-A the anterior spinal artery could be demonstrated with proper continuity. As laboratory parameters, including lumbar puncture, did not reveal any abnormalities suggesting an underlying inflammatory condition, we assumed cervical disc herniation as an underlying cause of spinal cord ischemia due to temporary compromise of vascular supply. The fact that our patient worked for quite a long time with flexed head in the kitchen at symptom onset might suggest a temporary compression of the arterial branches on the anterior surface of the spinal cord by the disc herniation, which was severe enough to cause hypoperfusion in the spinal cord. Normalization of DWI signal after surgery without signal abnormalities suggesting chronic ischemia such as atrophy or permanent T2 hyperintensity may be accounted as confirmation for temporary ischemia.

The rare existing previous reports of disc herniations and coexisting cervical or thoracic spinal cord ischemia do not describe treatment of the disc herniation by operative decompression due to evidence of cord ischemia (2,3,6).

Due to the sudden symptom onset with incomplete tetraparesis, we assumed that symptoms could be attributed to cord ischemia by cord compression from the disc herniation. We therefore performed emergency decompression by anterior cervical disc removal and fusion. The patient recovered quickly after surgery. No symptoms remained after 2 months. One may argue that our patient would also have recovered without surgery, but considering the risk of recurrent ischemia and the duration of long conservative therapy associated with longer hospitalization, we think that surgery is a considerable therapy option.

In conclusion, our patient suffered from the clinical signs of an anterior spinal artery syndrome, most probably caused by compression due to a disc herniation. The symptoms resolved completely after ACDF at the respective level. We recommend the consideration of surgical treatment in such cases instead of observation.

### Acknowledgements

None.

### Footnote

*Conflicts of Interest:* The authors have no conflicts of interest to declare.

*Informed Consent:* Written informed consent was obtained from the patient for publication of this case report and any

accompanying images.

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**Cite this article as:** Acker G, Schneider U, Grozdanovic Z, Vajkoczy P, Woitzik J. Cervical disc herniation as a trigger for temporary cervical cord ischemia. *J Spine Surg* 2016;2(2):135-138. doi: 10.21037/jss.2016.06.04