Glucocorticoid Induced Paraplegia in a Patient with Intracranial Dural Arteriovenous Fistula: Case Report

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Case Report

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Abstract

BACKGROUND:

Glucocorticoid inducing paraplegia has been reported and regard as a diagnostic clue for spinal dural arteriovenous fistulas (DAVFs). Intracranial DAVF in the posterior fossa draining into the perimedullary venous system can induce congestion of spinal cord while often be misdiagnosed and treated with steroid.

CASE PRESENTATION:

A 54-year-old woman presented progressive bilateral extremities weakness, bowel and bladder symptoms. A cervical MRI showed a longitudinally extensive spinal cord lesion from medulla oblongata to level of T4. A diagnose of NMO and cervical spondylopathy was made. On hospital day 2, methylprednisolone was prescribed at dose of 1g intravenous daily. After that, the patient experienced acute paraplegia. Treatment was stopped. And the patient improved on the hospital day 6, almost returned to her initial state. So the diagnosis of SDAVF was suspected. Spinal angiography was performed but the result was normal, and cerebral angiography demonstrated an DAVF fed by the right meningohypophyseal trunk and drain through right petrosal vein and into perimedullary venous system. Operation was performed successfully and the fistula was obliterated. The patient experienced an improvement of weakness and incontinence.

CONCLUSIONS:

We reported a case of intracranial DAVF with acute paraplegia response to intravenous glucocorticoid therapy. Intracranial DAVF can also lead venous congestion of the spinal cord, which induce similar clinical manifestation with spinal DAVF, the MRI examination of spinal cord can also find cord edema and typical enlarged medullary veins.

Background

It is presumed that venous hypertensive myelopathy is the pathophysiology of spinal dural arteriovenous fistulas (DAVFs). Intracranial DAVFs with perimedullary venous drainage which are very rare lesions can also lead to myelopathy and are like spinal DAVFs. The clinical presentation is nonspecial and include gait disturbances and sensory disorder, MRI can indicate longitudinally extensive parenchymal signal abnormalities which are similar to transverse myelitis, or neuromyelitis optica (NMO), so misdiagnose are often made and glucocorticoid therapy is performed. Acute paraplegia induced by glucocorticoid treatment in patients with spinal DAVFs has been reported, the similar phenomenon can also happen in intracranial DAVFs with perimedullary venous drainage which has not been reported.

Case Presentation
A 54-year-old woman, presented progressive bilateral extremities weakness, bowel and bladder symptoms for one month. Muscle strength of bilateral upper limbs were grade 4 and lower limbs were grade 3. Lumbar puncture was performed on the first day of hospitalization, the pressure of cerebrospinal fluid was 215 mmH2O, and contained 3 leukocytes, 28 red blood cells and 431mg/L of protein. A cervical MRI showed a longitudinally extensive spinal cord lesion from medulla oblongata to T4. A diagnose of NMO and cervical spondylopathy was made. Then on the second day of hospitalization, methylprednisolone was prescribed at dose of 1g intravenous daily diluted in 500ml of saline solution. After the first day of treatment, the patient complained of urinary retention, with worsen weakness of four limbs, muscle strength of upper limbs decreased to grade 3, and lower decreased to grade 2. Treatment was stopped. However, the muscle strength of lower limbs decreased to grade 1 on the fourth day of hospitalization, but the patient improved on the sixth day, almost returned to her initial state. The treatment was suspended, and diagnosis of spinal DAVF was suspected. Then the patient (wheelchair-bound) presented to our department. Radiologic review revealed abnormal flow voids and on the surface of cervical cord and intramedullary high signal. (figure 1) Spinal angiography was performed but the result was normal, and cerebral angiography demonstrated an DAVF fed by the right meningohypophyseal trunk and drain through right petrosal vein and into perimedullary venous system. (figure 2) Because of the winding vessels, the fistula point was not accessible to endovascular treatment, and the obliteration of the DAVF was obtained by microsurgical treatment through suboccipital retrosigmoid approach. Postoperatively, the patient experienced an improvement of weakness and incontinence. The control angiography showed that the DAVF disappeared, (Figure 3) and MRI indicated a remission of the cord edema.

Discussion And Conclusion

To our knowledge, this is the first report about a case of glucocorticoid induced paraplegia in patient with intracranial DAVF with perimedullary drainage. A retrospective study has suggested intravenous methylprednisolone could cause immediate worsening of motor and sensory symptoms in patients with spinal DAVF2. In our case which was not spinal DAVF but intracranial DAVF draining into perimedullary venous system, we also found the worsening of paraplegia with glucocorticoid. The similar phenomenon happened to patient with spinal DAVF was thought to be caused by the rapid infusion of saline solution with glucocorticoid which could result in an increase in volemia and venous pressure3. This may also occur in patients with intracranial DAVF, as long as the drainage is into perimedullary venous system which results venous congestion of the spinal cord. This kind of DAVF, of which the pathophysiological mechanism is similar with that of spinal DAVF, would be located at petrous region, tentorium or foramen magnum4. Although experienced neurologists will consider the diagnosis of spinal DAVF when face the typical abnormal void signals on MRI, intracranial DAVF can also lead to spinal myelopathy which is really rare but also should be considered. If a patient experienced acute paraplegia following glucocorticoid administration and no abnormality was found on spinal angiography, cerebral angiography would be necessary. We hope our case will inform the clinicians about the appropriate administration of glucocorticoid in patients with spinal DAVFs and intracranial DAVFs with perimedullary venous drainage.
It is important to recognize that intracranial DAVFs can also cause spinal myelopathy. Considering of worsening of myelopathic symptoms following intravenous glucocorticoid, the diagnosis of spinal DAVFs or intracranial DAVFs should be suspected.

**Declarations**

**Ethics approval and consent to participate**

Not applicable

**Consent for publication**

A written informed consent was obtained from the patient for publication of this Case Report and any accompanying images. A copy of the written consent is available for review by the corresponding author.

**Availability of data and material**

All data analyzed during this study are included in this article.

**Competing interests**

The authors declare that they have no competing interests.

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**Authors’ contributions**

Yongjie Ma substantial contribution to data collection and drafted the manuscript. Jie Lu was responsible for imagine analysis. Hongqi Zhang drafted the manuscript and made substantial contribution to manuscript revision. All the authors have read and approved the manuscript for publication

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**Figures**

![Figure 1](image1.png)

**Figure 1**

Sagittal T2-weighted MRI showed abnormal vascular flow voids around cervical cord [arrowhead] and intramedullary hyperintensity [arrow] (A), Isointensity on T1-weighted MRI (B), Intramedullary enhancement at C2-5 level on post-gadolinium T1-weighted (C).
Figure 2

Right internal carotid artery (ICA) on cerebral DSA showed the fistulas draining through the superior petrosal vein[arrowhead] into the perimedullary venous system[arrow], anteroposterior view (A) and lateral view (B).
Figure 3

Right ICA on cerebral DSA showed elimination of the fistula, anteroposterior view (A) and lateral view (B).

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