Neurological Sciences (2019) 40:2411–2414
https://doi.org/10.1007/s10072-019-03940-1

LETTER TO THE EDITOR

Glucocorticoid induced acute paraplegia in a patient with intracranial dural arteriovenous fistula

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Received: 13 March 2019 / Accepted: 13 May 2019 / Published online: 21 May 2019

Dear Editor,

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 the spinal cord while often be misdiagnosed and treated with steroid. It is presumed that venous hypertensive myelopathy is the pathophysiology of DAVFs. Intracranial DAVFs with perimedullary venous drainage which are very rare lesions can also lead to myelopathy and are like spinal DAVFs [1]. The clinical presentation is nonspecial and includes gait disturbances and sensory disorder; MRI can indicate longitudinally extensive parenchymal signal abnormalities which are similar to transverse myelitis, or neuromyelitis optica (NMO), so misdiagnoses are often made and glucocorticoid therapy is performed. Acute paraplegia induced by glucocorticoid treatment in patients with spinal DAVFs has been reported, a similar phenomenon can also happen in intracranial DAVFs with perimedullary venous drainage.

A 54-year-old woman presented progressive bilateral extremity weakness and bowel and bladder symptoms for 1 month. Muscle strength of bilateral upper limbs was grade 4 and of lower limbs was grade 3. Lumbar puncture was performed on the first day of hospitalization; the pressure of cerebrospinal fluid was 215 mm H_2O, and contained 3 leukocytes, 28 red blood cells, and 431 mg/L of protein. A cervical MRI showed a longitudinally extensive spinal cord lesion from the medulla oblongata to T4. A diagnosis of NMO and cervical spondylopathy was made. Then, on the second day of hospitalization, methylprednisolone was prescribed at a dose of 1 g intravenous daily diluted in 500 ml of saline solution. After the first day of treatment, the patient complained of urinary retention, with worsening 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 on the surface of the cervical cord and intramedullary high signal intensity (Fig. 1). Spinal angiography was performed but the result was normal, and cerebral angiography demonstrated a DAVF fed by the right meningohypophyseal trunk and drain through the right petrosal vein and into the perimedullary venous system (Fig. 2). Because of the winding vessels, the fistula point was not accessible to endovascular treatment, and the obliteration of the

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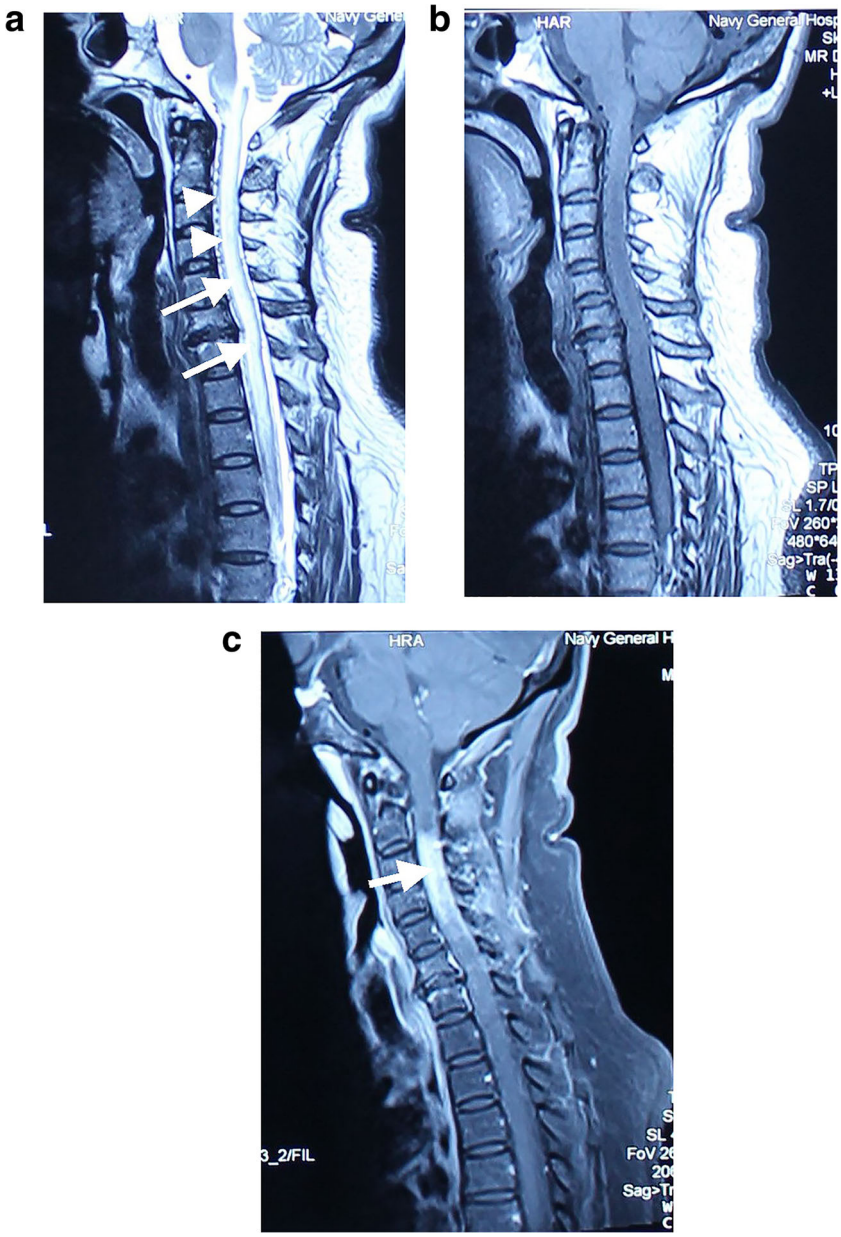

Fig. 1 Sagittal T2-weighted MRI showed abnormal vascular flow voids around the cervical cord (arrowhead) and intramedullary hyperintensity (arrow) (**a**). Isointensity on T1-weighted MRI (**b**). Intramedullary enhancement at C2-5 level on post-gadolinium T1-weighted (**c**)

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 (Fig. 3), and MRI indicated a remission of the cord edema.

Discussion

A retrospective study has suggested intravenous methylprednisolone could cause immediate worsening of motor and sensory symptoms in patients with spinal DAVF [2]. In our case which was not spinal DAVF but intracranial DAVF draining into the perimedullary venous system, we also found the worsening of paraplegia with glucocorticoid. The similar phenomenon happened to a 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 pressure [3]. This may also occur in patients with intracranial DAVF, if the drainage is into the perimedullary venous system which leads to venous congestion of the spinal cord [4]. This kind of DAVF, of which the pathophysiological mechanism is similar with that of spinal DAVF, would be located at the petrous region, tentorium, or foramen magnum [5]. Although experienced neurologists will consider the

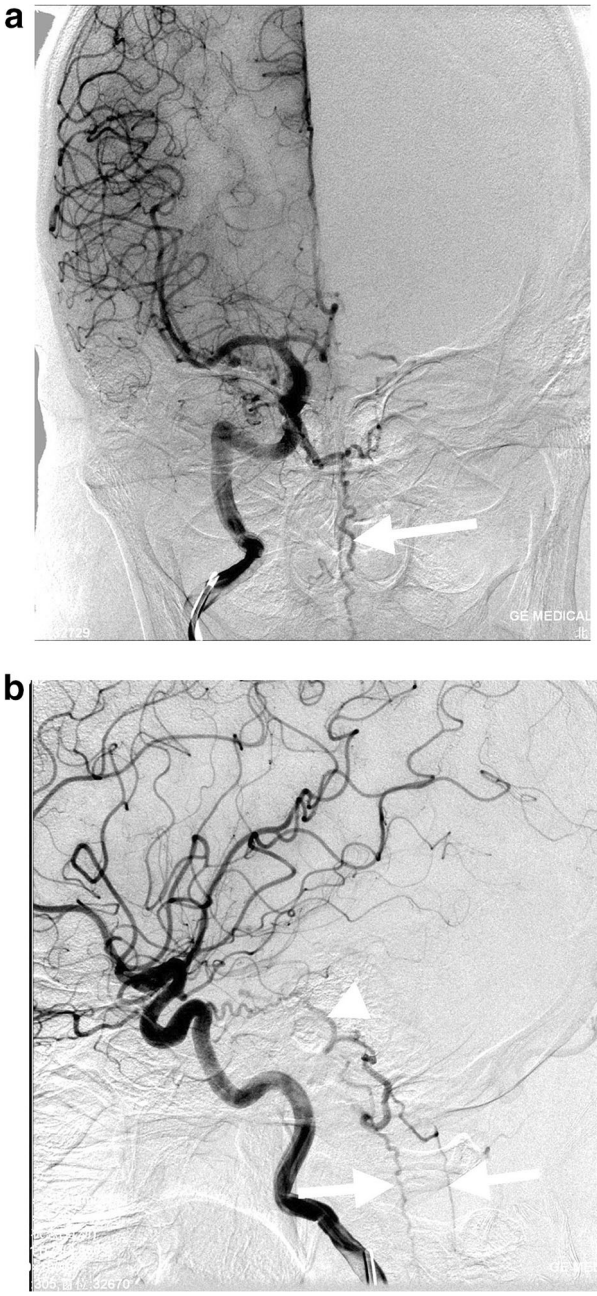

Fig. 2 Right internal carotid artery (ICA) on cerebral DSA showed the fistulas draining through the superior petrosal vein (arrowhead) into the perimedullary venous system (arrow). Anteroposterior view (**a**) and lateral view (**b**)

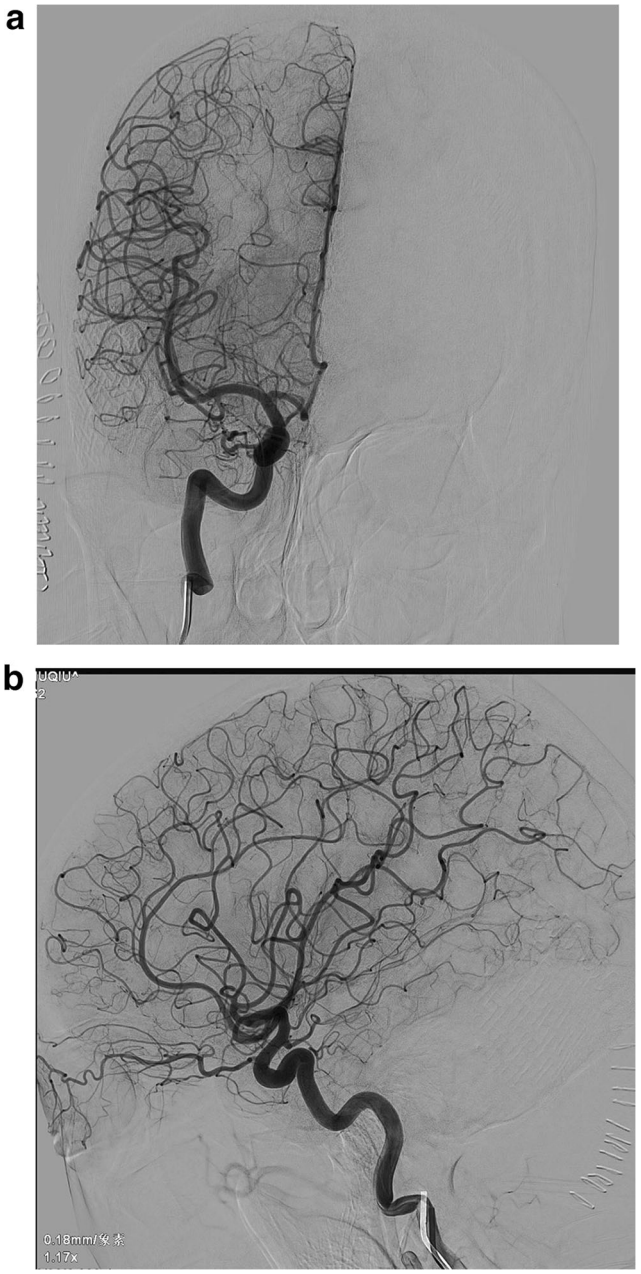

Fig. 3 Right ICA on cerebral DSA showed elimination of the fistula. Anteroposterior view (**a**) and lateral view (**b**)

diagnosis of spinal DAVF when face the typical abnormal void signals on MRI, intracranial DAVF can also lead to spinal myelopathy which is 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 worsening of myelopathic symptoms following intravenous glucocorticoid, the diagnosis of spinal DAVFs or intracranial DAVFs should be suspected.

Funding This work was supported by the National Natural Science Foundation of China (Award Number: 81671202) and Beijing Municipal Science and Technology Commission (Award Number: D161100003816001).

Compliance with ethical standards

This work was approved by the Institutional Ethics Committees.

Conflict of interest The authors declare that they have no conflict of interest.

Abbreviations DAVF, dural arteriovenous fistula; MRI, magnetic resonance imaging; NMO, neuromyelitis optica; T1WI, T1-weighted imaging; T2WI, T2-weighted imaging

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