A man in his 60s was evaluated for progressive myelopathy. His symptoms evolved insidiously over a year and included groin numbness, incomplete bladder emptying, and leg weakness. Examination revealed spastic paraparesis. Magnetic resonance imaging (MRI) of the cervical and thoracic spine demonstrated a T2 hyperintense lesion extending from T6 to the conus but no evidence of abnormal flow voids over the surface of the cord and no evidence of gadolinium enhancement. Magnetic resonance imaging of the brain was normal. His leg weakness worsened after walking and improved with rest. The following blood results were negative or normal: vitamin B₁₂, copper, West Nile virus, human T-cell lymphotropic virus types 1 and 2 antibodies, human immunodeficiency virus antibodies, methylmalonic acid, Lyme serology, erythrocyte sedimentation rate, lupus panel, antineutrophil cytoplasmic antibody, and VDRL. A lumbar puncture revealed normal findings. The spinal cord appeared normal; spinal cord biopsies were nondiagnostic. One month later, he was treated again with methylprednisolone. As before, approximately 1.5 hours after the infusion, he experienced acute paraplegia, which resolved after 48 hours. After this episode, his bladder function worsened and he required self-catheterization. His gait further deteriorated such that he required a wheelchair. A positron emission tomographic scan to detect systemic sarcoidosis or other inflammation yielded negative results.

He was referred to our institution. His medical history confirmed these details; symptoms were confined to the legs; and he had no pain and no upper extremity symptoms. He had severe sensory ataxia and spastic paraparesis, right worse than left; he was incontinent of bladder and bowel. There was a sensory level at approximately T12 affecting all sensory modalities.

Considering the worsening of myelopathic symptoms after walking and improvement at rest and worsening following intravenous corticosteroid, the working diagnosis was spinal dural arterial venous fistula (SDAVF) despite the previous negative angiogram result. A magnetic resonance angiogram showed dilatation of posterior pial venous plexus associated with cord expansion and intramedullary edema (Figure, A), strongly suggesting the presence of venous hypertension. An SDAVF originating from the right L2 pedicle was identified.

Figure. Radiologic Evidence of Spinal Dural Arterial Venous Fistula Ablation

A, Sagittal T2-weighted image of the thoracic spine demonstrates mild cord expansion associated with intramedullary hyperintensity extending into the conus (arrowhead). B, Angiogram demonstrating L2 spinal dural arterial venous fistula (arrowheads). C, Sagittal T2-weighted image of the thoracic spine 6 months following fistula ablation showing markedly diminished signal abnormality (arrowhead).
by catheter angiography (Figure B). The patient underwent L2 hemilaminectomy and ligation of SDAVF. At 6-month follow-up, he was able to walk with crutches; however, bladder function was unchanged. Magnetic resonance imaging showed marked improvement in intramedullary edema (Figure, C).

**Discussion** | The diagnosis of SDAVF is difficult because its presenting features are similar to those seen with other neurological disorders and its radiological identification is challenging. Therefore, there is often a delay to diagnosis of SDAVF; however, disability is highly preventable when recognized and treated early.1

An SDAVF is an abnormal fistulous connection between the arterial and the venous system, which enables the flow of high-pressure spinal arterial blood into a spinal vein resulting in venous hypertension. This arterialization of the venous system compromises the normal antegrade venous drainage and results in edema, infarction, and myelopathy.2 Acute neurologic exacerbations related to SDAVF have been reported with conditions that cause increased venous hydrostatic pressure such as valsalvae, exercise, singing, and more rarely with corticosteroids (Table).3

The proposed mechanism of corticosteroid-induced exacerbation of weakness is transient fluid retention, further exacerbated when administered with a saline infusion, leading to venous engorgement and further impairment of venous egress from the dural shunt. This exacerbates spinal cord edema, resulting in neurological worsening, which may resolve as the corticosteroid-induced hypervolemia wanes.

**Conclusions** | Transient paraplegia after receiving corticosteroids for a myelopathy associated with a conus lesion should prompt a search for SDAVF even in the absence of MRI evidence for dilated pial veins on MRI.

**Conflict of Interest Disclosures:** None reported.


**COMMENT & RESPONSE**

**Factors Contributing to Post–Lumbar Puncture Headache**

**To the Editor** Factors associated with post–lumbar puncture headache occurrence have been thoroughly highlighted by Monserrat et al.1 The use of a smaller-tipped needle can decrease the incidence of headache (the protocol used in the study1) provided that the number of punctures does not increase. Multiple punctures probably increase the incidence of headaches. If use of a smaller needle increases the number of punctures, the difference between small and large needles in producing headaches may be reduced. Therefore, the expertise of the person performing the puncture and the number of attempts are of importance; these seem to have been overlooked. Other important, yet missing, factors are pregnancy and needle bevel placement axis. The incidence of post-lumbar puncture headache increases with pregnancy, and it occurs less frequently when the needle bevel is placed parallel with the length of the neuraxis during insertion.2,3

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