A Rare Cause of Radiculomyelitis
Dural Arteriovenous Fistula

Ahmet K. Kilic, MD; Asli T. Kurne, MD; Isil Saatci, MD; Ersin Tan, MD

REPORT

Dural arteriovenous fistula is a very rare cause of myelitis that can only be treated interventionally or surgically.

OBSERVATIONS
A man in his 30s with paraparesis and urinary incontinence had a long-segment thoracic lesion on spinal magnetic resonance imaging. Transverse myelitis was the initial diagnosis. Although a pulse steroid and intravenous immunoglobulin treatment regimen was given, no definite clinical response was seen. A spinal angiogram was performed in our center demonstrating right T7 to T8 spinal dural arteriovenous fistula. The fistula was occluded with embolization and the patient showed recovery following the endovascular treatment.

CONCLUSIONS AND RELEVANCE
Myelitis usually is known to respond well to immunosuppressive treatments. Despite adequate medical treatment, if slow progression is seen in the follow-up clinically and radiologically, dural arteriovenous fistulas should be kept in mind in the etiopathogenesis.

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pinal dural arteriovenous fistulas occur rarely; they are venous shunts between 1 or more dural arteries and the medullary venous system.1,2 Usually the fistula originates from a segmental radicular artery.1 It causes a slowly progressive myelopathy1,2 and can mimic even a radiculopathy.3 Most of them are localized in the thoracolumbar region.2,3

Report of a Case

A man in his 30s presented with a 5-year history of lumbar pain. He was taking nonsteroidal anti-inflammatory drugs and myorelaxants. Two months before admission to the hospital, he felt paresthesias in his feet. Sharp and acute lumbar pain started suddenly in addition to his chronic pain. He had progressive numbness and weakness, starting from his left lower extremity to both legs. He had ataxia and difficulty in walking. Urinary incontinence developed at clinical onset and urinary retention progressed in the clinical follow-up.

In his first spinal magnetic resonance imaging (MRI) examination in an outside institution, there was T2 hyperintensity in the spinal cord from the T6 level to the conus (Figure 1A) and contrast enhancement around the cord was noted. Transverse myelitis was considered as the initial diagnosis at that time. After 5 days of intravenous pulse steroid treatment, he had made no apparent clinical recovery and intravenous immunoglobulin was added to the treatment scheme but did not show any additional benefit. He was referred to our center and 4/5 spastic paraparesis, saddle-type hypoesthesia, and the decrease of anal sphincter tonus were noted at admission. Deep tendon reflexes were brisk. Spinal MRI, including the sacral region, was repeated showing obvious enhancing tubular structures around the cord and within the thecal sac (Figure 1B). Negative results were obtained for hemogram, sedimentation, C-reactive protein, hepatitis markers, vasculitis markers, and human immunodeficiency virus. A lumbar puncture revealed a cerebrospinal fluid protein level at 53 mg/dL and glucose level at 59 mg/dL. Mature lymphocytes were seen on cerebrospinal fluid cytology. Oligoclonal band was negative. The IgG index was 0.58. The results from tests for Brucella, syphilis, cerebrospinal fluid/serum angiotensin-converting enzyme level, tuberculosis polymerase chain reaction, herpes simplex virus, Epstein-Barr virus, varicella-zoster virus, and Borrelia serology were negative.

Based on the spinal MRI findings (ie, long-segment intramedullary signal change accompanied by an appearance of possible abnormal vessels), diagnosis of spinal vascular malformation was considered and a spinal angiogram was performed. Spinal digital subtraction angiography revealed a dural arteriovenous fistula filling from the right T7 and T8 intercostal artery injections. The patient was treated with embolization under general anesthesia and totally occluded with n-butyle-2-cyanoacrylate (glue) following the superselective catheterization of the fistula site from the radicular branch of the right T7 intercostal artery (Figure 2). Follow-up MRI at 9 months showed the regression of myelopathy and the resolution of gadolinium enhancement (Figure 1C and D). The patient showed clinical recovery with minimal sequelae.

Author Affiliations: Graduate School of Health Sciences, Medical School of Hacettepe University, Ankara, Turkey (Kilic); Department of Neurology, Medical School of Hacettepe University, Ankara, Turkey (Kurne, Tan); Department of Radiology, Koru Hospitals, Ankara, Turkey (Saatci).

Corresponding Author: Ahmet K. Kilic, MD, Graduate School of Health Sciences, Medical School of Hacettepe University, Ankara, Turkey (kasimkilic@gmail.com).
Conclusions

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