

Case Report

Transverse Myelitis Caused by Spinal Dural Arteriovenous Fistula: A Case Report

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Email: bpeterson@whs.org**Received:** May 13, 2023**Accepted:** June 07, 2023**Published:** June 14, 2023**Abstract**

Spinal cord pathology is a diverse topic with a broad range of causes. Etiologies include trauma, inflammatory processes, infections, vascular disease, neoplasm, degenerative conditions and toxin exposure. Regardless of etiology, spinal pathology often presents as rapidly progressive paraparesis. Transverse Myelitis (TM) is a rare, acute segmental inflammatory spinal cord disorder characterized with motor, sensory, and/or autonomic dysfunction. This process usually occurs post-infection, but may also be related to acute neuro-inflammatory disorders. This paper will present a case of TM refractory to medication and describe the diagnosis of Spinal Dural Arteriovenous Fistula (SDAVF). SDAVF is a rare condition in which there is an arteriovenous fistula formed on the dural surface of the spinal cord which typically drains via retrograde flow through the medullary vein, thereby causing enlargement of coronal venous plexus.

Keywords: Transverse myelitis; Spinal cord pathology; Arteriovenous fistula

Case Presentation

42-year-old male with past medical history of Wolff-Parkinson White (WPW) Syndrome status post ablation presented to his primary care physician's office with three weeks of progressively worsening bilateral lower extremity weakness, urinary hesitancy, loss of bowel function, and decreased sensation with sexual intercourse. Symptoms started with low back pain that radiated to his hamstrings. Exam was pertinent for 4/5 strength in left lower extremity, reflexes 3+ and decreased sensation to sharp and dull touch on the bilateral shins. On initial presentation, lumbar puncture revealed elevated protein and no oligoclonal bands. Magnetic-Resonance-Imaging (MRI) lumbar spine revealed an ill-defined T2 hyperintensity in the conus medullaris that did not appear expanded or deformed, which could be indicative of demyelinating disease. Serological testing for infectious, neurological, rheumatological, toxic, and metabolic causes were all negative with the exception of Antinuclear Antibody (ANA). Complete Blood Count (CBC) was positive only for leukocytosis. During admission, patient developed progressively worsening symptoms including asymmetric hyperreflexia, complete urinary retention, and increasing subjective numbness of lower extremities. MRI brain showed T2 bright focus in

corona radiata, not strongly diagnostic for multiple sclerosis. He received three doses of high-dose IV methylprednisolone as well as one dose of IV dexamethasone, after which he became unable to stand on toes and developed difficulty with balance. He was transitioned to high dose Intravenous Immunoglobulin (IVIg). After 4 days of high dose IVIg, he developed headaches, hypertensive urgency, worsening right lower extremity weakness and a wide-based, ataxic gait. MRI dorsal spine revealed hyperintensity of the spinal cord from T6 to conus medullaris suggestive of TM. No abnormal postcontrast enhancement was noted, although there were flow voids indicating accumulation of fluid. Concern was raised for reaction to IVIg versus TM refractory to medications. He was transferred to a tertiary care center. Myelin oligodendrocyte glycoprotein antibody (anti-MOG) ordered at presentation due to suspicion for multiple sclerosis came back negative after transfer. At the tertiary care center, flow voids indicating drainage defect seen on MRI suggested the presence of a spinal dural AV fistula; T8 laminectomy was consequently performed. With intensive physical therapy, the patient is expected to make a full recovery.

Discussion/Conclusion

Transverse Myelitis (TM) is characterized by spinal cord dysfunction including paresis at the level of injury, weakness, and autonomic impairment, such as bowel or bladder incontinence and sexual dysfunction. It is suspected that TM is not purely a demyelinating disease, but can be associated with inflammatory disorders or autoimmune conditions, such as Multiple Sclerosis (MS) or Myelin Oligodendrocyte Glycoprotein Antibody Disease (MOGAD). Most cases of TM are thought to be idiopathic with rapid weakness, sensory changes, and dysfunction of the bowel and bladder. TM is diagnosed by MRI showing T2 hyperintense signaling. This correlates to inflammation as seen by increased cell counts in Cerebrospinal Fluid (CSF). Treatment consists of high dose IV steroids or IVIG. Spinal Dural Arteriovenous Fistula (SDAVF) is a rare spinal cord disorder, although it is the most common spinal vascular malformation. Fistulas between a radicular artery and a radicular vein in the dura of the spinal column cause a venous outflow deficit that can eventually lead to myelopathic symptoms. The diagnosis can be difficult to differentiate from other myelopathies and can go misdiagnosed due to the nonspecific symptoms. SDAVF has a male predominance and typically affects the thoracolumbar region. MRI imaging can corroborate diagnostic findings and typically show T2 hyperintensities with involvement of the conus medullaris or spinal cord, cord contrast enhancement, or T2 flow voids caused by venous congestion. Although lumbar puncture has been used to aid diagnosis, it is nonspecific and can point toward an inflammatory or infectious process. Treatment consists of laminectomy and endovascular embolization.

References

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