Neuroimaging Highlight

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Congenital Ventral Thoracic Spinal Cord Herniation

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A 53-year-old woman presented with progressive bilateral leg weakness that was managed conservatively over the past eight years. The patient had no history of trauma. The physical examination revealed severe gait impairment, a dense spastic paraparesis, pyramidal distribution weakness, positive Babinski signs bilaterally and hyperreflexia in the lower limbs. The patient had preserved proprioception and sensation in the lower limbs. Magnetic resonance imaging of the spine revealed a congenital ventrally displaced spinal cord with focal herniation through the dura at the T4-T5 level (Figure 1). This corresponded to a type K spinal cord herniation which is characterized by kinking.[1] The thoracic myelopathy was likely due to cord tethering and ischemia. The patient underwent a T4-T5 laminectomy with a left T5 transpedicular approach for an intra/extra-dural microscopic repair of the defect. Intraoperatively, there was ventral kinking of the spinal cord with a complex anterior herniation through a dural defect of gliotic tissue which was also tethered anteriorly to the posterior longitudinal ligament. To allow de-tethering of the spinal cord, the dura was ventrally marsupialized using a wide opening, as a primary dural repair was not deemed to be feasible. At six month postoperative follow-up, the patient had shown clinical signs of neurological improvement with reduced lower limb spasticity and improved gait.

Spontaneous herniation of the thoracic spinal cord is quite often misdiagnosed as dorsal arachnoid cysts, intradural spinal tumours, herniated discs or transverse myelitis.[2,3] The natural history remains unclear and therefore some patients with minimal symptoms can be monitored through serial imaging and careful clinical follow-up.[3] However, surgical intervention

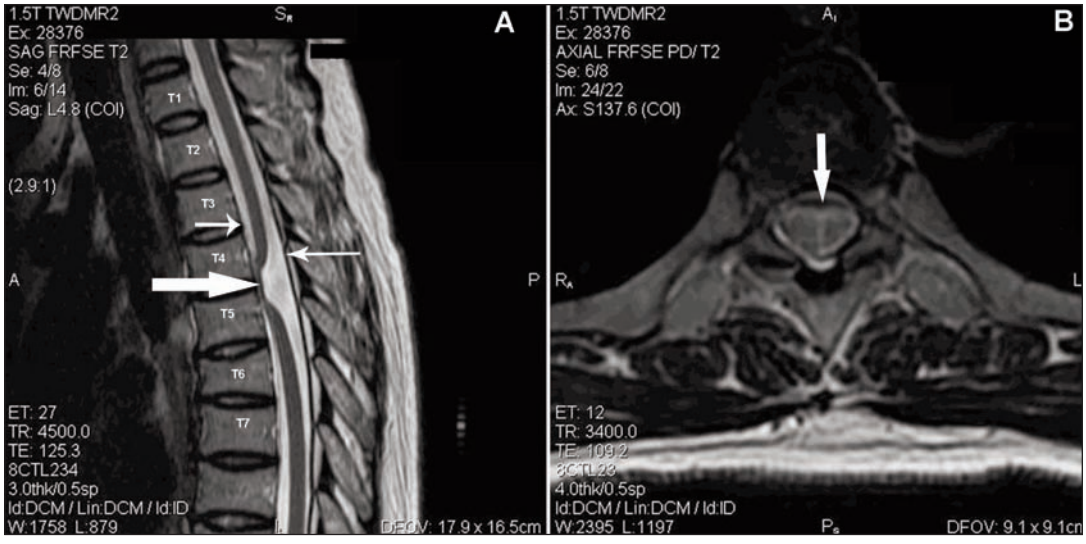

Figure 1: *A, B. Preoperative midsagittal and axial T2-weighted magnetic resonance image demonstrating a ventrally displaced spinal cord with focal herniation through the dura at T4-5. No associated cord signal changes or syrinx was present. Focal myelomalacia is present as a result of chronic pressure (big arrow). Ventral dura (Small arrow facing right). Dorsal dura (small arrow facing left). Ventral defect (Down-facing arrow).*

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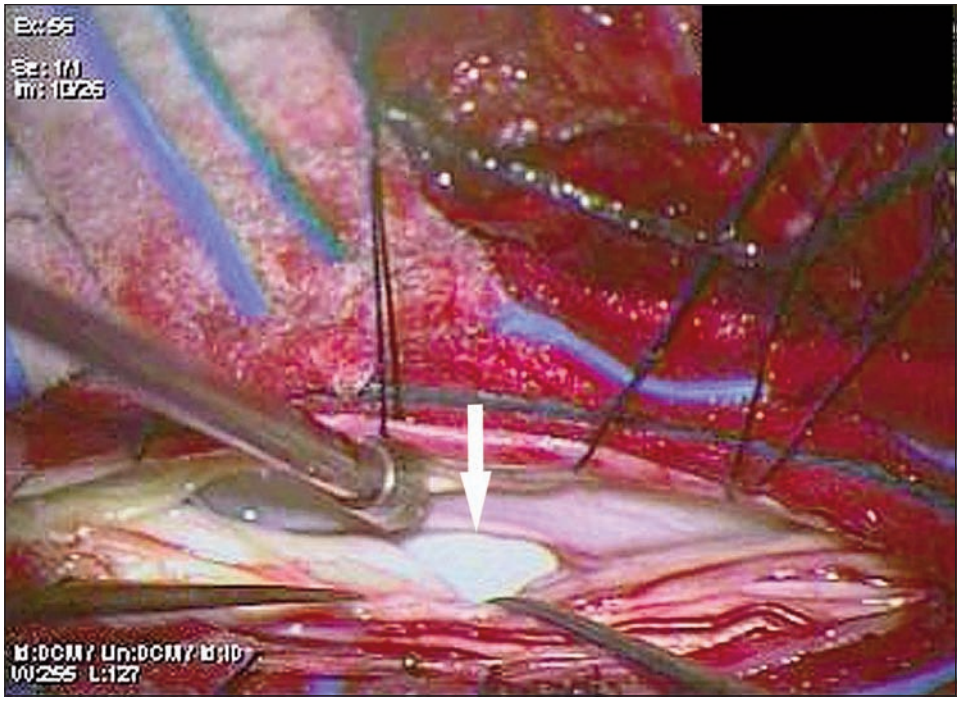

Figure 2: *Intraoperative imaging demonstrating ventral dural defect (arrow).*

should be considered for patients with progressive symptoms.[3] In principle, there are two approaches to surgical management: 1) enlarging the dural defect to prevent cord incarceration and 2) repairing the dural defect, primarily or placement of a dural graft.[2] It is the senior authors preference to repair the dural defect primarily with a graft unless the herniation is complex with significant tethering as was the case in the patient described in this report.[1,4] Surgically treated cases usually have a good long-term prognosis.[3] Most patients experience gradual symptom improvement over the initial two weeks postoperatively while the majority of remaining patients have an arrest of symptom progression.[3]

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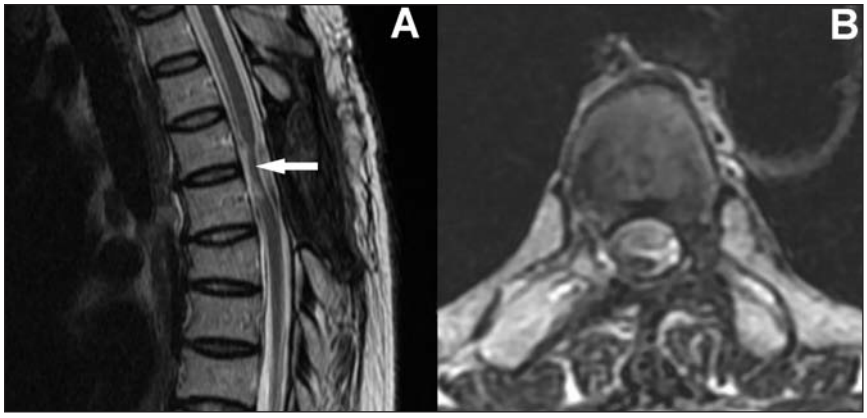

Figure 3: *A, B. Three month postoperative midsagittal and axial T2-weighted magnetic resonance image demonstrating the return of the spinal cord to a more normal position. Persistent atrophy of the spinal cord with focal myelomalacia is present (arrow).*

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