

Commentary

Brown-Sequard syndrome (BSS) is an incomplete spinal cord lesion, which occurs most often after traumatic injuries or tumor compression to the spinal cord. Cervical disc herniation as a cause of BSS was first described by Stookey in 1928,^[1] and despite several reports have been published^[2-5] it still represents a diagnostic challenge for physicians taking care of spinal disorders.

BSS involves ipsilateral loss of motor function due to corticospinal tract dysfunction, combined with ipsilateral loss of vibratory sensation and contralateral loss of pain and temperature sensation as a result of spinocerebellar and spinothalamic tract compromise respectively, reflecting hemisection of the spinal cord in the cervical or thoracic region. The spinothalamic tract crosses the midline of the spinal cord one to two segments cephalad of entry level; this explains contralateral deficit in sensation of pain and temperature starting at a dermatome a few levels below the cord injury on the contralateral side.

Yokoyama *et al.*^[6] described a 63-years-old man who presented with progressive right hemiparesis and disruption of pain and temperature sensation on the left side of the body, secondary to a large C3-C4 disc herniation compressing the spinal cord, as evidenced in the MRI; in addition, a severe canal stenosis from C4 through C7 was found.

Many reports have presented catastrophic outcomes in these cases; in fact, the literature has reported only about 50% of the cases reaching a normal motor and sensory function after treatment.^[7] Usually, compressive myelopathy by disc herniation exhibits a stepwise neurological deterioration, which suggests that the pathology results not only from direct compression, but also from vascular compromise; the patient's spinal cord blood flow can reach a critical threshold that keeps different spinal cord volumes in a penumbra state.

This paper presented a very successful outcome after a laminoplasty was performed, despite it has been described that patients undergoing anterior procedures have better recoveries than those in whom a posterior procedure is chosen.^[2,8] An early surgical decompression can prevent a cord infarct; the rapid neurological recovery observed in this case report could be explained by a decompression performed before irreversible cord damage was produced.

As described by Yokoyama *et al.*,^[6] an initial suspicion for this case was cerebrovascular disease; general physicians and specialists should be aware of this diagnostic possibility in patients presenting with an acute BSS. An early clinical suspicion and diagnostic confirmation by MRI, followed by spinal cord decompression should be warranted. Furthermore, intraoperative neuromonitoring should also be encouraged in these cases as a valuable tool aiming to preserve neurological function.^[9]

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