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## Cervical disc herniation and Brown-Sequard syndrome: A case report

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### Summary

Brown-Sequard syndrome caused by herniated cervical disc is relatively rare. We report a 45 year old female patient who presented with weakness of the right extremities and absent pain and temperature sensation on the left below the C4 dermatome. CT myelogram revealed disc herniation between C3/C4, C4/C5 and C5/C6 disc spaces but was worst at C4/C5 with anterior cord compression and evidence of posterior osteophytes on the C5 and C6 cervical vertebrae. Patient responded well to symptomatic and supportive therapy including physiotherapy. She declined surgical intervention which is usually recommended in discogenic Brown-Sequard syndrome.

**Keywords:** *Brown-Sequard syndrome, cervical disc herniation*

### Résumé

Le syndrome de Brown-sequard, causé par le disque cervical ressortit relativement rare. Nous avons fait le rapport d'une patiente âgée de 45ans qui s'était présentée avec des extrémités droites, l'absence des douleurs et une sensation de température à gauche en dessous du C4 dermatome. Le myélogramme CT révélait une herniation discale entre C3/C4, C4/C5, et C5/C6 espaces des disques mais était plus mauvais à C4/C5 la compression des ligaments antérieurs et l'évidence des ostéophytes sur les vertèbres cervicales C5 et C6. la patiente réagissait bien à la thérapie symptomatique et la thérapie supportive incluant la physiothérapie. Elle a refusé l'intervention chirurgical qui est généralement recommande pour en syndrome de Brown-sequard disco génique.

### Introduction

Brown-Sequard syndrome (BSS) is a relatively rare neurological condition reflecting hemi-section or compression of the spinal cord, often in the region of

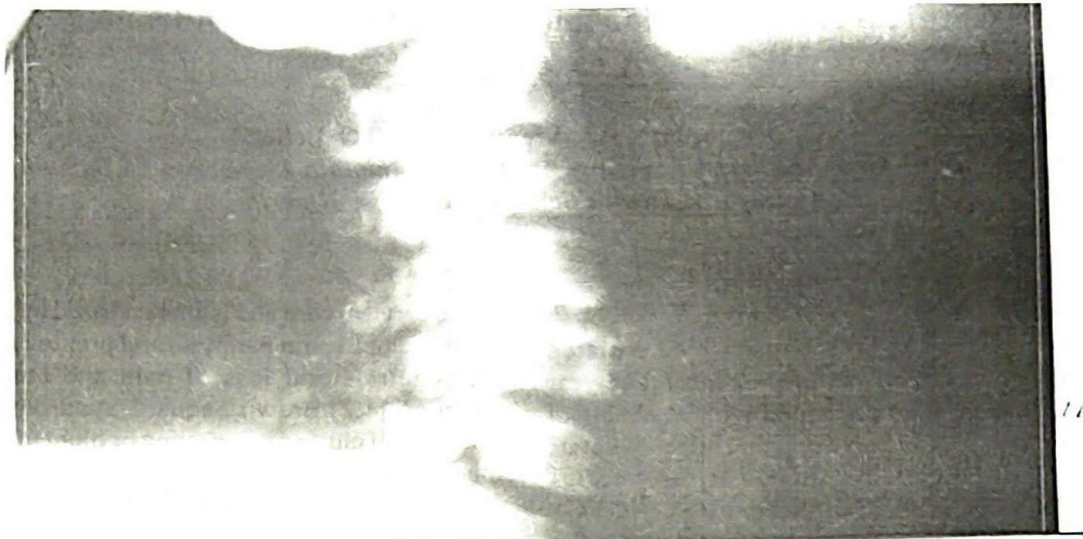
the the cervical cord and is characterized by ipsilateral upper motor neuron paralysis and proprioceptive loss with contralateral loss of pain and temperature sensation [1,2]. Brown Sequard Syndrome usually results from either a penetrating or blunt injury to the spine but numerous non-traumatic causes have been documented in the literature [2,3].

### Case report

An otherwise healthy 45 year old female secondary school teacher was referred from the accident and emergency of our facility, LAUTECH Teaching Hospital to the neurology clinic on 30/09/05 on account of sudden sharp neck pain radiating to the right side of the body, right-sided body weakness and left sided body numbness all of 7/52 duration. She developed the symptoms while trying to tie her head gear. Patient could no longer move her right side 24 hours after the onset of illness. There was no antecedent history of neck or head injury or recurrent neck pain in the past. No surgical procedure or manipulation of the neck. No headache, no seizure, no loss of consciousness and no associated sphincteric dysfunction initially.

General examination revealed an anxious-looking young woman wearing a cervical collar sitting in a wheel chair. Patient was fully conscious and alert. The pupils were normal and equal on both sides. There was no cranial nerve deficit. The tone and the deep tendon reflexes were globally exaggerated and there was spastic weakness on the right side, motor power was 1 in the right upper limb and 4 in the right lower limb, while it was normal on the left side. The gait was spastic and she had bilaterally sustained ankle clonus. The plantar responses were extensor bilaterally. There was loss of pain and temperature sensation up to C4 level on the left side and proprioceptive loss on the right side.

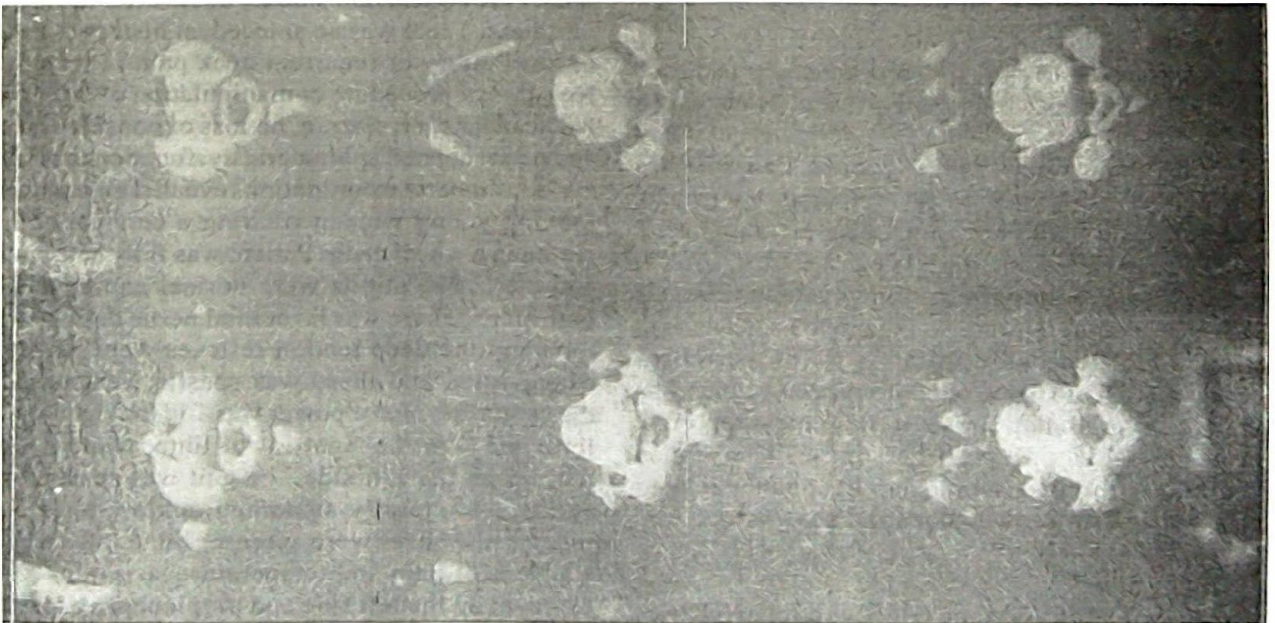
Plain Neck X-rays (lateral view) revealed reduction in the C4/C5 intervertebral disc space with reductive changes in the superior margins of C5 raising the possibility of spinal cord compression (Fig 1)



**Fig. 1:** X-ray cervical spine (lateral view) showing reduction in the C4/C5 disc space and anterior osteophytes on C3-C5

CT myelogram showed herniation /prolapse of disc materials between C3/C4, C4/C5 and C5/C6 disc spaces but was worst at C4/C5. There was associated anterior indentation of the spinal cord. Posterior osteophytes were also noted on C5 and C6 (Fig 2a, 2b and 2c).

An assessment of Brown-Sequard syndrome secondary to cervical disc herniation was made and patient was placed on tabs Doloneurobion, tizanidine, vitamin E, Tramadol and was commenced on physiotherapy. Patient was referred to the neurosurgeon but she declined surgical intervention.



**Fig. 2a:** CT myelogram showing disc herniation between C2-C4.

**Conclusion:** Severe cervical spondylosis with disc prolapse. MRI could not be done because it is not available in our centre.

At the third clinic visit, about a month after the initial one patient had started making clinical improvement, she could now feel pain as touch sensation on the

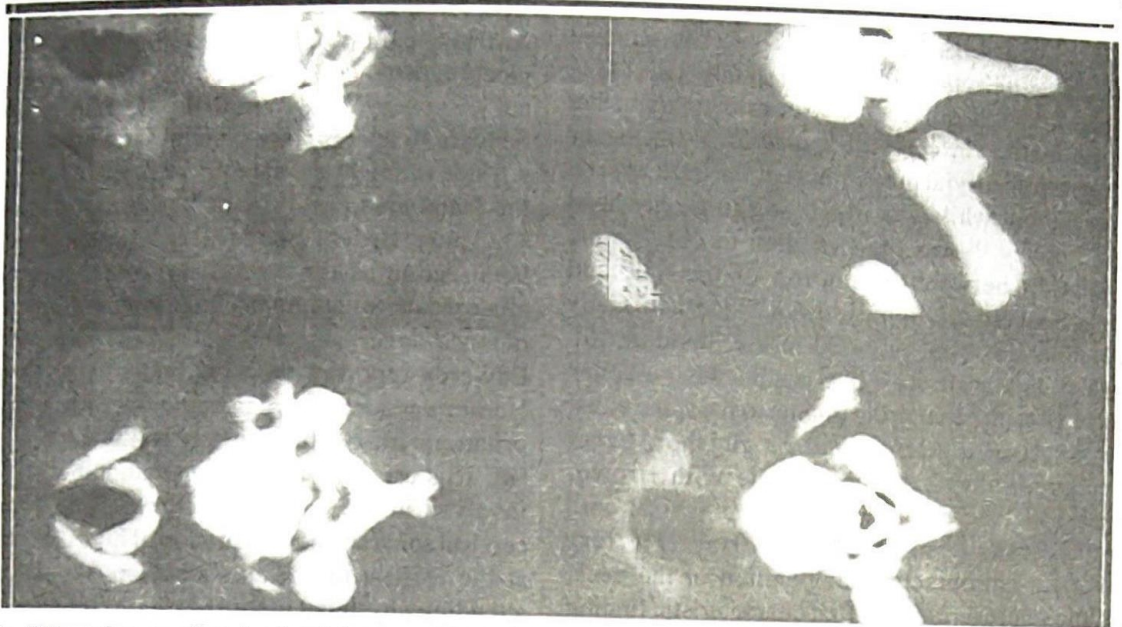


Fig. 2b: CT myelogram showing levels between C5 - T1

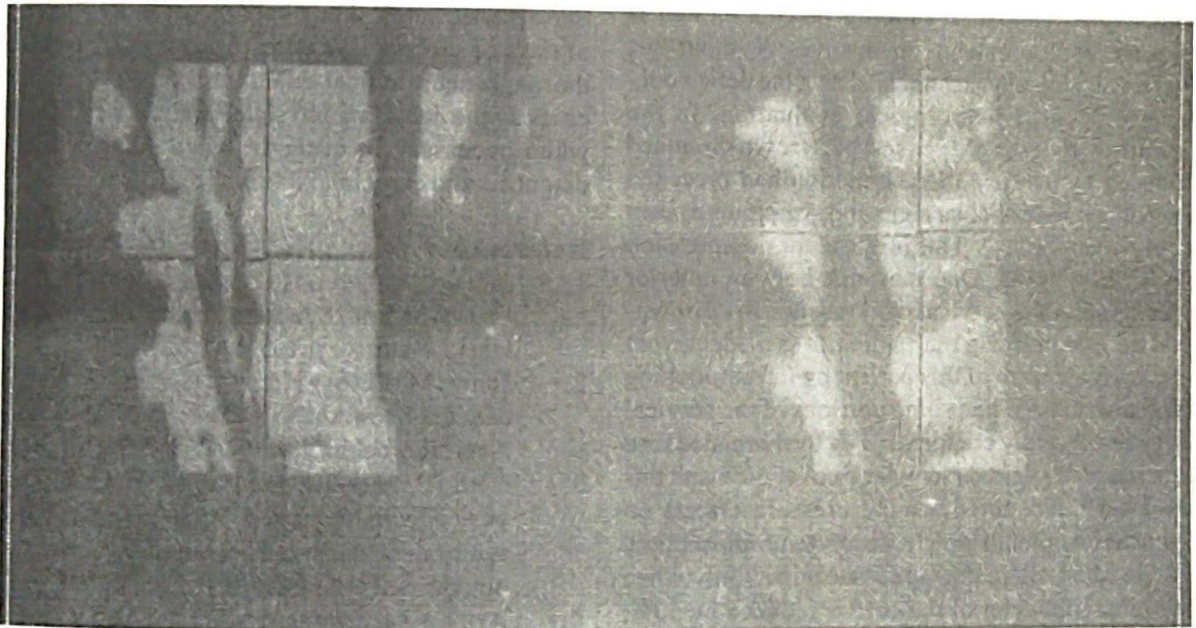


Fig. 2c: Sagittal reconstruction showing anterior cord compression

left side up to C4 and motor power was now 2 in the right upper limb and 4 in the right lower limb .Tizanidine was replaced with Baclofen for the spasticity. Three months after the initial assessment, she walked into the clinic with the aid of a walking stick and motor power was now 3 in the right upper limb and 4 in the right lower limb. Hypoesthesia on the left side had also tremendously improved. Patient is still being followed up at the Neurology clinic in the LAUTECH Teaching Hospital, Osogbo, Nigeria.

#### Discussion

Cervical Spondylosis is a chronic degenerative condition of the cervical spine that affects the vertebral bodies, and intervertebral discs of the neck causing disc herniation, spur formation etc as well as the spinal canal causing nerve roots and/or spinal cord compression [4]. Some authors also include degenerative changes in longitudinal ligaments and ligamentum flavum. Cervical Spondylosis can present as radiculopathy or myelopathy or a combination of

both. Brown-Sequard Syndrome is one of recognized clinical manifestations of cervical spondylosis [4]

In writing this report, we aim to highlight that disc herniation in cervical spondylosis can occur during routine trivial movements of the neck as seen in our patient while she was trying to tie her head gear. Stookey [5] was the first man to observe the association between herniation of the disc and Brown-Sequard syndrome in 1928. Since then, many such cases has been reported in the literature [6]. However, this rare neurological condition is most often associated with penetrating trauma to the spine either as a result of a stab wound or gunshot. Other associations that have been made with Brown-Sequard include epidural hematoma, spinal cord tumour, transverse myelitis, vertebral artery dissection, chiropractic manipulation of the neck, multiple sclerosis etc. The first characteristic finding of discogenic Brown-Sequard syndrome is contralateral deficit in sensation of pain and temperature. This is an obscure perception and is often ignored by patients themselves. Without this chief complaint, physicians can detect the deficit only by very detailed neurological evaluation. In the literature, half of the cases of Brown-Sequard produced by cervical disc herniation had preceded or accompanied radicular signs and symptoms as seen in our patient [1,2,4]. This is due to the compression of the nerve roots. One may ask how an anterior indentation of the cervical cord resulted in a Brown-Sequard Syndrome in our patient. It is evident from the CT Myelogram of this patient by further looking at the scannogram that some portions of the cervical cord were severely compressed by the herniated disc reducing their sagittal diameter by over 50% and this could have accounted for the pressure effects on the posterior column which subserves proprioception.

Most often, damage is sustained only when the sagittal diameter of the cord is reduced by 50%; pathologically this damage consists of a central necrosis involving mainly the grey matter, and demyelination associated with axonal damage located mainly deep within the posterior and lateral columns [7]. The mechanism is likely to be either shearing injury to the axons in white matter, and to cord microvasculature in the deep grey matter, analogous to closed head injury or to compression-induced intermittent ischemic damage.

It has a distinct propensity to be progressive, and shearing from anterior deforming agents alone such as seen in my patient without the necessity of an anteroposterior squeeze [7]. This explanation above accounts for the bilateral involvement of the

corticospinal tracts in the patient which resulted in global hypertonia, hyperreflexia and bilateral sustained ankle clonus. What gave out the diagnosis of Brown Sequard Syndrome is the thermo-anaesthesia on the left side and right-sided body weakness which spared the facial area.

The pure Brown-Sequard syndrome reflecting hemisection of the cord is not often observed. A clinical picture comprising fragments of the syndrome plus additional symptoms and signs is more common. Edwards reported the simultaneous presence of Horner's syndrome in a patient with Brown-Sequard syndrome after a blunt injury to the cervical spine [8]. This usually occurs when autonomic fibers are involved in the pathology. Immobilization of the cervical spine and administration of high dose steroids at the earliest moments should be the aim of the attending physician in most cases.

However, our patient still made significant improvement on symptomatic and supportive therapy. She declined surgical intervention because of the fear of risks of surgery. We want to stress the need for a thorough neurological evaluation of all suspected cases by the attending physician and prompt referral when necessary in order to make way for early diagnosis and early intervention [8,9].

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