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Case Report

Brown-Sequard Syndrome: A Case Series from a Regional Hospital in Nigeria

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Abstract

Brown-Sequard syndrome (BSS) is the constellation of symptoms and signs following hemisection or hemicompression of the spinal cord. The syndrome is an uncommon form of incomplete spinal cord injury with clinical presentation of ipsilateral motor weakness, impaired proprioception and vibratory sensation, with contralateral loss of pain and temperature sensation below the level of the lesion. We report two cases of BSS following penetrating (stab) injuries. The patients were managed non-operatively and they were followed up for 12 and 18 months, respectively, during which they made significant neurologic recovery on the Medical Research Council scale. The objective of this report is to highlight that BSS following trauma can be managed non-operatively with good neurologic outcome in cases where there is no extrinsic compression of the neural tissues.

Keywords: Brown-Sequard syndrome, incomplete spinal cord injury, penetrating injury, traumatic spinal cord injury

Introduction

Brown-Sequard syndrome (BSS) is the constellation of symptoms and signs following hemisection or hemicompression of the spinal cord. The syndrome is an uncommon form of incomplete spinal cord injury (SCI) characterised by ipsilateral motor weakness, impaired proprioception and vibratory sensation, with contralateral loss of pain and temperature sensation below the level of the lesion.[1]

BSS rarely occurs as typically described, but mostly as an incomplete SCI with some of the afore-described symptoms and some additional features.[2]

Most cases follow penetrating spinal trauma.[3] Other reported causes, however, include blunt spinal trauma, cervical spondylosis, extramedullary spinal cord tumour, spinal vascular malformation, radiation injury, among others.[4] Most patients often recover full motor function within 6 months of the injury. The neurologic recovery is better in patients who sustained blunt rather than penetrating injury.[5]

The objective of this report is to describe the clinical presentation and neurological outcome of two patients who presented at our facility with traumatic BSS and briefly review the pertinent literature on this subject.

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Case Reports

Case 1

A 47-year-old man admitted on account of weakness of his left lower limb, following stab injury to the back, 6 h before presentation. He was conscious, alert and haemodynamically stable. There was a laceration measuring about 2 cm on the left paraspinal aspect of the lower thoracic region of the back. Clinical examination revealed full power in both upper limbs and his right lower limb. There was, however, ipsilateral motor paralysis of the left lower limb, with muscle power of Grade 0 (Medical Research Council scale) in all left lower limb muscle groups, with ipsilateral impaired light touch sensation and contralateral loss of pain and temperature sensation below T11 dermatome. He also had difficulty voiding urine and was in retention; hence, urethral catheterisation was done. Anal tone was normal.

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Magnetic resonance imaging (MRI) scan revealed areas of high T2 signal (hyperintensity) at the 10th and 11th vertebral (T10/T11) levels, predominantly on the left [Figure 1]. Wound debridement and exploration under local anaesthesia with sedation was done within 12 h of admission, and it revealed no evidence of breach of the dura. Delayed primary wound closure was done after 72 h, as there was no sign of wound sepsis or cerebrospinal fluid leakage (CSF). Bladder training was commenced and control was regained within 72 h and muscle power progressively improved over the course of admission, and he was discharged home for outpatient physiotherapy after 4 weeks. At 18 months post-injury, he walks unsupported with muscle power of Grade 4 in his left lower limb. He still had impaired pain and temperature sensation on the right.

Case 2

A 37-year-old man admitted on account of weakness in his left upper and lower limbs following stab injury in the posterior triangle of the left side of his neck, 24 h before presentation. He was conscious, alert and haemodynamically stable. There was a laceration measuring about 6 cm on the lower left aspect of the neck posteriorly. He had been to a peripheral hospital where initial resuscitation was done and wound was closed primarily. Neurological examination revealed full power in his right upper and lower limbs. There was, however, paralysis of the left upper and lower limbs, with motor power of Grade 0 (Medical Research Council scale) in all muscle groups, with ipsilateral impaired light touch sensation below left C4 dermatome and contralateral loss of pain and temperature sensation below right C5 dermatome. Muscle tone and deep tendon reflexes were also decreased on the left side. He had no sphincteric dysfunction.

MRI scan showed soft-tissue disruption on the left side of the neck at C3/C4 levels with cord signal changes on T2-weighted images [Figure 2]. The wound stitches were removed, and the wound was debrided and explored under local anaesthesia with sedation. There was no evidence of breach of the dura on wound exploration. Delayed primary wound closure was done after 5 days, as there was no sign of wound sepsis or CSF. He subsequently had progressive neurologic recovery. At 12 months post-injury, he had recovered full muscle strength in his left lower limb (MRC Grade 5), while hand grip had MRC Grade 4. He still had impaired temperature and pain sensations on the right below C6, while on the left, there was hypoaesthesia to light touch below C4, with hypertonia, exaggerated reflexes and ankle clonus.

Literature review

BSS is an uncommon type of incomplete SCI typically identified by a clinical presentation reflective of hemisection or hemicompression injury of the spinal cord.[1] The syndrome is named after Charles-Edouard Brown-Sequard who described it following spinal cord hemisection experiments performed in animal models in 1840.[6]

More than 500 cases of BSS have been reported; however, its true incidence is not known. In the United States, it is estimated to account for 1%–4% of the 11,000 new cases of traumatic SCIs that occur each year, with the average age of affected patients around 40.[7,8]

BSS is often seen following penetrating spinal trauma[3] – as was the case in our patients – but many other aetiologies have been described.[4] Following penetrating injury, the weapon or fractured bone generated can cause direct injury to the spinal cord; oedema and ischaemia may result from injury to the spinal cord vasculature or contrecoup cord contusion may cause neurologic deficits.[8] Given the variability in the mechanisms of injury, classic BSS with pure ipsilateral motor and contralateral sensory deficits is rare.[9] In our patients, there was no CSF detected, which suggests that the cord injury may have been caused by contrecoup contusion within the walls of the spinal canal, rather than by a direct impact of the assaulting weapon.[8] In

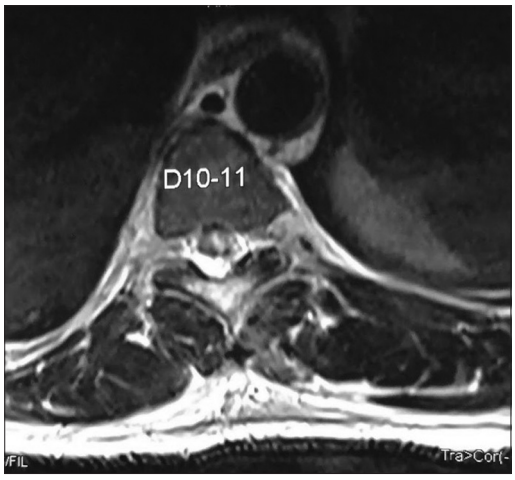

Figure 1: T2-weighted axial magnetic resonance imaging showing soft-tissue disruption on the left and hyperintensity signal cord changes at D10–11 level

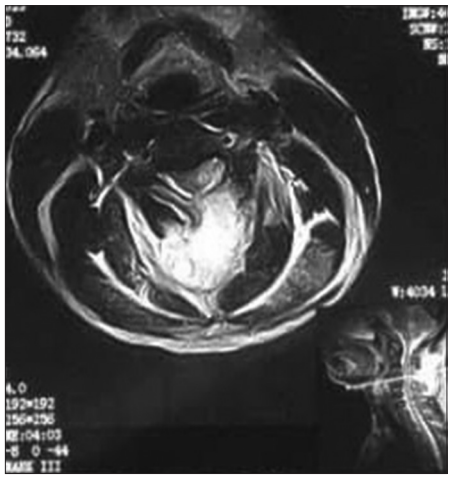

Figure 2: T2-weighted magnetic resonance imaging demonstrating soft-tissue disruption and hyperintense cord signal changes, more on the left at C3–C4 vertebral level

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the report by Takemura *et al*.,[8] CSF was detected, however, it resolved with non-operative care, which was monitored with serial MRI scans. Some authors have also described Brown-Sequard-plus syndrome in patients in which there are associated bladder/bowel dysfunction and Horner's syndrome.[5,8]

Management is usually conservative, involving wound debridement and closure, anti-tetanus prophylaxis, antibiotics, with aggressive early physical therapy and rehabilitation. This modality was adopted for both cases reported, as operative intervention following penetrating injuries has not been reported to be better than conservative treatment and is only indicated in the presence of persistent CSF, spinal cord/root compression and worsening neurology.[9,10]

Patients with BSS have a good potential for neurological recovery, with 75%–90% ambulating after rehabilitation and 82% and 89% regaining bowel and bladder continence, respectively.[5] In the cases reported, bladder and bowel control was regained within 72 h and they attained an ambulatory status with support within 6 weeks.

Conclusion

BSS following trauma can be managed non-operatively with good neurologic outcome in cases where there is no extrinsic compression of the neural tissues, anatomic hemisection or transection of the spinal cord.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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