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A 67-year-old otherwise healthy woman was assaulted with a knife, receiving multiple stab wounds. On arrival, vital signs were within normal limits. She was mentating appropriately. She complained of weakness to her right upper and lower extremities. Neurologic examination demonstrated 0/5 right triceps and grip strength to the right upper extremity and 0/5 motor strength throughout the entire lower extremity. Right sensory examination demonstrated hypesthesia at C7, anesthesia at C8 and T1, with hyperpathia T2 fading through the upper lumbar levels associated with complete proprioceptive loss. On the left, there was anesthesia at T4 and hypesthesia at T5 fading through the upper lumbar levels with retained left proprioception. Back examination demonstrated a 1 cm laceration to the left of midline at C7 with clear fluid leaking from the wound. MRI demonstrated abnormal signal at the C5–C6 level, consistent with traumatic injury secondary to stab wound.

Parasagittal short -T1 inversion recovery (STIR) MRI (figure 1) spinal cord hemisection at C6. Remaining portions of the imaged spinal cord appeared intact with no gross area of abnormal signal to suggest significant contusion or edema.

WHAT WOULD YOU DO?

- Bolus 2L lactated Ringer's solution and initiate norepinephrine given concern for neurogenic shock.
- Obtain spine team consultation given concern for Brown-Sequard syndrome (BSS).

- Initiate physical therapy given inability to walk.
- Consult neurology given concern for acute-onset Guillian-Barre syndrome.

WHAT WE DID AND WHY**Correct answer: B**

Complete spinal cord transection results in loss of sensory and motor function distal to the site of injury. Lateral hemisection syndrome, more commonly known as BSS, involves the dorsal column, corticospinal tract, and spinothalamic tract unilaterally. It results in weakness, loss of vibration and proprioception ipsilateral to, and loss of and temperature sensation contralateral to, the injury.

Based on examination and imaging findings, a diagnosis of spinal cord laceration with resultant BSS at C5–C6 was made. She was taken to the operating room to repair her dural laceration and was discharged to rehabilitation on postoperative day 3. She was seen in follow-up by neurosurgery 2 months later, at which time she demonstrated improving upper and lower extremity motor examinations. Vibratory sensation was returning. Proprioception remained absent in the right lower extremity.

BSS was described in 1855 by Charles-Edouard Brown-Sequard after experimentation with spinal cord hemisection in animals.¹ There are three mechanisms by which a spinal cord injury (SCI) is propagated following penetrating spinal injury. First, direct damage caused by the weapon or bony fragments causes permanent physical cord damage. Second, the cord's vascular supply is compromised, resulting in edema and ischemia. Third, cord contusion causes neurologic deficits. Although the neurophysiology of it is understood and the mechanism (secondary to spinal cord hemisection) is obvious, given the variability in the three aforementioned mechanisms, it is rare to see pure BSS with pure ipsilateral motor and contralateral sensory deficits.²

Each year, 11 000 cases of SCI occur in the USA.³ BSS is not common, but over 500 cases have been reported to date.⁴ Its true incidence is unknown; it has been estimated to constitute 1%–4% of traumatic SCIs,³ with the average age of affected patients around 40.³ Very few patients will actually exhibit pure BSS. 'Brown-Sequard-Plus' syndrome is appreciated more frequently, wherein there is a relative ipsilateral hemiplegia paired with a relative contralateral hemianalgesia.³

BSS is usually secondary to a penetrating SCI, but many other etiologies have been described. BSS may be caused by a cervical synovial cyst,⁵ a ruptured pheochromocytoma,⁶ transverse myelitis after influenza vaccination,⁷ and even as a rare manifestation of decompression sickness.⁸ Diagnosis is established based on classical examination findings and MRI.



Figure 1 MRI demonstrating abnormal signal at the C5–C6 level, consistent with traumatic injury secondary to stab wound.

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Management is conservative with aggressive early rehabilitation. Surgical intervention is indicated in the presence of cerebrospinal fluid leak, persistent spinal cord/root compression, or progressive deterioration.²

BSS demonstrates a favorable prognosis compared with other types of incomplete SCI.^{9,10} In fact, it has the best prognosis of all the SCI syndromes,¹¹ with >75% of patients walking without assistive devices after rehabilitation.^{10,11} It has been postulated that the most important predictive factor in terms of eventual functional status is whether the predominant plegic site was an upper or lower limb. In patients where the upper extremity is weaker than the lower extremity, walking at discharge is more likely.¹⁰ Recovery occurs as spinal cord edema resolves, with duration of recovery longer than 1–6 months.¹²

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REFERENCES

- 1 Rengachary SS, Colen C, Guthikonda M. Charles-Edouard Brown-Séquard: an eccentric genius. *Neurosurgery* 2008;62:954–64.
- 2 Lipschitz R, Block J. Stab wounds of the spinal cord. *Lancet* 1962;2:169–72.
- 3 McKinley W, Santos K, Meade M, Brooke K. Incidence and outcomes of spinal cord injury clinical syndromes. *J Spinal Cord Med* 2007;30:215–24.
- 4 Berman AL. Brown sequard syndrome. <https://rarediseases.org/rare-diseases/brown-sequard-syndrome/> (accessed 18 Dec 2017).
- 5 Kim SW, Ju CI, Kim HS, Kim YS, CI J, Kim YS. Brown-séquard syndrome caused by a cervical synovial cyst. *J Korean Neurosurg Soc* 2014;55:215–7.
- 6 Karkera M. A ruptured pheochromocytoma: an unlikely cause of Brown-Sequard syndrome. *J Clin Anesth* 2014;26:159–60.
- 7 Vieira MA, Costa CH, Vieira CP, Cavalcanti MA, Ferreira-Filho SP. Transverse myelitis with Brown-Séquard syndrome after H1N1 immunization. *Arq Neuropsiquiatr* 2012;70:555.
- 8 Tseng WS, Huang NC, Huang WS, Lee HC. Brown-Séquard syndrome: a rare manifestation of decompression sickness. *Occup Med* 2015;65:758–60.
- 9 Pollard ME, Apple DF. Factors associated with improved neurologic outcomes in patients with incomplete tetraplegia. *Spine* 2003;28:33–8.
- 10 Roth EJ, Park T, Pang T, Yarkony GM, Lee MY. Traumatic cervical Brown-Sequard and Brown-Sequard-plus syndromes: the spectrum of presentations and outcomes. *Paraplegia* 1991;29:582–9.
- 11 Bosch A, Stauffer ES, Nickel VL. Incomplete traumatic quadriplegia. A ten-year review. *JAMA* 1971;216:473–8.
- 12 Park SD, Kim SW, Jeon I. Brown-sequard syndrome after an accidental stab injury of cervical spine: a case report. *Korean J Neurotrauma* 2015;11:180–2.