Case report

Brown-Séquard syndrome without vascular injury associated with Horner's syndrome after a stab injury to the neck

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Case Description: This case reviews the acute care and rehabilitation course of a 44-year-old right-handed woman after an assault with a pocketknife. She suffered multiple stab wounds including penetrating injury to the left side of her neck. Physical examination revealed left hemiplegia (motor score = 57), impaired pinprick sensation on the right caudal to the C5 dermatome, impaired joint position sense on the left, and left ptosis and miosis. Initially she was unable to stand without maximum assistance. MR imaging revealed transection of the left hemicord at the C5 level without cord hemorrhage. CTA of the neck was negative for vascular injury. She completed 18 days of acute inpatient rehabilitation. She used forearm crutches for ambulation at time of discharge. Prior to discharge the patient provided written permission for a case report.

Discussion: Stab wounds are the most common cause of traumatic Brown-Séquard syndrome. Horner's syndrome is common in spinal cord lesions occurring in the cervical or thoracic region, however the combination of Horner's and Brown-Séquard syndromes is less commonly reported. In this case report, we review recommendations regarding initial imaging following cervical stab wounds, discuss anatomy and associated neurological findings in Brown-Séquard and Horner's syndromes, and review the expected temporal course of motor recovery.

Conclusions: Facilitating motor recovery and optimizing function after Brown-Séquard spinal cord injury are important roles for the rehabilitation team. Imaging is necessary to rule out cord hemorrhage or vascular injury and to clinically correlate cord damage with physical examination findings and expected functional impairments. Documenting associated anisocoria and explaining this finding to the patient is an important element of spinal cord injury education. Commonly, patients with Brown-Séquard injuries demonstrate remarkable motor recovery and regain voluntary motor strength and functional ambulation.

Keywords: Brown-Séquard syndrome, Horner's syndrome, Anisocoria

Introduction

Stab wounds of the spinal cord represent approximately 26% of all spinal cord injuries.¹ Stab wounds are the most common cause of traumatic Brown-Séquard syndrome.²⁻⁵ Horner's syndrome is common in spinal cord lesions occurring in the cervical or thoracic region, however the combination of Horner's and Brown-Séquard syndromes has been rarely reported. This rare combination has been reported following cervical corpectomy, blunt injury to the cervical spine, drill bit and screwdriver induced trauma to the spinal cord, as a result of epidural hematoma and one case following a knife stab wound.⁶⁻¹⁰

In this case report, we review recommendations regarding initial imaging following cervical stab wounds, discuss anatomy and associated neurological findings in Brown-Séquard and Horner's syndromes, and review the expected temporal course of motor recovery.

Case presentation

The patient was a 44-year-old, right-handed woman who was assaulted with a pocketknife. She suffered multiple stab wounds including penetrating injury to the left side of her neck. Physical examination revealed left hemiplegia (American Spinal Injury Association motor score = 57), impaired pinprick sensation on the right caudal to the C5 dermatome, impaired proprioception on the left, left ptosis and miosis. Computed

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tomography angiography (CTA) of the neck was negative for vascular injury. Magnetic resonance imaging (MRI) of the cervical spine revealed left hemisection of the spinal cord at the C5 level without spinal cord hemorrhage (Fig. 1).

The patient was managed non-operatively. Eight days after her injury she was admitted to our acute inpatient rehabilitation unit. Physical exam on the date of rehabilitation admission revealed antigravity strength of her left elbow flexors, trace activation of the left wrist extensors, elbow extensors, hip flexors and knee extensors and no visible or palpable contraction of the left ankle dorsi and plantar flexors. Pinprick sensation was impaired caudal to the C5 dermatome on the right. She required maximum assistance for standing and sidestepping. Urinary retention was also present.

The patient completed 18 days of acute inpatient rehabilitation. Proximal lower extremity extensor muscles regained full strength over this time period. Distal flexor muscles showed very little recovery. There was no recovery of left dorsiflexion strength, while ankle plantar flexion progressed to antigravity strength. At the time of discharge, her American Spinal Injury Association motor score was 72. She could ambulate greater than 500 feet using a left ankle foot orthosis and forearm crutches. Functionally, she became independent with activities of daily living and her bladder function returned to normal. Ptosis and miosis did not resolve and remained at the time of discharge.

Prior to discharge the patient provided written permission for a case report.

Figure 1 Left hemisection of the spinal cord at the C5 level.

Discussion

Following cervical stab wounds, a full neurologic examination is a necessary first step. Careful testing of motor function, reflexes, light touch sensation, pinprick sensation, proprioception, and cranial nerve function is essential. Vascular injury should be considered and ruled out with CTA. CTA will also help identify associated bone lesions such as the C5 spinous process fracture found in our patient. Cervical spine MRI should be included to evaluate for spinal cord hemorrhage and epidural hematoma. An epidural hematoma is a neurosurgical emergency. On MRI, a spinal epidural hematoma appears as a convex lesion with a well-defined border. An acute hematoma appears hypointense to isointense to the spinal cord on T1-weighted images and heterogeneous on T2-weighted images. After 24 hours, the hematoma will be hyperintense on T1-weighted images and isointense to hyperintense on T2-weighted images. There is no enhancement if contrast is used.¹¹

Charles-Édouard Brown-Séquard provided the first description of three distinct symptoms, which later became known as Brown-Séquard syndrome. This was published in 1855 in his monograph "Experimental and clinical researches in the physiology and pathology of the spinal cord." The syndrome was originally described as follows: unilateral paralysis of voluntary motion below the level of the lesion, ipsilateral hyperesthesia (seen only in animals), segmental atrophy and sensory loss at the level of the lesion, and contralateral analgesia and thermanesthesia a few segments below the lesion.¹² This definition has evolved over the years to a simplified form. The classic signs of Brown-Séquard syndrome include ipsilateral motor and proprioceptive loss and contralateral loss of pain and temperature sensation.

Brown-Séquard syndrome is a rare constellation of symptoms with the most common cause of the syndrome being penetrating trauma following stab wounds. Less common occurrences have been reported secondary to epidural hematoma, cervical disc herniation, space occupying lesions, and blunt trauma.^{6–10} The location of Brown-Séquard syndrome is approximately 75% thoracic, 17% cervical, and 8% lumbar.⁸

Motor paralysis is secondary to corticospinal tract disruption in spinal cord injury. In Brown-Séquard syndrome, motor weakness is seen ipsilateral to the side of the spinal cord lesion as the corticospinal tracts decussate in the lower medulla. Sensory findings have mixed etiology in Brown-Séquard syndrome. Fibers conveying position sense and vibratory sense ascend in the ipsilateral dorsal columns. Thus, loss of position sense and

vibratory sense is seen ipsilateral to the spinal cord lesion. In contrast, pain and temperature fibers ascend for one or two segments in Lissaur's tract before synapsing and crossing over to the contralateral spinothalamic tract. Therefore, loss of pain and temperature sensation is seen contralateral to the side of the lesion.

Horner's syndrome consists of ipsilateral ptosis, miosis and less commonly anhidrosis secondary to a lesion in the sympathetic pathway. Sympathetic pathway disruption can occur in the brain, spinal cord, or the sympathetic chain in the neck all causing Horner's syndrome on the same side as a lesion. Ocular sympathetic nerve fibers supply the superior tarsal muscle, known as Muller's muscle, as well as the iris dilator muscle. Thus, sympathetic pathway disruption causes lid ptosis and miosis.

Horner's syndrome has been rarely reported as a complication of Brown-Séquard syndrome spinal cord injuries.⁶⁻¹⁰ Horner's syndrome can be confirmed pharmacologically with the use of topical cocaine 4%. Cocaine acts by blocking re-uptake of norepinephrine at the presynaptic nerve terminal supplying the iris dilator muscle. In normal subjects, this results in pupil dilation however in Horner's syndrome the pupil will dilate poorly due to the lack of norepinephrine being released from the presynaptic nerve terminal due to a lesion somewhere along the sympathetic pathway. A post cocaine anisocoria value of 1.0 mm is diagnostic of Horner's syndrome. This test is rarely done as Horner's syndrome remains a clinical diagnosis and there is little prognostic value gained from performing the test.⁸ Specific to this case, the diagnosis of Horner's syndrome remained clinical with no pharmacologic confirmation performed.

The prognosis for recovery of ptosis and miosis secondary to Horner's syndrome is not known. In a recent study by Chacko *et al*,⁶ the clinical outcomes of 153 patients undergoing oblique corpectomy for cervical spondylitic myelopathy were assessed. Temporary Horner's syndrome in the immediate postoperative period was seen in 32 patients. Twenty-three of these patients had full resolution of ptosis and miosis at 6 months while 9 patients had permanent Horner's syndrome. There is no data available for functional recovery of Horner's syndrome following stab injuries to the cervical spine.

Anisocoria is present in up to 20% of the general population. However, it should not be associated with ptosis or anhidrosis. In patients with spinal cord injury, the presence of anisocoria in combination with ptosis and anhidrosis should alert examiners to the possibility of injury to the sympathetic chain or spinal cord sympathetic pathways. In the case of our patient,

ptosis and miosis were subtle physical examination findings and not detected until the patient complained of lid lag weeks after her injury. The cosmetic disturbance of ptosis distressed our patient and caused undue anxiety and worry related to its cause and expected recovery. These complications highlight the importance of diagnosing Horner's syndrome so proper patient education can be provided regarding the etiology and prognosis for recovery.⁶

The temporal course of motor recovery after Brown-Séquard spinal cord injuries was described in the now classic paper by Little and Halar in 1985.¹³ They described the course of motor recovery in 2 patients and reviewed the literature on an additional 59 patients. Shared qualities of motor recovery in Brown-Séquard syndrome include: (1) recovery of ipsilateral proximal extensor muscles before ipsilateral distal flexors, (2) recovery of weakness in the extremity with pain/temperature sensory loss before the opposite extremity, and (3) recovery of functional gait by 1 to 6 months. Commonly, patients with hemisection of their spinal cord will initially have complete lower extremity paralysis. However, most patients recover voluntary strength and functional ambulation. Our patient demonstrated a similar pattern of recovery, progressing to full ipsilateral knee extensor strength with no return of dorsiflexion strength. Remarkably, our patient required maximum assistance for bed mobility and standing on admission and less than three weeks later ambulated community distances with an ankle foot orthosis and forearm crutches.

Mechanisms to explain the described recovery pattern include resolution of conduction block, collateral sprouting, denervation supersensitivity, and contralateral cord mechanisms. Conduction block in descending motor pathways at the site of injury may be present. This can be secondary to edema, hematomyelia, ischemia, or direct pressure. Conduction block typically resolves over the course of weeks possibly explaining the rapid early recovery in voluntary motor strength in patients with Brown-Séquard syndrome. Collateral sprouting and denervation supersensitivity is theorized to allow spared axons to assume the role of damaged descending axons. Contralateral cord mechanisms are less well understood. Evidence suggests that spared descending motor axons in the contralateral cord mediate recovery by distributing inputs via collateral sprouting to damaged ipsilateral neurons.¹³

Facilitating motor recovery and optimizing function after Brown-Séquard spinal cord injury reflect the roles of the rehabilitation team. Imaging is necessary to rule out cord hemorrhage or vascular injury and to clinically correlate cord damage with physical

examination findings and expected functional impairments. Documenting associated anisocoria and explaining this finding to the patient is an important element of patient education regarding potential complications related to spinal cord injury. Commonly, patients with Brown-Séquard injuries demonstrate remarkable motor recovery and regain voluntary motor strength and functional ambulation.

Disclaimer statements

Contributors SJ: first author. Wrote the case report. Resident physician on inpatient unit during patients rehab admission. MJ: second author. Revised the manuscript. Resident physician who did the initial consult on this patient. JZ: third author. Revised and edited manuscript. Attending physician during patients rehab admission.

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