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Case Report

Brown-Sequard syndrome associated with Horner's syndrome after a penetrating trauma at the cervicomedullary junction

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Study design: Case report of a 21-year-old man that had concurrence of Brown-Séquard syndrome and Horner's syndrome after a penetrating trauma in the neck.
Objectives: This report analyzes the location of lesions that cause a combination of Horner's and Brown-Séquard syndrome. It is important to know the anatomic structure of spinal cord and the sympathetic nerve chain.
Setting: Spinal Cord Unit, Department of Physical Medicine and Rehabilitation, Hospital La Fe, Valencia, Instituto Oftalmológico de Alicante, Alicante, Spain.
Methods: Description of a single patient case report.
Results: The clinical findings and MRI showed a good correlation. The Horner's syndrome was confirmed with a 4% cocaine test. The patient received a conservative treatment with high-dose steroid therapy (NASCIS-3).
Conclusion: The patient presented with Brown-Séquard syndrome and Horner's syndrome. Clinical examination and MRI made a quick and correct diagnosis. The patient recovered completely after the conservative treatment.
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Keywords: Brown-Séquard syndrome; Horner's syndrome; spinal cord injury

Introduction

Concurrence of Horner's syndrome is common in a spinal cord lesion situated in the cervico-thoracic region.[1,2] However, the combination of Horner and Brown-Séquard syndromes is very rare. On review of the literature, this appears to be the second case reported.[3,4]

Case report

A 21-year-old man was involved in a street-fight; as a result, he was injured with a screw-driver in the left side of the upper neck. He was immediately brought to our emergency room. Physical examination at admission showed: Left hemiplegia (motor score = 52, using American Spinal Injury Association (ASIA) guidelines), impaired perception of pin-prick, temperature, and light touch on the right, below the C1 dermatome. Sensation to vibration and joint position were decreased remarkably on the left side, below C1 dermatome. Positive Babinski signs were present bilaterally. No dysarthria or problems with swallowing were present.

Brown-Séquard syndrome of the high cervical spine was suspected and the patient was treated with glucocorticosteroid protocol (NASCIS-3). He was admitted to the intensive care unit due to respiratory failure, caused by paralysis of the left hemidiaphragm.

Two days after he was transferred to our Spinal Cord Unit. The patient then began to feel pain and paresthesia around the right ear (an Arnold's neuralgia). A left Horner's syndrome (including slight lid drop and the left pupil smaller than the right) was also noticed.

Magnetic resonance imaging (MRI) of cervical spine showed increased signal intensity at the union between medulla oblongata and right upper cervical cord with no associated fractures (Figures 1 and 2). The distribution of MRI abnormalities corresponded well to the clinically expected lesions. It was decided to treat conservatively due to the good prognosis of these lesions.

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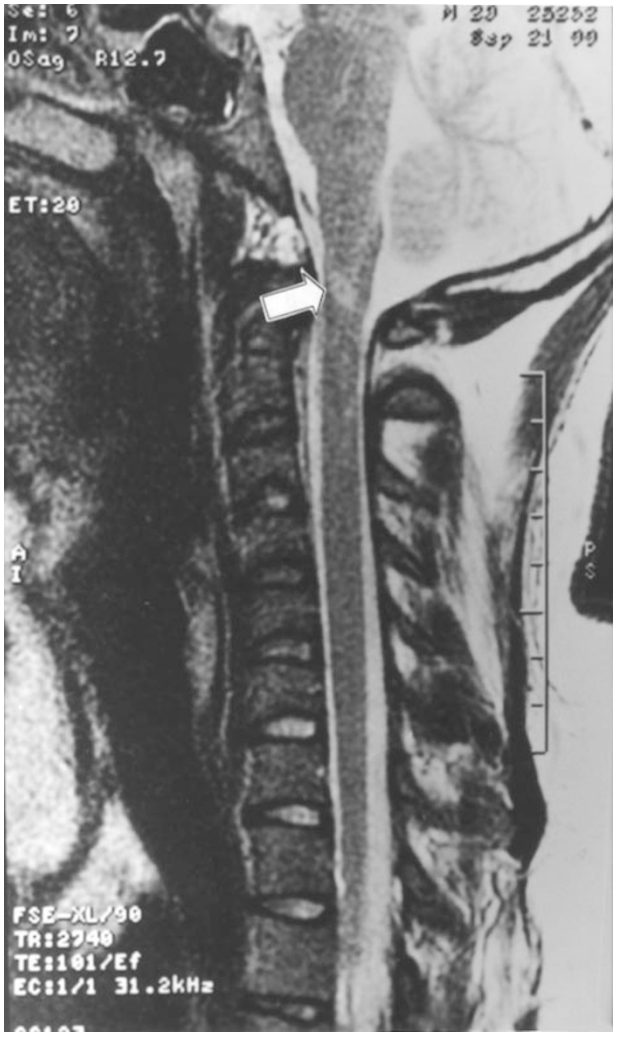

Figure 1 Sagital T_2 weighted image of a lesion at the cervicomedullary junction (arrow), with surrounding edema

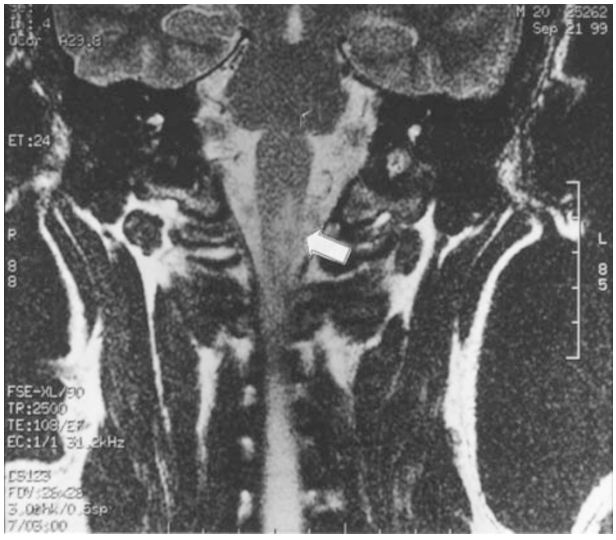

Figure 2 Coronal T_2 weighted image of the penetrating lesion affecting the cervicomedullary junction (arrow)

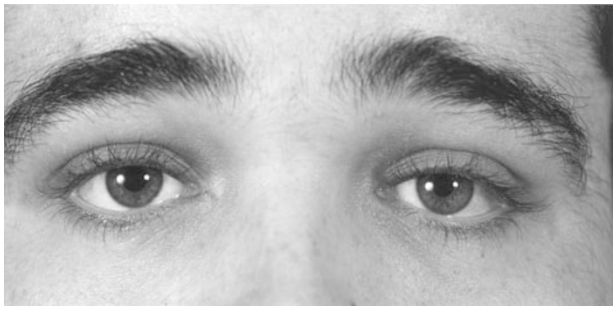

Figure 3 Four per cent cocaine test for Horner's Syndrome: Pre-test, showing slight ptosis and miosis of the left eye

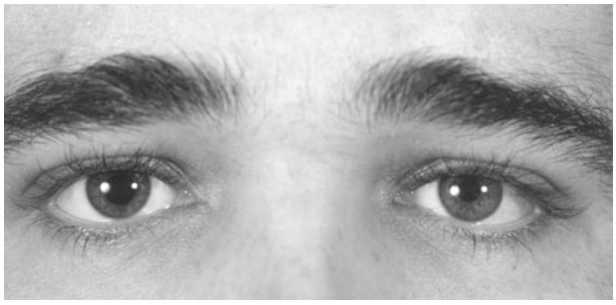

Figure 4 Four per cent cocaine test for Horner's syndrome: Post-test, revealing mydriasis of the normal eye 15 min after the instillation of the drop

Confirmation of Horner's syndrome was determined by 4% Cocaine test (Figures 2 and 3), done by an ophthalmologist. Hydroxyamphetamine was not available for testing.

Three months after the injury, the patient had only minor sensory changes and weakness (motor score = 95), bowel and bladder function were normal and he was able to walk with the aid of a crutch when leaving hospital.

Discussion

Brown-Séquard in 1849 was the first to report the pure syndrome,[5] characterized by ipsilateral hemiplegia and loss of tactile and propioceptive sensation and contralateral loss of pain and temperature sensation. It results from partial hemisection of the spinal cord. Distribution of Brown-Séquard syndrome is 75% thoracic, 17% cervical, and 8% lumbar.[6] Most of the traumatic Brown-Séquard syndrome cases are caused by stab wounds, although these lesions resulted from a contrecoup injury of the cord against the bone rather than from an intrinsic cord damage from direct penetration, as in our case.

The motor paralysis seen in this syndrome stems from disruption of the corticospinal tracts that

decussate only once in the lower medulla. Thus, the motor findings are seen on the same side as the injury. In contrast, the sensory findings seen in Brown-Séquard syndrome are mixed. The sensory fibers ascending adjacent to the motor tracts enter the cord through the dorsal root ganglion. Those fibers that convey position and vibration ascend ipsilaterally in the spinocerebellar tract; thus, those sensations are lost on the same side of the lesion. Pain and temperature fibers ascend the spinal cord for one or two segments in Lissaur's tract before synapsing and crossing to the contralateral lateral spinothalmic tract. Therefore, disruption of the cord causes loss of pain and temperature one to two levels below the lesion.[7]

A lesion in the sympathetic pathway, whether in the brain, spinal cord, or sympathetic chain in the neck, causes Horner's syndrome on the same side. Ocular sympathetics supply lid smooth muscle as well as the iris dilator muscle; thus there will be slight lid ptosis and miosis.

Confirmation of the diagnosis of Horner's syndrome may be determined pharmacologically with topical cocaine 4%. Approximately 90% of norepinephrine released in the neuromuscular junction of the iris dilator muscle is metabolized by re-uptake by the presynaptic nerve terminal. Cocaine acts by blocking this re-uptake mechanism, thereby increasing the amount of norepinephrine available to stimulate the muscle. The pupil will dilate in a normal eye, but in Horner's syndrome the pupil will dilate poorly, since little or no norepinephrine is being released into the synaptic cleft.

A postcocaine anisocoria value of at least 1.0 mm is required to make the diagnosis of Horner's syndrome.[8] Although other pharmacological tests (eg hydroxyamphetamine) have been used to localize Horner's lesions, the diagnostic accuracy and sensitivity are not really reliable. The topical diagnosis of Horner's syndrome is mostly dependent on the clinical history and the accompanying neurological signs and symptoms.

A central type lesion may present with telodiencephalic ischemic syndrome or symptoms and signs of the brain stem, or spinal cord. When postganglionic lesion is caused by lesions of the basal skull or cavernous sinus, the cranial nerve palsies usually are involved. The preganglionic lesions usually present with brachial plexopathy or Pancoast superior sulcus syndromes. The Horner's syndrome in our patient can be explained by way of the compression of the sympathetic pathway along with the lateral column of the cervical spinal cord at C1 level (a central type lesion).

The importance of the MRI in the diagnosis of Brown-Séquard syndrome has been emphasized in recent literature because it offers a noninvasive method to show the pathologic nature, location and extent of a lesion, specially if a spinal epidural hematoma is suspected,[2] which is a neurologic emergency.

Treatment of this lesion should follow the same principles as those of other acute spinal cord injuries. The recent NASCIS-3 emphasizes administration of steroid within 3 h of the injury. Early high-dose steroid therapy has been associated with a reduction in the convalescent and rehabilitative phases of acute spinal cord injury.[9] Recovery of neurological function is attributed to resolution of the conduction block in the injured axons as edema resolves as well as contribution from the uninjured side of the cord.[7]

The case presented is unusual, due to the combination of a high sympathetic lesion – the Horner's syndrome is usually found in lesions in the low cervical cord – and a Brown-Séquard syndrome in the junction between medulla oblongata and upper cervical cord, with the survival and subsequent good recovery of the patient after a high, penetrating injuring of the spinal cord.

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