Case Report

Horner's syndrome in traumatic first rib fracture without carotid injury; review of anatomy and pathophysiology

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Summary

Case report of a 51 year old man involved in a motor vehicle accident presenting with multiple thoracic wall injury, including bilateral first rib fractures. He slowly developed a right sided Horner's syndrome due to a right paravertebral haematoma. The initial imaging did not display any carotid injury, however the developing right paravertebral haematoma was not initially reported. We review the anatomy and pathophysiology of this well-known but rare condition to show how first rib fractures should raise suspicion of Horner's syndrome irrespective of the presence or absence of any underlying blunt carotid injury.

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Introduction

Motor vehicle accidents are a leading cause of trauma morbidity and mortality. There are many serious injuries that can occur, including the uncommon but often serious and sometimes fatal, blunt carotid injuries. Due to the anatomical relations of the carotid sheath, blunt carotid injury can have associated injuries such as Horner's syndrome. Isolated Horner's syndrome, which occurs rarely in blunt trauma, is usually in association with first rib fractures. We discuss a patient who developed Horner's syndrome four days after his initial injury and review the anatomy and pathophysiology of Horner's syndrome. We hope to raise awareness that injury to the sympathetic chain should be considered in traumatic first rib fractures, independent of blunt carotid injury.

Case report

A 51-year-old man was brought to the emergency room after a car drove over his chest at low speed. On arrival he was protecting his own airway, required oxygen to maintain saturations above 94%, was mildly tachycardic, normotensive and his Glasgow Coma Score (GCS) was 15. He had moderate subcutaneous emphysema and reduced air entry to his right hemithorax. A chest drain was inserted with resolution of his haemopneumothorax. CT Chest showed multiple right sided rib fractures including bilateral first rib fractures. During his admission he required analgesia and oxygenation for his chest injuries and orthopaedic operative intervention for right lower limb injuries. His chest drain was removed on Day 4 and his patient controlled analgesia (PCA) was withdrawn the following day.

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On Day 4 his wife noted a mild discrepancy in his pupillary sizes. On review his right pupil was mildly constricted with subtle ptosis. Due to concern for Horner’s syndrome, a CT Carotid Angiogram was arranged. The CT identified a right sided paravertebral haematoma at the thoracic inlet, along the expected course of the right sympathetic trunk; however there was no evidence of carotid injury (Figs. 1 and 2). Upon extensive evaluation of his initial thoracic CT, a larger haematoma was notable in the same region. There was no pupillary abnormality noted on his admission nor on his tertiary survey performed the following day. He was treated non-operatively and discharged; when followed up 6 weeks later he had a normal ophthalmological examination.

Discussion

Horner’s syndrome was first documented by Swiss ophthalmologist, Johann Freidrich Horner, in 1869 [1]. Horner’s syndrome is a collection of signs produced by interruption of the sympathetic pathway from the central nervous system to the eye and its adnexa [2]. The diagnosis is based on clinical findings of a “classic triad” of ptosis, papillary miosis and anhidrosis; however some studies have shown that the typical triad is uncommon, with anhidrosis occurring in only 4% of cases [3]. There are associated further features including enophthalmos, facial flushing, arteriolar or venular dilatation, transient lowering of intra-ocular pressure and hemi-atrophy of the face [2]. The diagnosis of Horner’s syndrome can be confirmed after an ophthalmic examination and pharmacological testing, usually with apraclonidine [4]. There are other conditions that can present similar to and/or cause Horner’s syndrome (see Table 1) [5–7].

Horner’s syndrome is usually associated with penetrating neck injuries, however only rarely occurs in the blunt traumatic setting, 0.08% [8]. It has been found to be present in 7% of blunt carotid injury (BCI) patients [9]; with BCI occurring in 0.33–0.67% of all blunt trauma patients [10]. In BCI the internal carotid artery is most commonly involved, with a suspected rapid deceleration and subsequent hyper-extension and rotation of the neck producing an intimal tear. BCI is associated with subsequently high morbidity and mortality rates. Death can result from transection of the carotid, or thrombosis secondary to a pseudo-aneurysm leading to massive strokes; with reported mortality from BCI as high as 31–57% [9,11]. Associated Horner’s syndrome is thought to occur due to disruption of the carotid sheath, injuring the posteriorly embedded sympathetic chain’s third order neurons [2]. In our case report we have direct identification of a compressive haematoma on the sympathetic pathway’s second order neuronal fibres causing the patient’s transient symptoms and signs.

First order neuronal fibres arise from the postero-lateral hypothalamus, descend through the brainstem and terminate at the ciliospinal centre of Budge (in the cervical spinal cord). Second order neuronal fibres travel through the cervical sympathetic chain and stellate ganglion, terminating in the superior cervical ganglion (Fig. 3). Third order sympathetic fibres form a plexus surrounding the internal carotid artery and ascends into the cavernous sinus, running a short course on the sixth cranial nerve and follows the ophthalmic division of the trigeminal nerve to the orbit, where it supplies the iris dilator muscles and the smooth muscle fibres of the upper and lower lids. Vasomotor and sweat gland fibres to the face have a separate path, following the external carotid artery after leaving the ganglion to supply the ipsilateral face.

The cervical part of the sympathetic trunk ascends from the thorax and crosses the medial side of the neck at the first rib (near its’ head). The inferior cervical ganglion lies behind the commencement of the vertebral artery, and usually fuses with the first thoracic ganglion to form the stellate ganglion. This mass lies over the front of the first rib neck, up to 10x5mm in size. From the stellate ganglion,
fibres run up medial to the vertebral artery and Vagus nerve, posterior to the carotid sheath. The cervical sympathetic trunk then ends at the superior cervical ganglion at the level of common carotid bifurcation [12].

Our patient’s transient right-sided Horner’s syndrome resulted from a para-vertebral haematoma at the thoracic inlet, secondary to the fractured first right rib, causing a direct compressive effect on the second order neuron fibres. We non-operatively managed this patient, allowing resorption of the haematoma which resulted in resolution of the patient’s condition.

Other examples of Horner’s syndrome secondary to diffuse external compressive forces have been described post insertion of intercostal chest drains [10]. In the majority of these cases when the tubes are repositioned this has led to improvement of the patient’s symptoms.

Table 1
Differential diagnosis of Horner’s syndrome.

| First order neuron lesions |
|---------------------------|
| Arnold-Chiari malformations |
| Basal meningitis |
| Basal skull tumours |
| Cerebrovascular accidents |
| Demyelinating disease, e.g. MS |
| Intra-pontine haemorrhage |
| Syringomyelia |
| Vertebrobasilar artery insufficiency causing lateral medullary syndrome |

| Second order neuron lesions |
|-----------------------------|
| Tumours (Pancoast, thyroid adenoma, neurofibroma, metastasis) |
| Aneurysm/dissection of Aorta, Subclavian or Common carotid artery |
| Neck trauma or post-surgical damage |

| Third order neuron lesions |
|---------------------------|
| Cluster headache |
| Carotid artery dissection |
| Nasopharyngeal tumours |
| Cavernous sinus mass |
| Herpes Zoster |
| Otitis media |
| Trauma or post-surgical damage |
| Raeder para-trigeminal syndrome |
Finally, it is important to be aware that such injuries can occur in blunt trauma without necessarily the presence of underlying carotid injury.

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