The hazards of being a gentleman farmer: a case of transient Horner’s syndrome

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Introduction

We present a case highlighting that shotgun shooting may result in cervical sympathetic chain stretch resulting in transient Horner’s syndrome. Shooters should therefore be aware of this rare complication and adapt their mount to avoid untoward insult to the sympathetic nerve chain.

Case history

A 48-year-old farmer presented to our emergency department with a 12 h history of a right temporal headache, nausea and right eye lid droop. He denied any history of weight loss or recent injury or pain involving his arms, shoulders, or neck. He had no relevant medical history, he was a non-smoker, and was not taking any medication. The preceding day he had been involved in his regular sport of game shooting.

On examination he was found to have right incomplete ptosis, miosis (2 mm smaller in diameter from the left pupil), enophthalmos and right-sided facial anhidrosis. There was no bruising, swelling or tenderness along the right neck or shoulder. His pulse was 76 beats per minute and his supine blood pressure was 168/103 mmHg. There were no other untoward clinical signs. The electrocardiogram confirmed sinus rhythm. A chest radiograph and computerized tomography (CT) of the head were within normal limits. Carotid Doppler ultrasound studies in conjunction with brain and cervical magnetic resonance imaging with contrast-enhanced angiography and diffusion-weighted imaging (MRI/MRA) were performed the day after admission to hospital. This confirmed normal vasculature and excluded dissection of the cervical and petrous sections of the internal carotid artery. Furthermore, no spinal cord insult or soft tissue pathology or acute ischaemic infarcts or lesions were identified. The apices of both lungs were adequately visualized.

The patient was discharged home on aspirin 75 mg daily, and within four weeks the symptoms and signs had completely resolved.

Discussion

Horner’s syndrome consists of a constellation of signs resulting from oculosympathetic pathway disruption. Aside from the classic triad of ipsilateral partial ptosis, miosis and anhidrosis, other signs that may occur include: enophthalmos, conjunctival hyperaemia, facial flushing, elevation of the lower eye lid (upside-down ptosis) and dilation lag.1,2

The oculosympathetic pathway consists of three neurons. The first-order (central) neurons begin in the posterolateral hypothalamus, descend through the brainstem and synapse...
within levels C8–T2 of the intermediolateral gray matter of the spinal cord, also known as the cilioparasympathetic nucleus. The central neurons’ pathway through the brainstem is not, however, as clearly delineated in the literature as the second- and third-order neurons. Lesions involving first-order neurons are not as common, though they are more readily localized as they are often accompanied by corresponding symptoms and signs. Second-order (preganglionic) neurons exit the spinal cord through C8–T2 ventral roots, proceed through the cervical sympathetic chain before synapsing in the superior cervical ganglion which is located posterior to the internal carotid artery at the level of the second and third cervical vertebrae. These neurons share important anatomical relationships with several structures in the neck, including the apex of the lung and the brachial plexus. Lesions involving these neurons can arise from head and neck trauma, iatrogenically after regional surgery or following spinal anaesthesia, or from malignant processes particularly apical lung and mediastinal tumours.

The third-order (postganglionic) neurons travel within the adventitia of the internal carotid artery, proceed into the cavernous sinus and then follow the ophthalmic division of the trigeminal nerve as it passes through the superior orbital fissure towards the iris. The close relationship of these neurons with the internal carotid artery makes it possible to establish an essential diagnosis to consider, especially when Horner’s syndrome occurs in conjunction with facial pain or a headache.

Diagnosing the cause of Horner’s syndrome mainly relies on the presenting history and radiological imaging. Brain and cervical MRI/MRA or CT angiography have better outcomes in identifying vascular lesions as opposed to ultrasound, which studies have shown to be less reliable and operator dependent. Pharmacological testing with hydroxyamphetamine eye drops may also help localize lesions involving the third-order neurons. However, this test does not establish the cause for the Horner’s syndrome and hence was not utilized in our patient.

**Conclusion**

Our case report demonstrates that shotgun shooting may result in cervical sympathetic chain stretch resulting in transient Horner’s syndrome. Correctly ‘mounting’ a shotgun is essential not only for recoil management, but also for accurate aim. The gun is placed high in the shoulder, with the heel of the butt sitting slightly above and pulled down into the shoulder and the base of the neck (which puts strain on the cervical sympathetic chain). The cheek is then placed firmly into the stock with the neck extended.

Shooters should therefore be aware of this rare complication and adapt their mount to avoid an upward insult to the sympathetic nerve chain that may result in Horner’s syndrome. However, any adaptation to one’s natural mount may be detrimental to aim and hence success out in the field.

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