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Clinical Communications: Adults

HORNER'S SYNDROME AFTER SCALENE BLOCK AND CAROTID DISSECTION

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□ **Abstract—Background: Horner's syndrome refers to the clinical triad of ptosis, miosis, and anhidrosis resulting from disruption of the ocular and facial sympathetic pathways. A myriad of etiologies can lead to Horner's syndrome; awareness of the underlying anatomy can assist physicians in identifying potential causes and initiating appropriate care. Case Report: Two patients presented to our Nashville-area hospital in 2014. Patient 1 was a 26-year-old man who noticed facial asymmetry one day after an outpatient orthopedic procedure. His symptoms were attributed to his posterior interscalene anesthesia device; with deactivation of this device, the symptoms rapidly resolved. Patient 2 was a 42-year-old man who presented to our emergency department with persistent headache and ptosis over several weeks. Computed tomography angiography revealed ipsilateral carotid dissection and the patient was admitted for further management. Why Should an Emergency Physician Be Aware of This?: The pathologies underlying Horner's syndrome are exceedingly diverse. Although classic teaching often focuses on neoplastic causes, and more specifically Pancoast tumors, neoplasms are discovered only in a small minority of Horner's syndrome cases. Other etiologies include trauma, cervical artery dissection, and infarction. With a better understanding of the pertinent anatomy and array of possible etiologies, emergency physicians may have more success in identifying and treating the causes of Horner's syndrome.** © **2016 Elsevier Inc.**

□ **Keywords—Horner's syndrome; interscalene block; carotid artery dissection**

INTRODUCTION

Horner's syndrome, or oculosympathetic paresis, is a constellation of symptoms related to the disruption of the sympathetic pathways innervating the head, face, and eye. Classically, Horner's syndrome manifests with the triad of ptosis, miosis, and anhidrosis; in some cases of Horner's syndrome, enophthalmos and vascular dilatation may also be seen.

Understanding Horner's syndrome requires knowledge of the anatomic distribution of the sympathetic tracts. First-order neurons descend from the hypothalamus to the cervical spine. Second-order neurons exit the spinal cord, course over the lung apex, and then ascend to the superior cervical ganglion. Third-order neurons then ascend within the carotid sheath, through the cavernous sinus, and alongside the distal trigeminal nerve before innervating the dilator pupillae and the levator palpebrae superioris.

Given this anatomy, Horner's syndrome is further divided into pre- and postganglionic forms. Preganglionic refers to first- or second-order syndromes, which present with the full classic triad of symptoms. Postganglionic refers to third-order Horner's syndrome. The majority of fibers related to sweat production do not follow the internal carotid artery (ICA), and instead branch off at the superior cervical ganglion. Thus, Horner's syndrome caused by lesions distal to the ganglion may show anhidrosis limited to the lateral nose and medial forehead or no anhidrosis at all (1).

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CASE REPORT 1

A 26-year-old man presented to our emergency department one day after arthroscopic repair of a left shoulder Bankart fracture. His procedure had been uncomplicated, and he had been discharged on a left posterior interscalene brachial plexus continuous pain pump. Overnight, he had developed facial asymmetry, with ptosis and miosis of his left eye. He became anxious and diaphoretic, and then realized he had anhidrosis of his left face. He further noted numbness in his left shoulder, neck, and lower face.

On our evaluation, we noted ptosis of the left eyelid by about 2–3 mm, and miosis by a similar amount. The patient's left face was warm and dry when compared to the right. A photograph of his presentation is seen in Figure 1. The remainder of his cranial nerve examination, apart from ptosis and miosis, was entirely intact. He had appropriate speech, hearing, and no visual changes. Further evaluation revealed a functional left posterior interscalene pump. The patient stated the pump had been providing reasonable analgesia. This is shown in Figure 2.

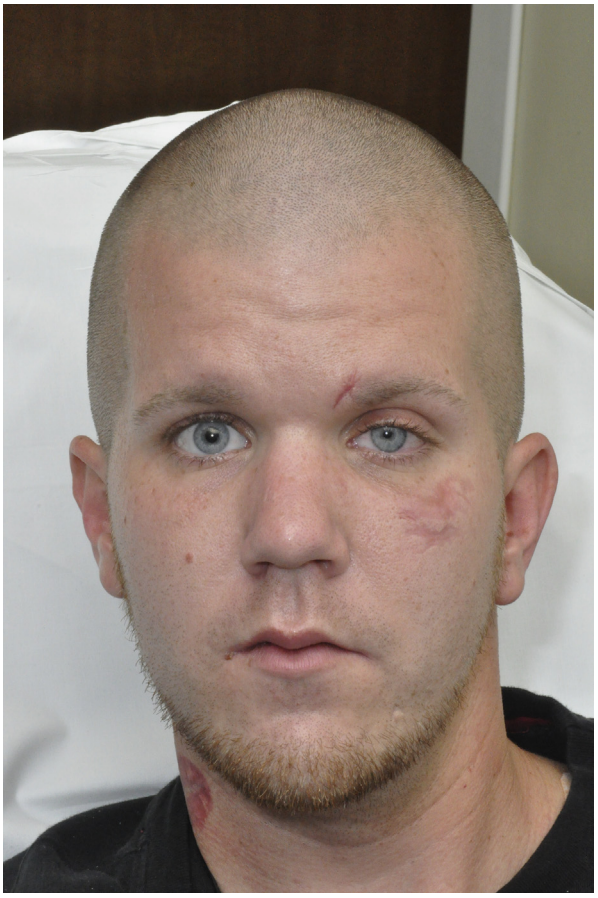

Figure 1. Case 1. Presentation.

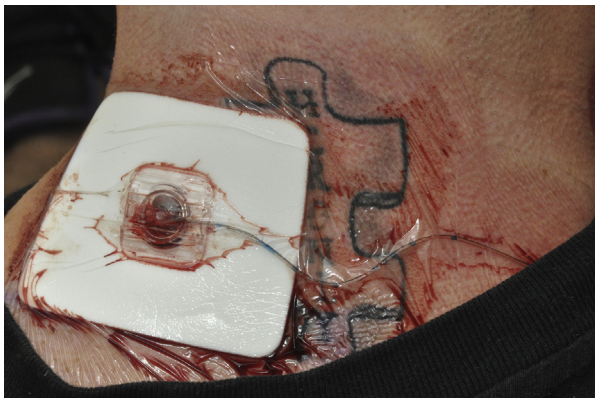

Figure 2. Case 1. Presence of posterior interscalene block.

We evaluated the patient with a chest x-ray, which was negative, and subsequently considered the possibility that his Horner's syndrome could be related to his posterior interscalene device. After consulting with anesthesia, we deactivated the device, and over the course of several hours, the patient's symptoms began to resolve. We sent him to surgical clinic, where the device was removed in its entirety, and over the next few days, the patient recovered to full neurologic baseline.

CASE REPORT 2

A 42-year-old man presented to our emergency department with a 2-week history of left eyelid droop and left-sided headache. He noticed the eyelid droop 2 days after the onset of headache. He had previously presented to another emergency department and received a noncontrasted computed tomography (CT) scan of the head, which was negative. He was referred to a neurologist and had a normal outpatient magnetic resonance imaging (MRI)/magnetic resonance angiography of the brain. As his symptoms persisted, he continued to be concerned about having a stroke and decided to seek further emergency care.

On our evaluation, the patient was noted to have obvious ptosis of his left eyelid along with miosis of the left pupil, as shown in Figure 3. Apart from this, his entire physical examination and thorough neurologic examination were normal. Interestingly, though not seen on examination, the patient noted that he was not sweating on the left forehead when he looked in the mirror earlier in the day.

Given the patient's history and negative MRI, we were concerned about acute Horner's syndrome caused by ICA dissection. CT angiography (CTA) was obtained and can be seen in Figure 4. As noted by the arrow, CTA confirmed dissection of the left ICA.