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Clinical Observations

Horner Syndrome After Tonsillectomy: An Anatomic Perspective

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ABSTRACT

BACKGROUND: Horner syndrome after tonsillectomy has been reported rarely in the literature. Furthermore, postoperative Horner syndrome lasting more than a 1 month is an even more rare occurrence. **PATIENT:** We present a persistent postoperative Horner syndrome in a 5-year-old child following tonsillectomy. **RESULTS:** Clinical diagnosis of Horner syndrome is confirmed pharmacologically, and damage to the oculosympathetic pathway at the level of the superior cervical ganglion is radiographically demonstrated. **CONCLUSION:** Conventional monopolar electrosurgical dissection led to irreversible damage of ganglionic neural tissue at the level of the palatine tonsilar fossa and permanent Horner syndrome.

Keywords: Horner syndrome, anisocoria, ptosis, meiosis, anhidrosis, tonsillectomy, oculosympathetic paresis

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Introduction

Horner syndrome is characterized by ipsilateral ptosis, meiosis, and anhidrosis resulting from a disturbance of the oculosympathetic pathway. Acquired interruption of the sympathetic innervation to the pupillary dilator muscle, Mueller muscle in the upper eyelid and its analogous muscle in the lower eyelid, and the facial sweat glands can occur from primary (central), secondary (preganglionic), or tertiary (ganglionic and postganglionic) nerve lesions. Tertiary lesions can variably affect sudomotor pathways to the face, and therefore, tertiary Horner syndrome may not include facial anhidrosis. In accordance with common custom, we will refer to this scenario of oculosympathetic paresis as Horner syndrome throughout this report. Anisocoria that is more apparent in dim illumination is the key to diagnosis. Horner syndrome can be pharmacologically confirmed with the cocaine test, and further localization is performed with radiographic imaging when appropriate. Horner syndrome after tonsillectomy has been reported rarely.[1-5]

Patient Description

A 5-year-old boy underwent tonsillectomy and adenoidectomy at an outside institution. The procedure was performed under general anesthesia using standard technique with monopolar diathermy for tissue dissection and hemostasis. The child's parents noted a droopy left upper eyelid immediately after the surgery in the recovery room. The child was discharged on a standard oral course of amoxicillin/clavulanate.

Eight days postoperatively, the child presented to the pediatric emergency room at Cohen's Children's Medical Center for a persistent "droopy eyelid." On evaluation, his visual acuity was 20/20 bilaterally. Pupillary examination revealed 2-mm anisocoria in the light, and 3-mm anisocoria in dim illumination, with the left being the meiotic pupil. There was no relative afferent pupillary defect. He had 3.0 mm of left upper eyelid ptosis. Both anterior segments and fundi were unremarkable. There was no iris heterochromia. A diagnosis of acquired left Horner syndrome was made (Fig 1).

The child was evaluated 3 days later in the neuro-ophthalmology clinic, and his examination was unchanged. Facial anhidrosis after exercise was not observed. After the instillation of 10% cocaine into the eyes

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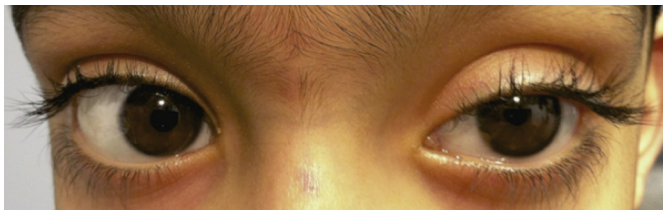

FIGURE 1.
Left-sided meiosis and ptosis, consistent with Horner syndrome, postoperative day 8. (Color version of this figure is available in the online edition.)

bilaterally, the left pupil did not react, whereas the right pupil dilated, resulting in a 4-mm anisocoria. At this time, the Horner syndrome was attributed to postoperative inflammatory changes. However, we elected to pursue neuroimaging to definitively rule out other more ominous etiologies such as carotid sheath hematoma, carotid dissection, and although clinically unlikely, neuroblastoma.

Imaging results

On postoperative day 14, the child underwent magnetic resonance imaging and magnetic resonance angiography. Brain imaging was unremarkable. Neck imaging revealed no masses, vascular stenosis, occlusion, or carotid dissection. However, T_1-weighted images of the neck demonstrated soft tissue thickening and infiltration within the left parapharyngeal, retropharyngeal, and left carotid spaces (Fig 2). Contrast-enhanced computed tomography of the neck one day later revealed low attenuation soft tissue swelling involving the left carotid space and left parapharyngeal and retropharyngeal spaces. No discrete fluid collection or

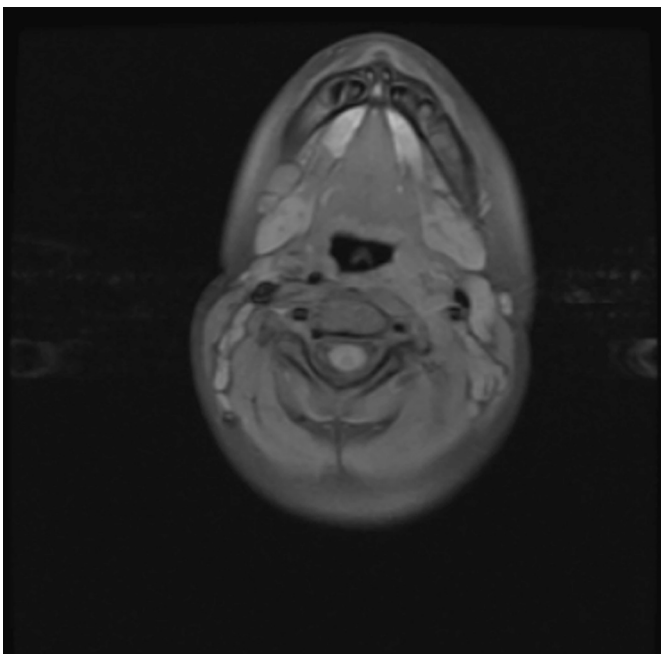

FIGURE 2.
MRI neck with contrast, performed on postoperative day 14. Selected fat saturated T_1-weigted axial image through the neck demonstrates soft tissue thickening and infiltration of left parapharyngeal space and carotid space adjacent to distal extracranial internal carotid artery. MRI, magnetic resonance imaging.

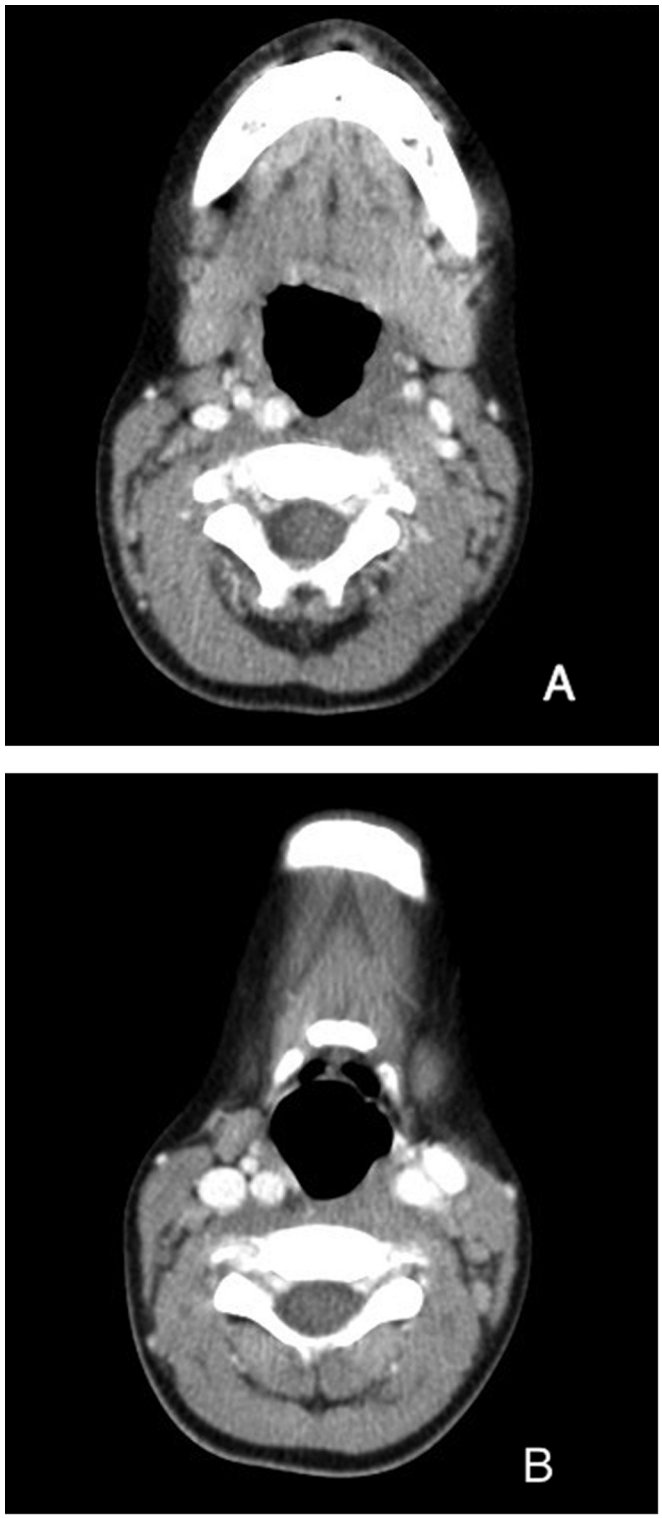

FIGURE 3.
CT neck with contrast, performed on postoperative day 15. Selected axial image demonstrates asymmetric low attenuation soft tissue swelling involving left parapharyngeal and retropharyngeal spaces and left carotid space, without fluid collection or rim enhancement (A). Medial deviation of the right common carotid artery (B) and right internal carotid artery (A) is observed in the right parapharyngeal and retropharyngeal locations, an anatomic variant. Medial deviation also involves the left common carotid artery into the left retropharyngeal soft tissues, an anatomic variant (B). CT, computed tomography.