Acquired Horner syndrome secondary to cervical disc herniation

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ABSTRACT

Background: Horner syndrome (HS), defined by the triad of ptosis, miosis, and facial anhidrosis, arises from disruption of the oculosympathetic pathway at any point along its course. The most frequently documented causes include trauma, neoplasms, and iatrogenic injury. Conversely, cervical myelopathy resulting from disc herniation is an uncommon etiology of HS.

Case Presentation: We describe an unusual presentation of HS in a 64-year-old woman with a large C5-C6 central disc herniation causing severe stenosis and cervical myelopathy.

Conclusion: This case highlights the importance of considering HS as a potential clinical indicator of cervical myelopathy. Prompt recognition and timely referral for surgical decompression are essential, as the recovery is influenced by both the duration and severity of sympathetic fiber compression.

Keywords: Horner syndrome, cervical disc herniaton, cervical myelopathy.

Introduction

Horner syndrome (HS) is a classic neurological condition, typically presenting with unilateral mild upper eyelid ptosis, miosis, and anhidrosis, resulting from disruption of the oculosympathetic pathway [1]. This pathway comprises a three-neuron chain: the first-order neuron, that originates in the hypothalamus and descends through the spinal cord; the second-order neuron, that extends from the sympathetic trunk, passing through the brachial plexus and lung apex, before ascending to the superior cervical ganglion; and the third-order neuron, that follows the carotid artery into the cavernous sinus, traversing the orbit to innervate the iris dilator and Müller's muscle, contributing to slight upper lid elevation and lower lid retraction [1].

Although often associated with subtle clinical signs, HS may signal life-threatening conditions, including central nervous system tumors, intracranial hemorrhage, stroke, demyelinating diseases, arterial dissection, and malignancies [2].

Cervical myelopathy due to a disc herniation commonly manifests with difficulty in walking, spastic weakness of upper limbs and hands, hyperreflexia, and patchy sensory loss; however, in rare cases, it may lead to a different presentation, like HS [3]. A thorough clinical history is essential for identifying the lesion's etiology and guiding diagnostic evaluation.

Case Presentation

A 64-year-old woman initially presented to an ophthalmologist, for evaluation of left eye ptosis. On examination, she was found to have left eye ptosis, and miosis. The apraclonidine 0.5% test was performed, revealing a positive test, with reversion of ptosis and anisocoria, confirming HS. The patient was sent to the emergency room of a local health unit hospital for further evaluation.

Her past medical history included arterial hypertension, dyslipidemia, type 2 diabetes mellitus, and lumbar degenerative pathology, which had been evaluated by orthopedics and had no surgical indication. No other relevant past medical history.

At hospital admission, she presented a blood pressure of 146/78 mmHg, a heart rate of 76 beats per minute, apyretic, and a pulse oximetry of 98% on room air. She had left miosis and 2 mm upper lid ptosis. The neurological examination was otherwise unremarkable and with no clinical signs of myelopathy. The laboratory studies were largely

unremarkable except for isolated serum gamma-glutamyl transferase elevation (178 U/l). Cerebral and supraaortic vessels angio-computed tomography (angio-CT) was performed, excluding an acute ischemic or hemorrhagic event and carotid dissection, identifying no significant atherosclerosis at the carotid bifurcation. She was admitted to the Internal Medicine ward to complete a clinical investigation.

A neck ultrasound was performed, revealing no neck masses or thyroid enlargement. Additionally, thoracic CT revealed no pulmonary masses, excluding Pancoast syndrome. Cranioencephalic angio-magnetic resonance imaging (MRI) was performed, excluding ischemic, hemorrhagic or structural lesions in cerebral parenchyma.

MRI of the cervical spine revealed a large C5-C6 central disc herniation causing severe stenosis and cord compression, along with STIR cord signal change suggesting edema versus gliosis (Figure 1). In the absence of overt clinical signs of myelopathy at presentation, the diagnosis of cervical myelopathy was established on a radiological basis.

Given the physical exam and imaging findings, in consultation with neurosurgery, the patient was elected to undergo a C5-C6 anterior cervical discectomy and cervical disc replacement. At postoperative follow-up, progressive improvement was observed. Clinical assessments at 3 and 6 months demonstrated complete resolution of miosis and near-complete resolution of ptosis. At the most recent follow-up, at 12 months postoperatively, symptoms had stabilized, with no further improvement noted.

Discussion

HS arises from disruption of the sympathetic nervous pathway at any point along its three-order neuron pathway[1].

Figure 1. *Cervical spine MRI revealing a large C5-C6 central disc herniation.*

Sympathetic fibers originate in the posterolateral hypothalamus and descend through the brainstem and cervical spinal cord, reaching the ciliospinal center of Budge, located in the intermediolateral cell columns between C8 and T2. From there, pre-ganglionic sympathetic neurons exit the spinal cord, travel along the sympathetic chain over the apex of the lung, and ascend within the carotid sheath. They synapse in the superior cervical ganglion, near the common carotid artery bifurcation [1].

Most recognized etiologies of HS occur somewhere along this sympathetic pathway [2]. Malignancies of the lung and breast represent almost one quarter of all pre-ganglionic HS cases, with the classic neoplastic association being a Pancoast tumor [2].

Post-ganglionic fibers from the superior cervical ganglion ascend along the adventitia of the internal carotid artery, forming the internal carotid nerve or sympathetic plexus. This plexus supplies the iris dilator muscle and Müller's muscle in the upper and lower eyelids, and also play a role in ipsilateral facial sweating [4]. Consequently, disruption of the sympathetic pathway produces the hallmark triad of miosis, ptosis, and anhidrosis seen in HS.

Post-ganglionic sympathetic disruption secondary to internal carotid artery dissection represents one of the most frequent causes of HS. Additional documented etiologies include cervical neuroblastoma, lymph node enlargement, and iatrogenic damage [1,4,5].

In this case, a systematic differential diagnosis of HS was undertaken, given the wide range of potentially serious underlying causes. Vascular etiologies, including carotid artery dissection and acute cerebrovascular events, were excluded by angio-CT of the cerebral and supraaortic vessels. Neoplastic causes were also considered and ruled out through cervical ultrasound and thoracic CT, excluding neck masses, thyroid pathology, and apical lung lesions. Central nervous system causes were further excluded by cranioencephalic angio-MRI. In the absence of vascular, neoplastic, or intracranial abnormalities, cervical spine MRI ultimately identified the compressive degenerative lesion responsible for the patient's sympathetic dysfunction.

Our patient presented with a central C5-C6 central disc herniation causing severe spinal canal stenosis and cervical myelopathy, likely affecting the sympathetic fibers traveling through the cervical spinal cord. Following surgical decompression, our patient's symptoms showed partial improvement.

Although the anatomical pathway of the oculosympathetic chain is well established, involvement due to cervical disc herniation remains exceedingly rare, and there are few reported cases of an acquired HS associated with a herniated cervical disc[3,6]. Most published cases describe lateral disc herniations or are associated with additional neurological deficits. In contrast, the present case involves a large central C5-C6 disc herniation causing severe canal

stenosis and radiological myelopathy, in the absence of overt clinical signs of myelopathy. Identification of a degenerative disc lesion as the primary mechanism of sympathetic impairment, therefore, expands the spectrum of structural spinal causes clinicians should consider.

Moreover, the partial improvement observed following decompression underscores the value of prompt therapeutic intervention. The extent to which symptoms resolve is influenced by both the severity and duration of sympathetic fiber compression, and delayed diagnosis increases the risk of permanent neurological injury [7].

Conclusion

This reinforces the clinical importance of recognizing HS as a potential early manifestation of cervical myelopathy, even in the absence of overt sensory or motor deficits. Patients presenting with HS should undergo a thorough clinical evaluation, and early neuroimaging is essential to exclude compressive spinal lesions. This case adds to the limited literature identifying cervical disc herniation as a potentially reversible structural cause of HS and underscores the need for heightened clinical awareness to facilitate earlier diagnosis and intervention.

What is new?

Cervical disc herniation can rarely disrupt the oculosympathetic pathway, leading to HS – an uncommon but clinically relevant presentation of cervical myelopathy.
Early cervical MRI is essential when HS occurs together with neurological symptoms, as delayed diagnosis may result in irreversible spinal cord injury.
Surgical decompression can lead to partial symptomatic recovery, highlighting the importance of timely intervention before permanent sympathetic or spinal cord damage occurs.

List of Abbreviations

angio-CT   Angio-computed tomography
HS   Horner syndrome
MRI   Magnetic resonance imaging

Consent for publication

Written informed consent was obtained from the patient for publication of their clinical history and associated data.

Ethical approval

Ethical approval is not required at our institution to publish an anonymous case report.

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Summary of the case

1	Patient (gender, age)	64 years, female
2	Final diagnosis	Acquired HS secondary to cervical disc herniation
3	Symptoms	Left eye ptosis and miosis
4	Medications	None
5	Clinical procedure	Surgical decompression
6	Specialty	Internal Medicine and Neurosurgery