



unremarkable except for isolated serum gamma-glutamyl transferase elevation (178 U/l). Cerebral and supraortic 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.

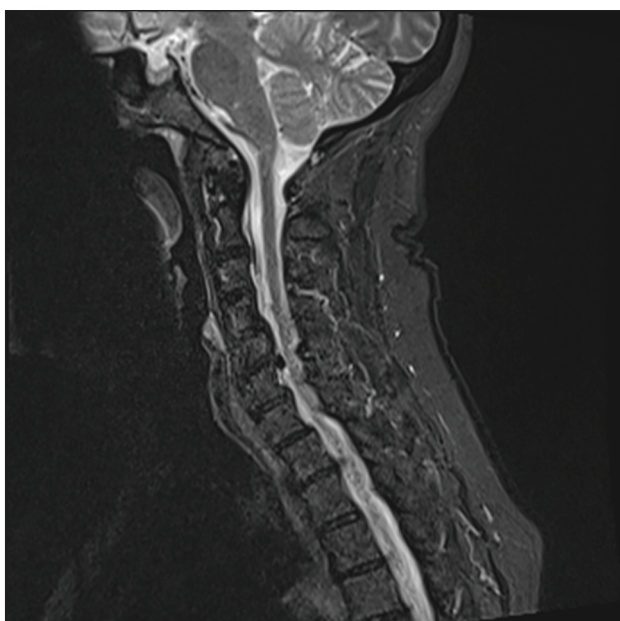
A neck ultrasound was performed, revealing no neck masses or thyroid enlargement. Additionally, thoracic CT revealed no pulmonary masses, excluding Pancoast syndrome. Cranioencephalic angio-magnetic resonance imaging (MRI) was performed, excluding ischemic, hemorrhagic or structural lesions in cerebral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

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## List of Abbreviations

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

## Conflict of interests

The authors declare that there is no conflict of interest regarding the publication of this article.

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## 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