

1 Acquired Horner syndrome 2 secondary to cervical disc 3 herniation

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7 ABSTRACT

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

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

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

16 **Keywords:** Horner syndrome, cervical disc herniation, cervical myelopathy.

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17 **Introduction** 40
18 Horner syndrome (HS) is a classic neurological condition, 41
19 typically presenting with unilateral mild upper eyelid ptosis, 42
20 miosis, and anhidrosis, resulting from disruption of the
21 oculosympathetic pathway [1]. This pathway comprises a
22 three-neuron chain: the first-order neuron, that originates
23 in the hypothalamus and descends through the spinal cord;
24 the second-order neuron, that extends from the sympathetic
25 trunk, passing through the brachial plexus and lung
26 apex, before ascending to the superior cervical ganglion;
27 and the third-order neuron, that follows the carotid artery
28 into the cavernous sinus, traversing the orbit to innervate
29 the iris dilator and Müller's muscle, contributing to slight
30 upper lid elevation and lower lid retraction [1].

31 Although often associated with subtle clinical signs, 43
32 HS may signal life-threatening conditions, including 44
33 central nervous system tumors, intracranial hemorrhage, 45
34 stroke, demyelinating diseases, arterial dissection, and 46
35 malignancies [2].

36 Cervical myelopathy due to a disc herniation 47
37 commonly manifests with difficulty in walking, spastic weakness 48
38 of upper limbs and hands, hyperreflexia, and patchy 49
39 sensory loss; however, in rare cases, it may lead to a 50

40 different presentation, like HS [3]. A thorough clinical history 41 is essential for identifying the lesion's etiology and 42 guiding diagnostic evaluation.

Case Presentation

43 A 64-year-old woman initially presented to an ophthalmologist, 44 for evaluation of left eye ptosis. On examination, 45 she was found to have left eye ptosis, and miosis. 46 The apraclonidine 0.5% test was performed, revealing a 47 positive test, with reversion of ptosis and anisocoria, 48 confirming HS. The patient was sent to the emergency room 49 of a local health unit hospital for further evaluation.

50 Her past medical history included arterial hypertension, 51 dyslipidemia, type 2 diabetes mellitus, and lumbar 52 degenerative pathology, which had been evaluated by 53 orthopedics and had no surgical indication. No other relevant 54 past medical history.

55 At hospital admission, she presented a blood pressure 56 of 146/78 mmHg, a heart rate of 76 beats per minute, apyretic, 57 and a pulse oximetry of 98% on room air. She had left 58 miosis and 2 mm upper lid ptosis. The neurological examination 59 was otherwise unremarkable and with no clinical 60 signs of myelopathy. The laboratory studies were largely 61

62 unremarkable except for isolated serum gamma-glutamyl
 63 transferase elevation (178 U/l). Cerebral and supraaor-
 64 tic vessels angio-computed tomography (angio-CT) was
 65 performed, excluding an acute ischemic or hemorrhagic
 66 event and carotid dissection, identifying no significant
 67 atherosclerosis at the carotid bifurcation. She was admit-
 68 ted to the Internal Medicine ward to complete a clinical
 69 investigation.

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

76 MRI of the cervical spine revealed a large C5-C6 cen-
 77 tral disc herniation causing severe stenosis and cord com-
 78 pression, along with STIR cord signal change suggesting
 79 edema versus gliosis (Figure 1). In the absence of overt
 80 clinical signs of myelopathy at presentation, the diagnosis
 81 of cervical myelopathy was established on a radiological
 82 basis.

83 Given the physical exam and imaging findings, in con-
 84 sultation with neurosurgery, the patient was elected to
 85 undergo a C5-C6 anterior cervical discectomy and cervi-
 86 cal disc replacement. At postoperative follow-up, progres-
 87 sive improvement was observed. Clinical assessments at 3
 88 and 6 months demonstrated complete resolution of miosis
 89 and near-complete resolution of ptosis. At the most recent
 90 follow-up, at 12 months postoperatively, symptoms had
 91 stabilized, with no further improvement noted.

92 Discussion

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



150
 151 **Figure 1.** Cervical spine MRI revealing a large C5-C6 central
 152 disc herniation.

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

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

109 Post-ganglionic fibers from the superior cervical gan-
 110 glion ascend along the adventitia of the internal carotid
 111 artery, forming the internal carotid nerve or sympathetic
 112 plexus. This plexus supplies the iris dilator muscle and
 113 Müller's muscle in the upper and lower eyelids, and also
 114 play a role in ipsilateral facial sweating [4]. Consequently,
 115 disruption of the sympathetic pathway produces the hall-
 116 mark triad of miosis, ptosis, and anhidrosis seen in HS.

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

122 In this case, a systematic differential diagnosis of
 123 HS was undertaken, given the wide range of potentially
 124 serious underlying causes. Vascular etiologies, including
 125 carotid artery dissection and acute cerebrovascular events,
 126 were excluded by angio-CT of the cerebral and supraaor-
 127 tic vessels. Neoplastic causes were also considered and
 128 ruled out through cervical ultrasound and thoracic CT,
 129 excluding neck masses, thyroid pathology, and apical
 130 lung lesions. Central nervous system causes were further
 131 excluded by cranioencephalic angio-MRI. In the absence
 132 of vascular, neoplastic, or intracranial abnormalities, cer-
 133 vical spine MRI ultimately identified the compressive
 134 degenerative lesion responsible for the patient's sym-
 135 pathetic dysfunction.

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

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

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

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

164 Conclusion

165 This reinforces the clinical importance of recognizing HS
 166 as a potential early manifestation of cervical myelopathy,
 167 even in the absence of overt sensory or motor deficits.
 168 Patients presenting with HS should undergo a thorough
 169 clinical evaluation, and early neuroimaging is essential to
 170 exclude compressive spinal lesions. This case adds to the
 171 limited literature identifying cervical disc herniation as a
 172 potentially reversible structural cause of HS and under-
 173 scores the need for heightened clinical awareness to facil-
 174 itate earlier diagnosis and intervention.

175 What is new?

176 Cervical disc herniation can rarely disrupt the oculosympa-
 177 thetic pathway, leading to HS – an uncommon but clinically
 178 relevant presentation of cervical myelopathy.
 179 Early cervical MRI is essential when HS occurs together with
 180 neurological symptoms, as delayed diagnosis may result in
 181 irreversible spinal cord injury.
 182 Surgical decompression can lead to partial symptomatic
 183 recovery, highlighting the importance of timely interven-
 184 tion before permanent sympathetic or spinal cord damage
 185 occurs.

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 189 completion of this work.

190 List of Abbreviations

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

238 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

Conflict of interests

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Consent for publication

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Ethical approval

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 203
 204

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