

CASE REPORT

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Horner syndrome secondary to edema of the longus colli muscle: a case report

Avery Zhou^{1*} , Cory Nichols², Janda Paul^{2,3} and Aroucha Vickers^{2,4}

Abstract

Background: Horner syndrome is caused by disruption to any part of the oculosympathetic nerve supply, and is classically characterized by the triad of ptosis, miosis, and facial anhidrosis. Two cases of Horner syndrome secondary to longus colli edema have previously been reported in the literature; however, this is the first case of bilateral asymmetric longus colli inflammation causing unilateral Horner syndrome.

Case presentation: An 18-year-old male was found unresponsive in his car after a motor vehicle accident and was found to have new onset anisocoria with a miotic left pupil and left-sided ptosis consistent with Horner syndrome. Imaging was unremarkable except for the MRI neck soft tissues, which revealed abnormal increased signal intensity consistent with extensive edema in the left longus colli muscle and a shorter segment of edema in the right longus colli muscle.

Conclusion: The patient's presentation and imaging results suggest Horner syndrome secondary to edema of the longus colli muscle, as the second-order sympathetic innervation to the eye runs under the longus colli. Traumatic Horner syndrome from longus colli contusion is a rare and benign entity that may self-resolve as inflammation and compression decrease. Imaging of the soft tissues of the neck is vital in assessing this rare occurrence. More common and concerning etiologies should be excluded with proper vascular, cerebral and spinal imaging before attributing Horner syndrome to a benign cause.

Keywords: Horner syndrome, Anisocoria, Longus colli muscle, Case report

Background

Horner syndrome is a condition characterized by the triad of ptosis, miosis, and facial anhidrosis [1]. It is caused due to disruption of the sympathetic nerve supply, which typically includes tumors, trauma, aneurysm, and dissection [2, 3]. Two cases of Horner syndrome secondary to longus colli edema have been reported, one in the context of cervical spine surgery and the other secondary to ipsilateral longus colli contusion which simulated a tumor [4, 5]. However, to the best of our knowledge, this is the first case of bilateral asymmetric longus colli inflammation causing unilateral Horner syndrome.

Case presentation

An 18-year-old male with a history of polysubstance abuse was found unresponsive in a car crashed into a dumpster. He was unaccounted for approximately 7 h, and naloxone administration had no effect. The patient was brought to the hospital by emergency medical services and was intubated for airway protection. Initial findings were significant for leukocytosis, positive cannabinoids on urine toxicology screen, and rhabdomyolysis with a creatine kinase that peaked to 4131 U/L (22–198 U/L). Computed tomography (CT) of his head [Canon Aquilion Prime SP 80 (160) slice scanner, USA] was negative for acute intracranial abnormality, and CT of his cervical spine revealed no fractures. The patient was admitted into the intensive care unit, where on day 2 of hospitalization he was extubated.

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The patient remained combative and agitated throughout his initial hospitalization, and Neurology was consulted on day 4 for agitation and anisocoria. Upon examination on day 4, the patient was restless and did not provide much history aside from admitting to unspecified illicit drug use. He was afebrile but had been hypertensive with systolic blood pressures over 160 mmHg. An electroencephalography (EEG) showed no epileptiform morphology (XLTEK brain monitor Natus Neurology, Canada). On physical examination, the patient was alert and oriented and was noted to have anisocoria with a miotic left pupil which was more prominent in the dark, and mild left-sided ptosis. This presentation was consistent with a left Horner syndrome. The remainder of the neurological examination was unremarkable.

Neuroimaging was performed to assess the oculosympathetic pathway, which included a CT angiography of the head and neck, magnetic resonance imaging (MRI) brain with and without contrast, and MRI neck soft tissues with and without contrast (Siemens MRI Magnetometer Aera 1.5 T, Germany). Imaging studies were unremarkable, except for MRI neck soft tissues which revealed abnormal increased signal intensity consistent with extensive edema in the left longus colli muscle, as well as shorter segment edema in the right longus colli muscle (Figs. 1, 2). As the patient's hypertension and agitation had resolved, it was considered reasonable to trial the patient on high-dose steroids in attempt to relieve the likely causative edema in the left longus colli. Unfortunately the patient left against medical advice before efficacy could be assessed and was lost to follow-up.

Conclusions

The MRI neck soft tissue finding of edema along the tract of the sympathetic nerve supply to the eye, accompanied by the patient's presentation of anisocoria, suggests the diagnosis of Horner syndrome secondary to trauma. Horner syndrome is caused by a disruption to any part of the oculosympathetic nerve supply, and is classically characterized by the triad of ptosis, miosis, and facial anhidrosis. This pathway originates in the hypothalamus, descends through the brainstem, exits the cervicothoracic junction, ascends over the lung, and travels along the carotid to the sudomotor fibers of the face, the pupillary dilators, and Muller's muscle [2]. Within this pathway, the second-order sympathetic innervation to the eye runs under the longus colli muscle (Fig. 3) [6]. Soft tissue edema compressing

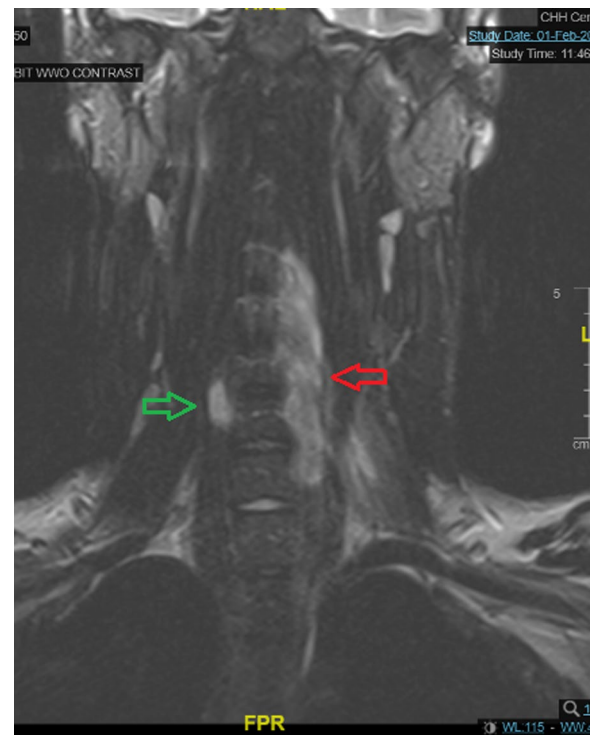


Fig. 1 MRI neck soft tissues with contrast, showing increased signal intensity in the left longus colli muscle from C4 to C7 (red arrow) and in the right longus colli muscle from T6 to T7 (green arrow)

this portion of the sympathetic nerve results in Horner syndrome of the ipsilateral side [3], as was seen in this case. Although the MRI also disclosed edema of the right longus colli muscle, this was less extensive than the left-side edema and thus did not manifest in symptoms. While there were several days that elapsed between the initial insult and MRI, and thus likely some degree of resolution of edema, it is believed that the asymmetry noted represented a close approximation of the original insult, as at no point from EMS contact to neurology consultation were there any documented pupillary abnormalities other than the Horner's miosis involved in this case.

Although the patient discharged himself before treatment efficacy could be determined, Horner syndrome due to benign causes such as soft tissue edema will likely self-resolve as inflammation and compression decrease. However, more common and concerning etiologies of Horner syndrome include brainstem ischemia, demyelinating plaques, Pancoast tumors, vascular dissections,

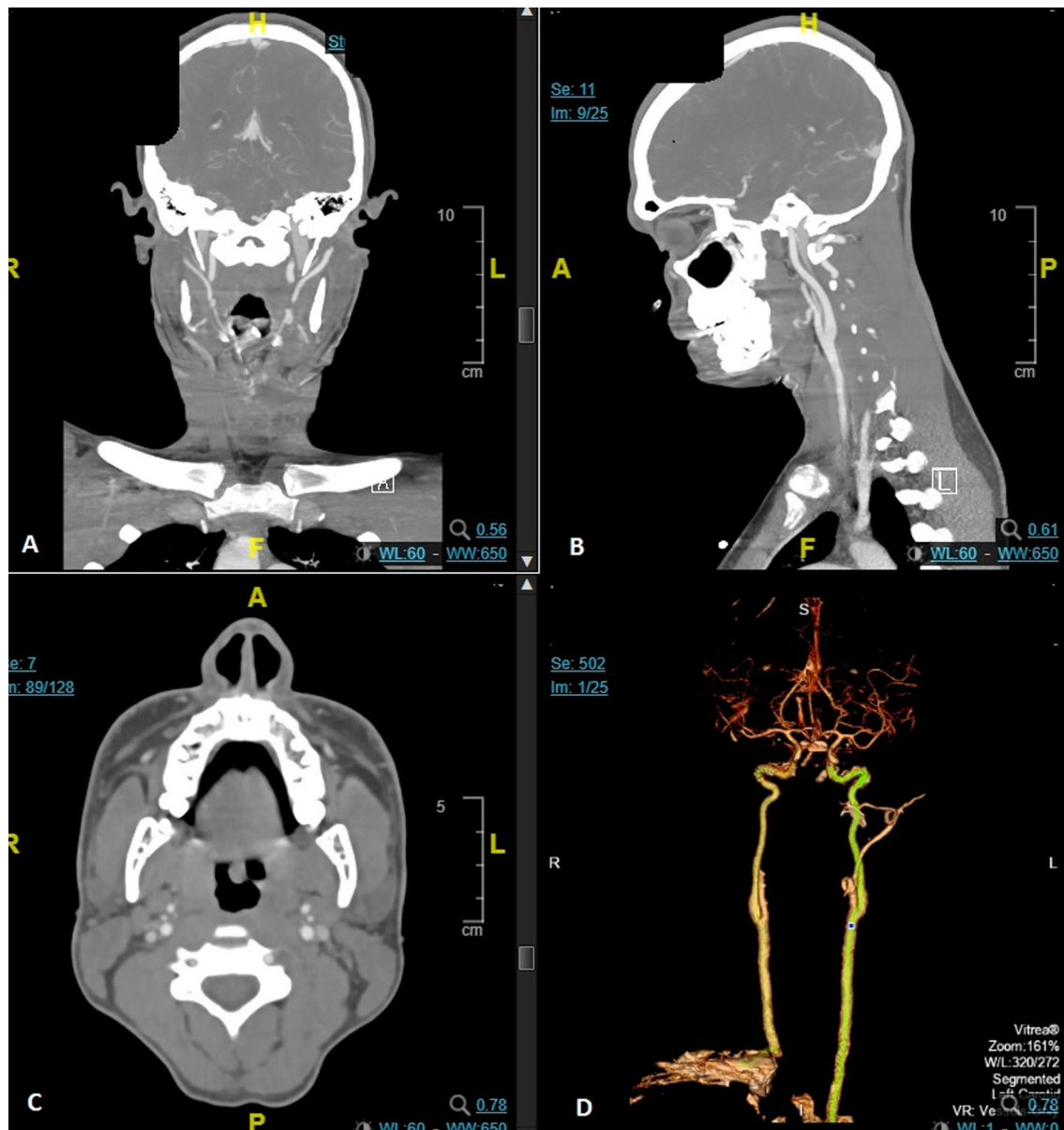
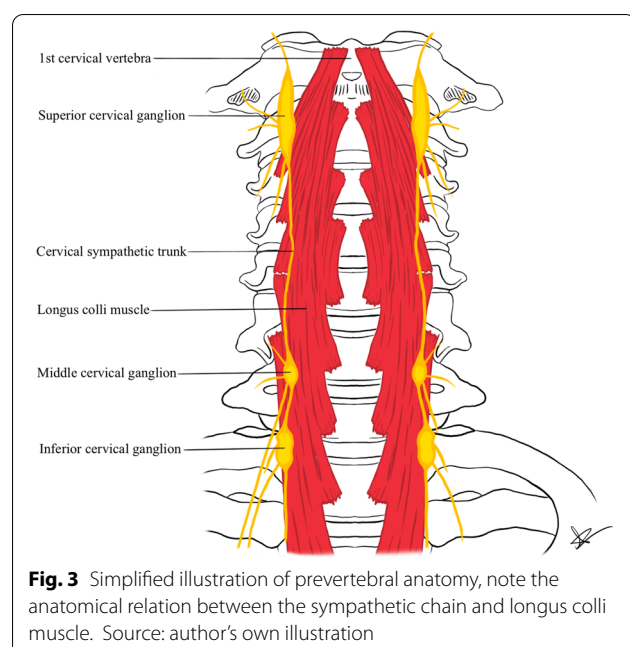


Fig. 2 A–C CT angiogram of the head and neck, showing no vascular lesions. **D** Maximal Intensity Projection (MIP), also demonstrating no vascular lesions

and cavernous sinus thrombosis [3, 7]. These diagnoses should be excluded before attributing Horner syndrome to a benign cause. Vascular and cerebral imaging are routinely performed for interrogation of Horner syndrome [8], and soft tissue imaging of the neck down to the level

of T2 can also be of utility as demonstrated in this case. To the best of our knowledge, this is the first case of bilateral asymmetric longus colli inflammation causing unilateral Horner syndrome.



Abbreviations

CT: Computed tomography; EEG: Electroencephalography; MRI: Magnetic resonance imaging.

Acknowledgements

Not applicable.

Author contributions

All authors designed the report, acquired the data, and analyzed the data. AZ and CN drafted the manuscript. JP and AV revised it for intellectual content. All authors read and approved the final manuscript.

Funding

The authors declare no sources of funding.

Availability of data and materials

Not applicable.

Declarations

Ethics approval and consent to participate

Case reports are exempt from IRB approval in our institution which is why no IRB approval was taken.

Consent for publication

Not applicable. This case report does not include identifiable data.

Competing interests

The authors declare that they have no competing interests.

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Received: 2 July 2022 Accepted: 30 September 2022
Published online: 22 October 2022

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