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Trends in Ophthalmology
Open Access Journal

Case Report

Horner Syndrome Due to Pharyngeal Foreign Body

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Received: May 27, 2019 **Published:** June 04, 2019

Abstract

Horner syndrome is a neurological pathology due to a disruption in the oculosympathetic pathway and it is characterized by ptosis, miosis and facial anhidrosis. We present the case of a 47-year-old patient with a history of pharyngeal foreign body who came to the emergency room complaining about ptosis in the left eye, which was not suspected by the preliminary imaging tests and finally was diagnosed as Horner Syndrome. This case highlights the importance of knowing the nerve pathway and the signs and symptoms that may occur depending on the level of the lesion, allowing the correct location of the damage.

Keywords: Horner syndrome; Sympathetic pathway; Pharyngeal foreign body

Introduction

Horner Syndrome (HS), first described by Dr. Claude Bernard in 1852, is caused by a damage in the oculosympathetic pathway, and its mainly characterised by unilateral ptosis and miosis, and occasionally ipsilateral hemifacial anhidrosis. Other signs and symptoms such as hemiparesis, oculomotor nerve palsy or cervical pain, may help to locate the damage [1].

Case Report

A 47-year-old male patient with a history of a fish bone removal located behind the left palatine tonsil by the Ears-Nose-Throat specialist, visited our emergency room one week after the last episode referring a 2-day acute onset drooping of the upper left eyelid. On examination we observed anisocoria, a smaller pupil size of the left eye compared to the right pupil worse in dark. Moreover, a left eyelid ptosis was noticed with a palpebral aperture of 8mm. No anhidrosis or other signs were noticed, and the patient did not refer other symptoms. Ocular examination under slit lamp including anterior chamber and fundus were normal. To locate the damage, we held a pharmacological eye-drop test using Apraclonidine 0.5% in both eyes, witnessing a pupil dilation in the left eye and a discrete pupil contraction in the right eye. Hydroxyamphetamine test was not held as it is not available in our centre. After suspecting an acute HS, we carried out a head and neck non contrast Magnetic Resonance Imaging (MRI) to rule out carotid artery dissection. Results in T1 sequence showed a hyperintense left internal carotid artery at its entrance in the base of the skull and a narrowing of its lumen (Figures 1 & 2), being diagnosed of a carotid artery dissection and intramural hematoma due to a fish bone foreign body trauma.

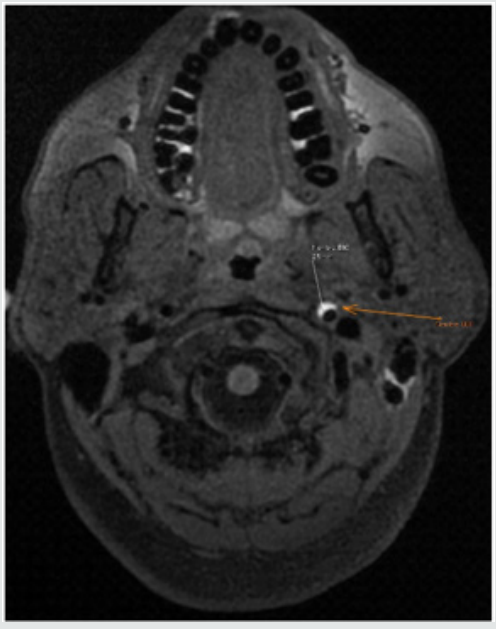

Figure 1: Non contrast MRI axial plane of supra-aortic arteries in T1 sequence proving an intramural hematoma. Notice the half-moon shape of the hematoma with a size of 2'5mm in the axial plane.

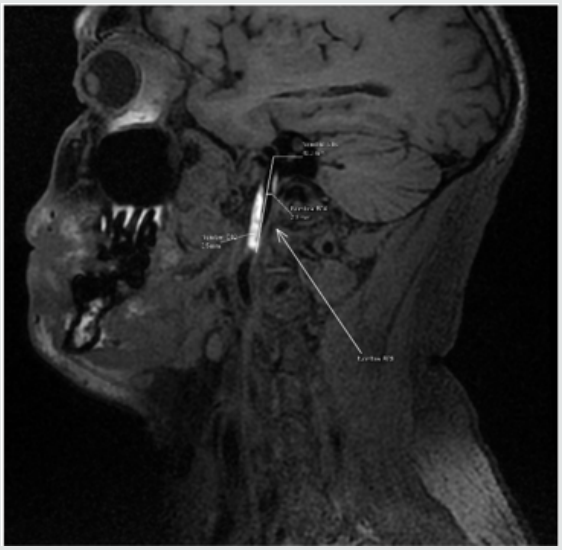

Figure 2: Non contrast MRI sagittal plane of supra-aortic arteries in T1 sequence. Notice the intramural hematoma with a size of 2'5mm in the axial plane and 4cm in the craniocaudal axis.

Discussion

HS may be caused by damage at different parts of the oculosympathetic pathway. The first order (central) neuron is localised in the posterolateral hypothalamus. Fibres descend lateral to the spinal bulb to the first synapse in the spine cord C8-T2, also called ciliospinal center of Budge and Waller. Damage at any of these structures may cause a central ipsilateral HS and can associate hemiparesis and contralateral hypoesthesia if the lesion is localised at the hypothalamus [1]. A lateral spine bulb infarction may cause ipsilateral HS and ataxia, and contralateral loss of pain. This triad is also known as Wallenberg syndrome [2]. Preganglionic HS is caused due to a lesion of the second order neuron, which originates from the ciliospinal center of Budge and Waller and travels over the pulmonary apex, ascending towards the stellate ganglion and carotid artery, and finally synapses in the superior cervical ganglion located near the common carotid bifurcation and the angle of the mandible [1]. In a series of cases, malignant tumours proved to cause up to 25% of preganglionic HS. The most common tumours were lung and breast [3].

The third-order (postganglionic) neuron originates from the superior cervical ganglion, travelling within the adventitia of the internal carotid artery into the cavernous sinus, where the fibres run next to the sixth cranial nerve (abducens) before joining the ophthalmic branch of the trigeminal nerve to enter the orbit together with the long ciliary nerve, which innervated the dilator muscles of the iris [4,5]. Damage of the internal carotid artery usually presents with stroke symptoms as hemiparesis, dysarthria, unilateral headache and cervical pain, as well as an incomplete HS, as anhidrosis is not present. This is explained as the damage affects the sympathetic fibres of the internal carotid artery, but not those that travel together with the external carotid artery, that will innervate the facial sweat glands [1,6]. Damage of the superior cervical ganglion may cause postganglionic HS, as it is located approximately 1.5cm behind the palatine tonsil and it may be injured by trauma or surgical procedures such as amygdalectomy [1]. Any damage that involves the cavernous sinus may cause HS as well as palsy of one or more oculomotor nerves [7-10]. In our case, damage is presumably located at the postganglionic nerve located within the internal carotid artery and at its proximity to the palatine tonsil. The absence of anhidrosis matches the site of the damage, as the fibres that accompany the external carotid artery are intact [1].

Conclusion

HS may have various aetiologies, many of them life threatening. Therefore, awareness of the oculosympathetic pathway anatomy and a meticulous examination are strictly recommended to attain a correct diagnosis and localisation of the damage.

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