A Case of Isolated Pre-Ganglionic Horner Syndrome Secondary to Lipoma of Carotid Space

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INTRODUCTION

Horner Syndrome (HS) is a condition resulting from damage along the oculo-sympathetic pathway. This pathway provides sympathetic innervation to the eye through three orders of neurons located 1) central in the hypothalamus, 2) pre-ganglionic in the thoracic spinal cord, and 3) post-ganglionic in the superior cervical ganglion. This disruption of sympathetic innervation results in a classic triad of ipsilateral ptosis, miosis, and anhidrosis. This case reviews the clinical presentation of an isolated pre-ganglionic HS owing to a lipoma of the carotid space.

CASE PRESENTATION

A 56-year-old African American male presents to the primary eye care clinic for a routine comprehensive eye examination. Past ocular history was unremarkable, and past medical history was significant for hypertension in which he was being treated with hydrochlorothiazide. Clinical examination revealed notable right upper eyelid ptosis, miosis, anhidrosis, and dilation lag.

TREATMENT AND MANAGEMENT

Magnetic resonance imaging (MRI) was obtained of his head, neck, and upper chest to determine the etiology of his HS. Imaging revealed a diagnosis of a 14 x 6 x 9 mm lipoma of the carotid space. Based on the location of the lesion and HS being the only sequelae in this asymptomatic patient, no treatment was indicated. The condition will continue to be monitored regularly, including re-evaluation of lacrimation, and initiating a dry eye treatment regimen if indicated.

FIGURE 1: Examination Pertinent Findings

OD		OS
20/20-2	VA(sc)	20/20-3
3mm (bright), 4mm (dim) (+)dilation lag RRL, no RAPD	Pupils	4mm (bright), 6mm (dim) RRL, no RAPD
FTFC	EOMs	FTFC
FROM	CVF	FROM
upper eyelid ptosis	Adnexa	unremarkable

FIGURE 2: Pharmacological Testing with 0.5% Apraclonidine

OD		OS
BEFORE 0.5% APRACLONIDINE		
MRD1 = 2 mm MRD2 = 8 mm	MRD	MRD1 = 4 mm MRD2 = 8mm
3 mm (bright) 4 mm (dim)	Pupils	4 mm (bright) 6 mm (dim)
45 MINUTES AFTER 0.5% APRACLONIDINE OD		
MRD1 = 4 mm MRD2 = 7mm	MRD	MRD1 = 4 mm MRD2 = 8mm
2.5 mm (bright) 5.5 mm (dim)	Pupils	3.5 mm (bright) 6.5 mm (dim)

FIGURE 3: Pupil Appearance Before and After 0.5% Apraclonidine Instilled OU

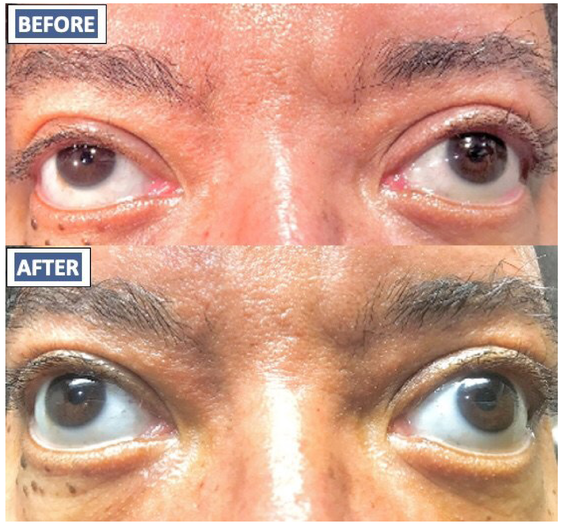
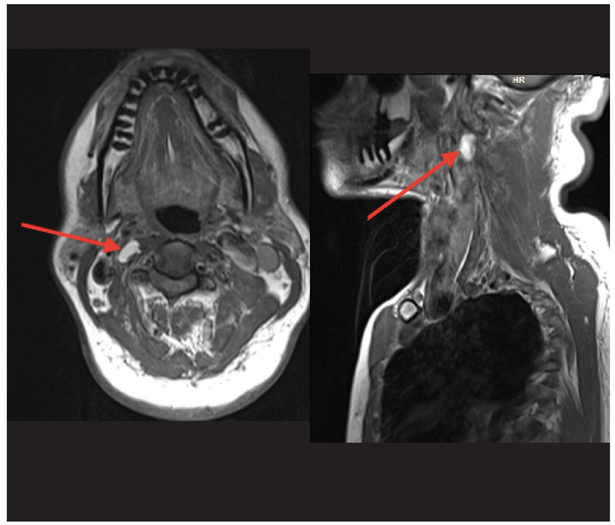


FIGURE 4: MRI Indicating Lipoma in Carotid Space



DISCUSSION

Causes of acquired HS include trauma, tumor, stroke, carotid artery anomalies, surgery in the region of the oculo-sympathetic pathway, or idiopathic occurrence to name a few. Findings consist of ipsilateral upper eyelid ptosis due to paresis of Muller's muscle, ipsilateral facial anhidrosis, and anisocoria greater in dim due to paresis of the iris dilator. Iris dilator paresis also causes impaired movement during dilation, a finding called dilation lag. HS may also be congenital where iris hypochromia may be a clinical feature. Pharmacological testing can aid in diagnosis and localization of HS. Use of apraclonidine 0.5-1% will not dilate a normal pupil but will dilate a HS pupil regardless of first, second, or third order neuron disruption. Upon instillation of apraclonidine, reversal of anisocoria is observed where the affected pupil dilates due to the up-regulation of alpha-1 receptors in HS. This elicits an exaggerated response of the iris dilators due to denervation supersensitivity to this agonist agent.

CONCLUSION

There are several etiologies resulting in Horner Syndrome, some of which are life-threatening. Pharmacological testing along with imaging of the oculo-sympathetic pathway are essential to determine the location of a potential lesion, which thereby directs further treatment and management. This case highlights the importance of Horner Syndrome identification, which was valuable in leading to the diagnosis of a tumor in the carotid space.

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