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EC NEUROLOGY
Case Report

Isolated Horner Syndrome as a Sole Manifestation of Ischemic Stroke

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Abstract

A middle aged female presented with acute onset right sided ocular symptoms with coryza. Clinical findings were suggestive of a right sided Horner's syndrome without significant motor, sensory or cranial nerve deficits. Closer scrutiny revealed a right sided patchy sweating abnormality. Pharmacological testing with apraclonidine confirmed the suspicion of Horner's syndrome. Suspecting a central lesion, an MRI brain was performed which revealed a localised and well circumscribed hypothalamic infarction. The patient was managed conservatively and did well on follow up.

Keywords: *Neuroimaging; Neurology; Neuroophthalmology; Stroke*

Introduction

Horner syndrome is not a very common clinical finding. Elucidation of the causal lesion is sometimes difficult and recovery and prognosis depends upon the etiology. Most of the causes of central Horner syndrome are usually manifestations of brainstem pathology and the two case reports found in the literature due to hypothalamic involvement had accompanying motor manifestations (facial weakness, dysarthria and brachial weakness) leading to suspicion of a central pathology. We present this case as it represents a very rare manifestation of hypothalamic involvement (infarction) which manifested as isolated Horner syndrome as the only neurologic manifestation leading to a diagnostic challenge. This possibly would be the first report of such kind.

Case Presentation

A 47 year old female, known hypertensive, presented to the neurology services with a frontal headache of 3 days duration associated with coryza. She also complained of vague discomfort in her right eye without any dimness of vision which she had developed the day prior to her presentation. Clinical examination revealed a right sided partial ptosis with anisocoria; the right pupil was constricted in comparison to left. The lower eyelid was also elevated in comparison to right (Figure 1). There were no other significant ocular findings including ophthalmoscopy. Ocular movements were full and painless. No limb weakness, dysarthria or symptoms or signs pertaining to lower cranial nerves were elicited. Her NIHSS score was 0. There were no systemic features, cardiac auscultation was normal. No carotid bruit was present. However, on closer examination there were patchy areas of hypohidrosis involving the right upper and lower face, right arm and trunk. Blood pressure was 140/90 mmHg without any postural drop and equal in both limbs.

Figure 1: *Right sided ptosis.*

Investigations

Suspecting a central Horner syndrome pharmacological testing was performed with apraclonidine 1% drops which were instilled separately in both eyes to observe for response. While there was no effect in the left eye, the right eye dilated after instillation of apraclonidine. An MRI brain was next performed to look for a suspected central cause of the Horner's syndrome. A well delineated area with Diffusion restriction with corresponding FLAIR hyperintensity was apparent in the right hypothalamic area (Figure 2 and 3) without any other significant findings. MRA using time of flight sequences were normal and trans cranial Doppler did not reveal any high intensity transient signals. A holter study done for 24 hours was also normal. Blood biochemical examination revealed elevated low density lipoprotein levels. Blood sugar levels, homocysteine levels and collagen vascular screen were normal.

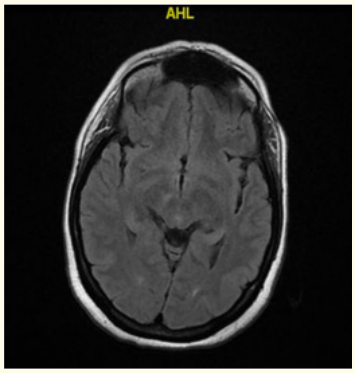

Figure 2: *T2 Flair Hyperintensity.*

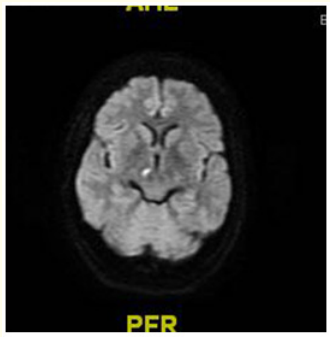

Figure 3: *DWI showing restriction.*

Treatment

The patient was started on aspirin 150 mg once daily, atorvastatin 40 mg once daily at bed time and hydrochlorothiazide 12.5 mg once daily.

Outcome and follow-up

The patient did well. Her modified Rankin score was 0 and did not have recurrence of any other neurological events. During her first follow up she continued to do well with subtle eye signs persisting.

Discussion

Horner syndrome is a constellation of clinical findings comprising of anisocoria, partial ptosis and anhidrosis. The causal lesion can be localised to any part of the 3 tier sympathetic chain starting with the first order neuron from the posterolateral hypothalamus, travelling

through the brainstem and ending in the cilio-spinal centre of Budge (C8-T2). The second order pre-ganglionic neurons exit at the level of T1 and synapse in the superior cervical ganglion (C3-C4). Bifurcation of the pupillo-motor fibres and sudomotor fibres occurs soon after and while the former travels along the internal carotid artery the latter accompanies the external carotid artery. The pupillo-motor fibres travel across the cavernous sinus and travelling through the superior orbital fissure via the long ciliary nerves supply the Mullers muscle and iris dilators [1]. The patient we describe had anhidrosis which affected the upper part of her chest and upper limb in addition to her face, a feature which helps distinguish a central from a peripheral lesion. However, central lesions most often have accompanying motor or other features like dysarthria, dysphagia, ataxia and nystagmus which help us to localise the lesion to the brainstem. Other clues to a central lesionmight be headache or obtundation which may be reflection of raised intracranial pressure. Horner's syndrome due to hypothalamic infarct has been sparsely reported. Stone., *et al.* in 1986 [2] reported on a middle aged male an association of hypothalamic infarction and Horner's syndrome. Another report by Austin., *et al.* in 1991 [3] highlighted a similar association. A further association was reported by Gieraerts., *et al.* in 2015 [4]. However, in all such cases extra ocular accompaniments (motor manifestations in the form of facio-brachial weakness, language dysfunction in the form of aphasia or paraphasia and brainstem feature like ataxia) was present thus pointing towards a possible central lesion. However, an isolated Horner syndrome with acute confusional state was reported by Sahin., *et al.* very recently (2018) where the lesion was due to a hypothalamic bleed [5]. Pharmacological testing in our patient and the radiological finding of selective hypothalamic involvement on MRI clearly establishes a cause and effect relationship. This case is unique because of the isolated ocular findings in the absence of motor, language or brainstem features. However, the distribution of hypohidrosis beyond the ipsilateral facial distribution was an important clue for a central location of the lesion.

Conclusion

Central Horner syndrome should be suspected if the area of hypohidrosis spreads beyond the affected facial distribution. Isolated Horner syndrome in the absence of other localising features may be a rare presentation of hypothalamic involvement. Neuroimaging, usually diffusion and susceptibility weighted magnetic resonance imaging are required to differentiate among the various casual lesions. Prognosis in such cases depends upon the causal lesions with isolated syndromes having better outcomes.

Source of Support

Nil.

Conflict of Interest

None declared.

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