The Yield of Diagnostic Imaging in Patients with Isolated Horner Syndrome

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KEYWORDS

• Isolated Horner syndrome • Anisocoria • Imaging yield • Carotid artery dissection

KEY POINTS

- Patients with Horner syndrome (oculosympathetic defect) may be associated with signs and symptoms that help determine the cause and localization of the defect.
- Isolated Horner syndrome is defined as an oculosympathetic defect *without* associated signs and symptoms (except for pain) and it presents a diagnostic dilemma in which neuroimaging is indicated.
- Imaging of isolated Horner syndrome in a consecutive case series yielded a structural cause in 20% of patients with the most common cause being a carotid dissection.

INTRODUCTION

Horner syndrome is a clinical constellation of symptoms and signs classically including ipsilateral ptosis, pupillary miosis, and facial anhidrosis due to a lesion of the oculosympathetic pathway. The 3-neuron length of the oculosympathetic pathway produces a diagnostic challenge for the clinician with a patient who presents with a clinically isolated Horner syndrome.[1]

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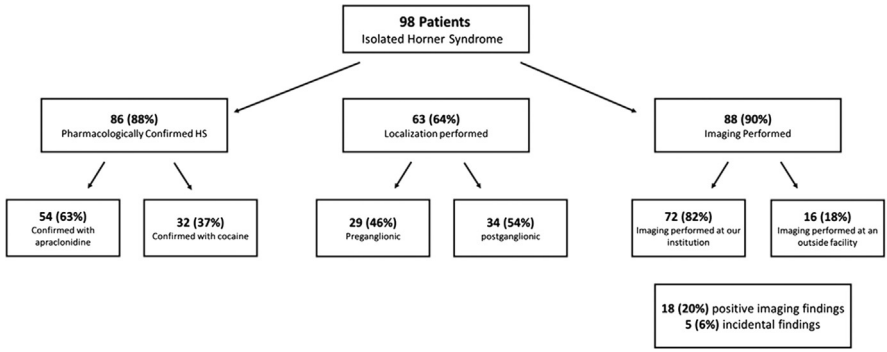

Fig. 1. The distribution of the testing, localization, and imaging yield of the 98 patients with isolated Horner syndrome (HS).

Determining the cause of Horner syndrome is justified, because a sinister underlying condition could be present, in which the patient would benefit from early diagnosis. Topical pharmacologic testing with apraclonidine or cocaine can be used to diagnose Horner syndrome or it may be determined clinically.[2] Hydroxyamphetamine pharmacologic testing can be used to localize the oculosympathetic lesion as being preganglionic (first-order or second-order neuron lesion) or postganglionic (third-order neuron lesion),[3] but it now has limited availability. Some clinicians use pharmacologic testing to help focus the interpretation of subsequent imaging.[4] There are a number of studies that advocate for a systematic approach to localization of the lesion using associated signs and symptoms, and then performing subsequent anatomically focused imaging with either MRI or computed tomography (CT) with angiography. For example, Reede and colleagues[2] advocate for identifying whether the Horner syndrome is a first-order, second-order, or third-order neuron lesion and performing focused imaging with either CT or MRI. Digre and colleagues[5] separated patients based on preganglionic and postganglionic lesions with pharmacologic testing or clinical localization, and imaged this select region. Davagnanam and colleagues[4] developed an imaging algorithm separating patients with first-order neuron lesions from those with second-order and third-order neuron lesions. In this algorithm, first-order neuron lesions were imaged with MRI, including the brain, cervical spinal cord, and upper thoracic spinal cord. Second-order and third-order neuron lesions were imaged with CT angiography from the orbits to T4 to T5. Davagnanam and colleagues[4] touched on the challenges of approaching patients with clinically isolated Horner syndrome, suggesting that these patients would be best evaluated within 6 weeks. However, they do not recommend specific imaging modalities, but rather that the study type be chosen at the clinician's discretion. Of note, the investigators defined isolated Horner syndrome to include patients who lack localizing signs, but also did not have a personal history of malignancy or pain.

Our study was motivated by the clinical conundrum presented by isolated Horner syndrome, as these patients do not lend themselves to decision trees of traditional neurologic localization and have a wide variety of potentially sinister causes. We defined a patient to have an isolated Horner syndrome if the patient presented without other clinical signs to aid in localization following a thorough history and physical examination. For this study, patients with pain or headache were not excluded and could be considered to have isolated Horner syndrome. Ultimately, we sought to determine