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ORIGINAL ARTICLE

Etiological evaluation of Horner syndrome in the modern imaging era and proposal for a practical algorithm

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ABSTRACT

Objectives: This study aimed to investigate the etiology in patients with Horner syndrome (HS) using neuroimaging studies based on demographic and clinical characteristics and to contribute to clinical practice by developing a practical algorithm based on our findings and integrating current neuroimaging techniques.

Patients and methods: This retrospective, two-center study evaluated 54 patients (28 males, 26 females; mean age: 49.3 ± 14.1 years; range, 21 to 76 years) with HS between January 2013 and February 2022. Demographic and clinical features of the patients, the response to apraclonidine, and neuroimaging study findings were evaluated.

Results: Of the cases included in the study, 18 (33.3%) were idiopathic, three (5.5%) were congenital, 13 (24.1%) were central (first-order neuron), 11 (20.4%) were preganglionic (second-order neuron), and nine (16.7%) were postganglionic (third-order neuron). Localized etiology was affirmed in 33 (61.8%) of 54 patients.

Conclusion: This study showed that neuroimaging methods easily identified etiology in patients with additional neurological findings in HS. The underlying causes could not be defined in a significant part of patients with isolated HS. However, it is essential to carry out detailed neuroimaging studies according to clinical findings to exclude life-threatening causes in patients with HS.

Keywords: Horner syndrome, miosis, oculosympathetic syndrome, ptosis, pupil.

Horner syndrome (HS) is a clinical syndrome characterized by miosis, ptosis, and, in some cases, facial anhidrosis. It results from damage to the oculosympathetic pathway.[1,2] Although data on its incidence are limited, HS is known to be more common in adults. In a recent national study from Korea, the cumulative incidence was reported as 2.12 per 100,000 in the pediatric population and 2.93 per 100,000 in adults. The highest incidence was observed in children aged 0–4 years and in adults aged 50–54 years.[3]

The oculosympathetic pathway is a long and complex three-neuron system extending from the hypothalamus to the eye. First-order neurons originate in the hypothalamus and descend to the C8-T2 levels of the spinal cord. Damage at this level results in central HS. Second-order neurons exit the spinal cord and reach the superior cervical ganglion via the brachial plexus. Third-order neurons then pass through the adventitia of the internal carotid artery (ICA) and the cavernous sinus before terminating in the iris dilator muscle.[4,5] Oculosympathetic damage may occur at any point along this pathway and can result from a wide range of pathologies. While some causes are benign, others are life-threatening and require urgent

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diagnosis and treatment.[6] Therefore, prompt and detailed clinical evaluation is essential in patients with HS. In addition to neurological examination, pharmacological testing should be performed. Topical application of 2–10% cocaine or 0.5–1% apraclonidine is commonly used to differentiate HS from other causes of miosis.[2,7] In recent years, apraclonidine has been used more frequently than cocaine for this purpose. Apraclonidine is an adrenergic agonist that is primarily used in the treatment of glaucoma.[8,9] Under normal conditions, it predominantly stimulates alpha-2 receptors and causes pupillary constriction by inhibiting noradrenaline release from presynaptic sympathetic nerve endings. In HS, sympathetic denervation leads to upregulation of postsynaptic alpha-1 receptors in the iris dilator muscle. As a result, the weaker alpha-1 agonist effect of apraclonidine becomes dominant. This causes pupil dilatation and reversal of anisocoria.[10] Once the diagnosis of HS is confirmed, further investigations should be systematically planned. These investigations aim to localize lesions along the oculosympathetic pathway and to identify clinically significant underlying conditions.[11]

The primary objective of this study was to determine the etiology of HS in our patient population using neuroimaging methods. Demographic and clinical factors were also taken into account. In addition, we aimed to develop a straightforward diagnostic algorithm based on our experience and current neuroimaging techniques.

PATIENTS AND METHODS

In our study, the medical records of 54 patients (28 males, 26 females; mean age: 49.3 ± 14.1 years; range, 21 to 76 years) diagnosed with HS at the Neurology Clinics of Gazi and Hacettepe University between January 2013 and February 2022 were retrospectively reviewed. Past medical history, demographic details, and clinical characteristics of the patients, including the underlying etiology and any accompanying neurological features, were also evaluated. We also recorded whether there was a response to 0.5% apraclonidine for HS confirmation. Detailed neurological and neuroophthalmological examinations were noted. Neuroimaging data of the patients were collected, and brain computed tomography (CT), brain and neck magnetic resonance imaging (MRI), vascular imaging with computed tomography angiography (CTA), magnetic resonance angiography (MRA), or digital subtraction angiography (DSA), as well as chest radiograph and CT results, were recorded. Written informed consent was obtained from all participants. The study protocol was approved by the Gazi University Clinical Research Ethics Committee (Date: 21.03.2022, No: 215). The study was conducted in accordance with the principles of the Declaration of Helsinki.

Statistical analysis

Statistical evaluation of the data was performed using IBM SPSS version 20.0 software (IBM Corp., Armonk, NY, USA). In the analysis of the variables, their conformity to the normal distribution was examined by visual (histogram, probability graphs) and analytical methods (Kolmogorov-Smirnov test). The main differences between more than two groups were analyzed with the one-way analysis of variance. Descriptive statistics were presented as numbers and percentages for categorical variables, mean ± standard deviation (SD) and minimum-maximum values for numerical variables. Results were considered statistically significant when the p-value was <0.05.

RESULTS

Patients included in the study were classified into four etiological groups: idiopathic/congenital, central, preganglionic, and postganglionic HS. Of the total cohort, 18 (33.3%) patients were idiopathic, three (5.5%) congenital, 13 (24.1%) central, 11 (20.4%) preganglionic, and nine (16.7%) postganglionic. Ptosis and miosis were present in all patients included in the study. However, assessment of anhidrosis was incomplete due to missing or insufficient documentation in the available clinical records.

Apraclonidine testing could be performed in 44 (81.5%) of 54 patients. Due to temporary unavailability of apraclonidine in our country, testing could not be conducted in 10 patients, including one idiopathic, five central, two preganglionic, and two postganglionic HS cases. All patients who underwent apraclonidine testing demonstrated a positive response, consistent with the established diagnosis of HS. Cocaine and hydroxyamphetamine tests could not be performed because these agents are controlled substances in Türkiye. In Table 1, the classification according to localization, demographic, and clinical characteristics of 54 patients who were followed up with the diagnosis of HS are summarized.

Isolated HS was observed in 30 (55.6%) patients, whereas 24 (44.4%) patients presented

TABLE 1
Demographic and clinical characteristics of 54 patients with HS

	n	%	Mean ± SD
Age at diagnosis			
Idiopathic/congenital HS			46.2 ± 18
Central (first-order neuron) HS			50.9 ± 10.1
Preganglionic (second-order neuron) HS			54.7 ± 10.9
Postganglionic (third-order neuron) HS			47.4 ± 12
Total patients			49.3 ± 14.1
Sex			
Female	26	48.1	
Male	28	51.9	
Apraclonidine (+)	44	81.5	
Unknown	10	18.5	
Isolated HS	30	55.6	
Idiopathic/congenital	21	100	
Central HS	0	0	
Preganglionic HS	5	45.5	
Postganglionic HS	4	44.4	
HS side			
Right	31	57.4	
Left	23	42.6	

HS: Horner syndrome; SD, standard deviation.

with additional neurological or systemic symptoms. Among patients with isolated HS, an underlying etiology was identified in 30%, while 70% were classified within the idiopathic/congenital group. A statistically significant difference was observed in the distribution of isolated HS across the four etiological groups ($p < 0.001$; Figure 1).

Pairwise comparisons revealed a significant difference between the idiopathic/congenital group and both the central and preganglionic HS groups ($p < 0.001$). All patients in the idiopathic/congenital group presented with isolated HS, whereas none of the patients with central HS had isolated presentations. A significant difference was also observed between central and preganglionic HS ($p = 0.011$); while all first-order neuron lesions were associated with non-isolated HS, 54.5% of second-order neuron lesions were non-isolated. In addition, a significant difference was found between central and postganglionic HS ($p = 0.017$). No statistically

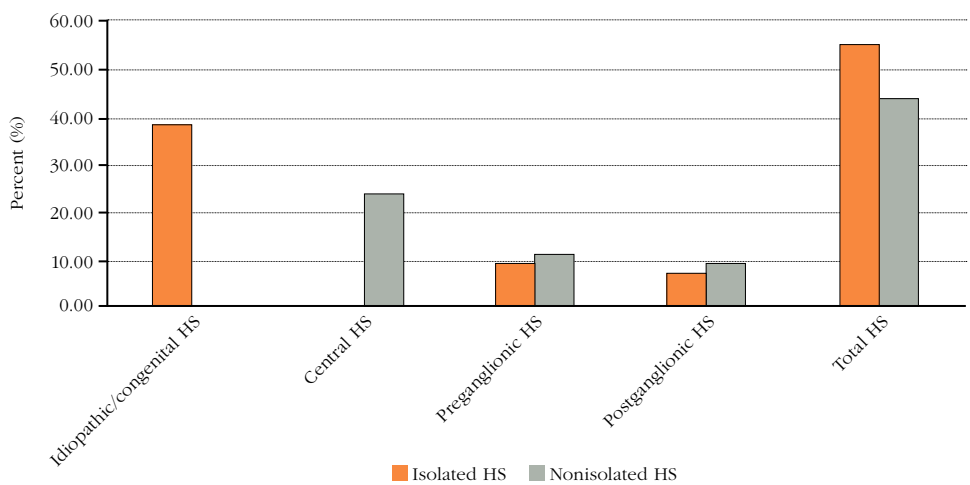

Figure 1. Comparison of etiological causes in isolated and non-isolated HS.
HS: Horner syndrome.

significant differences were detected in the remaining pairwise comparisons.

In the idiopathic/congenital group, no symptoms or abnormal neurological examination findings were observed other than ptosis, miosis, occasional anhidrosis, and iris heterochromia in one patient.

In the central HS group, the etiology was cerebrovascular in 10 (76.9%) patients, including lateral medullary syndrome in eight patients. The remaining cases were attributed to thalamic infarction (n = 1), mesencephalic infarction (n = 1), mesencephalic cavernous hemorrhage (n = 1), spinal cord ependymoma (n = 1), and syringomyelia (n = 1). All patients in the central HS group presented with non-isolated HS. Accompanying neurological findings included hemiparesis (n = 8), facial or hemisensory hypoesthesia (n = 7), central facial palsy (n = 3), dysphagia (n = 2), and ataxia (n = 1). Brain MRI was performed in all patients. Brain and neck CTA or MRA was obtained in 11 (84.6%) patients, and DSA in three (23.1%) patients. Chest radiographs were requested in 11 (84.6%) patients, while cervical MRI was diagnostic in two (15.4%) patients with spinal involvement. Imaging findings were fully concordant with clinical localization in this group.

The preganglionic HS group included 11 patients. The most common etiologies were post-surgical or procedure-related causes in six (54.5%) patients, including thoracic outlet surgery, central venous catheterization, post-sternotomy, thyroidectomy, and other postoperative causes. Malignancy-related HS was identified in two (27.3%) patients, including one patient with a Pancoast tumor and one with breast cancer-associated cervical lymphadenopathy. In addition, one patient presented with isolated cervical lymphadenopathy of unknown primary origin. Other causes included Eagle syndrome (n = 1) and shoulder sling use following trauma (n = 1). Non-isolated HS was observed in six (54.5%) patients, presenting with hypoesthesia (n = 3), dysphagia (n = 2), and upper limb motor weakness (n = 1). Brain MRI was performed in all patients. Computed tomography angiography or MRA was obtained in five (45.5%) patients, chest CT in six (54.5%) patients, cervical MRI in two (18.2%) patients, and positron emission tomography in one (9.1%) patient. Imaging strategies were tailored according to clinical presentation and suspected etiology.

TABLE 2
The underlying causes of HS according to localization

Etiology	n	%
Central HS		
Lateral medullary infarction	8	24.2
Other Ischemic stroke	2	6.1
Cerebral hemorrhage	1	3
Syringomyelia	1	3
Spinal mass	1	3
Total	13	39
Preganglionic HS		
Lung cancer	1	3
Neck masses	2	6.1
Post-surgical and interventional procedure	7	21.2
Eagle syndrome	1	3
Total	11	33
Postganglionic HS		
Carotid dissection/thrombosis	4	12.1
Cavernous sinus involvement	1	3
Cluster headache	3	9.1
Cervical schwannoma surgery	1	3
Total	9	27.3

HS: Horner syndrome.

The postganglionic HS group consisted of nine patients. Internal carotid artery pathology was identified in four (44.4%) patients, including ICA dissection in three patients and ICA occlusion in one patient. All patients with ICA dissection presented with neck pain and focal neurological deficits, most commonly hemiparesis and hemisensory loss. In all patients in whom arterial dissection was clinically suspected, brain and neck MRA was performed.

Other causes included cervical schwannoma surgery (n = 1) and cavernous sinus involvement secondary to nasopharyngeal carcinoma (n = 1), accompanied by sixth cranial nerve palsy. Three (33.3%) patients developed HS during cluster headache attacks; in two patients, HS resolved completely during attack-free periods. Brain MRI was performed in all patients, MRA in eight (88.9%) patients, and DSA in two (22.2%) patients. Chest radiographs were requested in eight (88.9%) patients. In cases with confirmed ICA dissection, additional chest CT was not routinely performed. The underlying causes of HS in patients are detailed in Table 2.

DISCUSSION

In this retrospective, two-center study, despite the available advanced imaging methods, no etiological lesion could be demonstrated in 21 (38%) of 54 patients with HS and were classified as idiopathic HS. Among patients with an identified cause, central HS was the most frequent

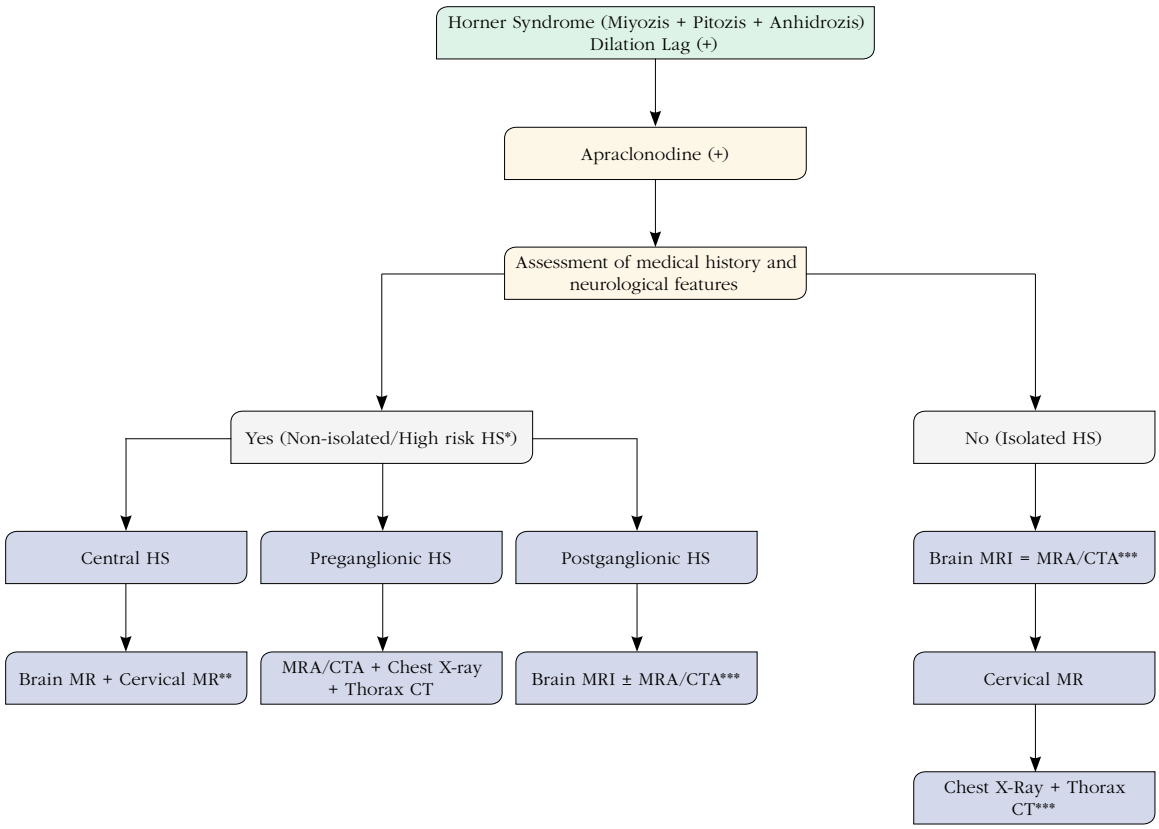

Figure 2. A diagnostic algorithm in HS.

HS, Horner syndro me; MRI, magnetic resonance imaging; MRA, magnetic resonance angiography; CTA, computed tomography angiography; MR, magnetic resonance; *, high-risk features include (Focal neurological deficits, acute onset pain or trauma, history of malignancy, vascular risk factors, progressive or persistent symptoms); **, if a space-occupying lesion is suspected in Chest X-ray, thorax CT should be performed; ***, vascular imaging, preferably MRA, could be performed in patients presenting with acute HS to rule out carotid artery dissection. When MRA is contraindicated or unavailable, CTA would be appropriate.

anatomical localization, and all patients in this group presented with additional neurological signs and symptoms. These findings highlight the strong association between lesion localization and clinical presentation in HS.

Consistent with previous reports, the complete classical triad of ptosis, miosis, and anhidrosis was rarely observed. In clinical practice, the manifestations of HS are often subtle, and anhidrosis may be overlooked. Therefore, the presence of accompanying neurological deficits plays a crucial role in lesion localization and guides the choice of neuroimaging. In contrast, in patients presenting with isolated HS, localization based on clinical findings alone is frequently not possible, making imaging essential for identifying the underlying cause.

In a population-based study of 1,470 patients from South Korea, Han et al.[3] reported that approximately 43% of HS cases were idiopathic, a finding comparable to our results. However, in the study by Han et al.,[3] HS developing after surgical procedures was the most common cause among patients with an identified etiology, accounting for 58% of cases. In contrast, only four (7%) patients in our cohort were diagnosed with iatrogenic HS. The relatively high proportion of idiopathic HS in our study may be explained by the fact that the participating hospitals are tertiary referral centers, where patients with unresolved diagnostic problems are more frequently admitted.

In contrast to our findings, Sabbagh et al.[12] reported that the most common cause of pharmacologically confirmed HS in their cohort of 159 patients was postprocedural events, including surgery and central venous catheterization, accounting for 21% (n = 34) of cases, followed by carotid artery dissection in 9% (n = 14) and trauma in 8% (n = 13). In a study of 52 patients that included both outpatients and inpatients, Almog et al.[6] found that among patients with a known etiology, preganglionic causes (44%) were

the most prevalent, followed by central (28%) and postganglionic (28%) causes, which occurred at similar frequencies.

In our cohort, hospitalized patients frequently presented with additional neurological symptoms, and stroke was the most common etiology. Among stroke-related cases, lateral medullary syndrome was the most frequent underlying cause. Similarly, in the study conducted by Top et al.,[13] stroke was reported as the most common etiology of HS. In addition, the incidence of first- and second-order neuron involvement was comparable, whereas postganglionic causes were relatively infrequent; these findings are consistent with our results. The high prevalence of stroke-related HS in our study may be attributed to the presence of specialized stroke units within the participating centers.

Malignancy-related HS was identified in four patients (7% of the total cohort). In the preganglionic group, two patients had malignancy-associated HS, one due to recurrent breast cancer presenting with cervical lymphadenopathy and the other secondary to lung cancer. In the postganglionic group, HS occurred as a result of cavernous sinus involvement from nasopharyngeal carcinoma, while in the central group, HS was associated with a spinal cord ependymoma. In a large retrospective study by Maloney et al.[14] involving 450 patients with HS, an underlying etiology was identified in 270 (60%) patients. Among those with a determinable cause, 13% were attributed to central lesions, 44% to preganglionic lesions, and 43% to postganglionic lesions. Sabbagh et al.[12] reported that HS occurring after surgical procedures was reported as the most common etiology in several studies. In contrast, iatrogenic HS accounted for only 7% (n = 4) of cases in our cohort, a finding consistent with the results reported by Top et al.[13] In our cohort, clinically significant conditions such as malignancy or metastatic recurrence, carotid artery dissection, and Eagle syndrome were identified in a subset of patients presenting with isolated HS. These findings underscore the importance of maintaining a high index of suspicion, particularly in patients with a history of malignancy, acute pain, or vascular risk factors. While a substantial proportion of isolated HS cases remained idiopathic, selective and targeted imaging was instrumental in detecting severe underlying pathology in others.

Although apraclonidine test negativity has been described in the literature, apraclonidine testing was performed in 44 (81%) patients in our study, and all tested patients demonstrated positive responses.[9,13]

Neuroimaging plays a central role in the diagnostic evaluation of HS, and different modalities provide complementary information.[15] Brain MRI is the most informative initial imaging technique in patients with suspected central involvement, particularly when additional neurological signs are present.[16] In patients with acute onset painful HS or a history of trauma, urgent imaging of the brain and neck using CTA or MRA is essential to exclude craniocervical arterial dissection. Computed tomography angiography may be preferred in the acute setting due to its rapid acquisition time. In patients without symptoms suggestive of brain or brainstem involvement who present with neck pain, dermatomal sensory deficits, or weakness, and in whom cranial MRI is unremarkable, cervical and upper thoracic spinal cord MRI should be performed.[17,18]

Neuroimaging was performed in a localization-guided and clinically tailored manner across the study cohort. Brain MRI constituted the cornerstone of imaging evaluation and was performed in all patients with idiopathic/congenital (n = 21), central (n = 13), and postganglionic (n = 9) HS, and in 10 (90.9%) of 11 patients with preganglionic HS.

Overall, vascular imaging of the brain and neck using MRA or CTA was performed in 49 (90.7%) of 54 patients. Magnetic resonance imaging was the most frequently used modality, obtained in 36 (66.7%) patients, whereas CTA was performed in 13 (24.1%) patients. Some patients underwent both modalities. Importantly, brain and neck MRA was performed in all patients with clinical suspicion of arterial dissection, and this approach was diagnostic in patients with confirmed ICA dissection. Computed tomography angiography was not used as the initial imaging modality in patients with suspected dissection in this cohort.

Digital subtraction angiography was reserved for selected cases and was performed for diagnostic confirmation and therapeutic purposes, particularly in patients with suspected or confirmed vascular pathology. Chest imaging was frequently utilized to evaluate potential thoracic or malignant causes. Chest radiographs were obtained in the majority of patients across all subgroups, while chest CT was selectively performed in patients with suspected thoracic pathology or malignancy. Cervical MRI was used in selected patients presenting with neck pain or suspected cervical or spinal involvement

and was diagnostic in patients with spinal cord pathology. In the central (first-order neuron) HS group, imaging demonstrated a high diagnostic yield. Brain MRI, complemented by vascular imaging, identified cerebrovascular pathology in most patients, predominantly brainstem infarction related to lateral medullary syndrome, and imaging findings were fully concordant with clinical localization. In the preganglionic (second-order neuron) HS group, imaging strategies were individualized. Chest CT, cervical MRI, and vascular imaging contributed to the identification of procedure-related causes, malignancy-associated pathology, and cervical or thoracic lesions in selected cases. In the postganglionic (third-order neuron) HS group, vascular imaging yielded clinically significant findings, with ICA pathology identified in a substantial proportion of patients. In cases with confirmed ICA dissection, additional chest CT imaging was not routinely performed. In contrast, in the idiopathic/congenital HS group, despite comprehensive imaging including brain MRI, vascular imaging, and chest imaging in most patients, no causative lesion could be identified, resulting in a low diagnostic yield.

Overall, these findings indicate that imaging yields the highest diagnostic value in patients with additional neurological signs and suspected central or vascular involvement, while in isolated HS, selective and risk-based imaging is necessary to avoid missing potentially severe underlying conditions. This subgroup-dependent variation in diagnostic yield supports an individualized imaging strategy rather than a uniform approach for all patients with HS.

There remains no universal consensus regarding the optimal imaging strategy for patients with HS. Davagnanam et al.[16] proposed a diagnostic protocol based on neuronal localization of the lesion. In contrast, Chen et al.[19] suggested that a single imaging algorithm encompassing the entire oculosympathetic pathway, regardless of localization, may be more practical and easier to apply in clinical practice. Similarly, Sadaka et al.[20] recommended brain MRI for postganglionic HS and comprehensive neuroimaging for preganglionic HS, highlighting the need for tailored imaging approaches based on the suspected level of involvement (Figure 2).

Based on our findings, the presence of additional neurological signs in patients with HS facilitates lesion localization and increases the diagnostic yield of neuroimaging. In contrast, identifying the underlying cause of HS remains more challenging in patients presenting with isolated HS. In our cohort, isolated HS was observed in 30 (55.6%) patients, whereas 24 (44.4%) patients presented with accompanying neurological or systemic symptoms. Among patients with isolated HS, the underlying etiology could not be identified in 70%, and these cases were, therefore, classified as idiopathic.

Nevertheless, clinically significant conditions were identified in a subset of patients with isolated HS. Isolated presentations were observed in five patients with preganglionic HS and four patients with postganglionic HS. Notably, one patient with isolated HS had a prior history of breast cancer, and further diagnostic evaluation revealed tumor recurrence. In addition, three patients with postganglionic isolated HS presented during cluster headache attacks, in whom carotid artery dissection was excluded. Other isolated HS cases were associated with iatrogenic factors, including surgical procedures, catheterization, and shoulder sling use.

These findings indicate that although the diagnostic yield of imaging is lower in isolated HS compared with non-isolated presentations, targeted and risk-based imaging remains essential to avoid missing clinically important and potentially life-threatening conditions. Our results support a localization-based imaging approach, as previously proposed in the literature; however, they also demonstrate that rigid, uniform algorithms may not be appropriate for all patients. In isolated HS, particularly in younger individuals without clinical risk factors, extensive imaging may limit diagnostic yield, whereas insufficient imaging may result in missed diagnoses in patients with occult malignancy or vascular pathology. Therefore, imaging strategies should be individualized, balancing expected diagnostic yield against patient history, clinical risk factors, and symptom characteristics.

Several limitations of this study should be acknowledged. The retrospective design and relatively small sample size limit generalizability; however, given the rarity of HS, the inclusion of patients over a nine-year period from two tertiary referral centers provides meaningful clinical insight. In addition, pharmacological confirmation and localization using cocaine or hydroxyamphetamine testing could not be performed due to limited availability, and apraclonidine testing was not possible in all patients. Nevertheless, the clinical findings were typical of HS in all cases.

In conclusion, although the etiology of HS remained undetermined in a substantial proportion of patients, clinically significant underlying conditions were identified in a meaningful subset, particularly among those with isolated HS. Following clinical assessment and pharmacological confirmation, imaging should be incorporated into the diagnostic algorithm in a targeted and risk-based manner. Brain MRI, brain and neck CTA or MRA, and thoracic imaging should be selected according to clinical presentation to maximize diagnostic yield while avoiding unnecessary investigations.

Data Sharing Statement: The data that support the findings of this study are available from the corresponding author upon reasonable request.

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