Cervicogenic Headaches

A Literature Review and Proposed Multifaceted Approach to Diagnosis and Management

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KEYWORDS

• Cervicogenic headaches • Secondary headaches • Neck pain

KEY POINTS

- A standardized approach to diagnosing cervicogenic headaches can not only provide sustained relief for patients but also clarity for future research.
- Cervicogenic headaches can manifest in variable symptoms and overlap with other primary headaches, with or without neck pain.
- A confirmatory diagnosis of cervicogenic headaches requires certain clinical features, establishing a cervical origin, and identifyin.

INTRODUCTION

The relationship between headache and the cervical spine has been well described. However, the concept of cervicogenic headaches, or headaches that originate from disease or injury affecting the cervical spine, has only been accepted in the last 4 decades. Since then, there has been considerable controversy surrounding the diagnosis of cervicogenic headaches and around whether it is in fact a unique entity at all. This is largely due to its variable and wide range of clinical presentations and that the clinical symptoms tend to overlap with several primary and secondary headaches, as well as nonheadache neurologic diagnoses.

There are 3 general approaches to the diagnosis of cervicogenic headaches. The first aims to make the diagnosis based on clinical features, as is common practice with most primary headaches. The second is focused on establishing a cervical source for head pain. Finally, there is increasing support for the use of various treatment modalities

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aimed at the cervical spine resulting in headache alleviation as a confirmation of diagnosis. Here, we aim to provide a review of the current available literature on cervicogenic headaches including its epidemiology and pathophysiology and provide a holistic framework for its diagnosis and management.

BACKGROUND AND DEFINITION

In its broadest sense, cervicogenic headache is a condition that presents with head pain with or without accompanying neck pain secondary to dysfunction in the cervical spine, specifically with its component bony, disc and/or soft tissue elements. In its earliest form, it was described by French neurologist Barre in 1926 with the term "syndrome cervical sympathetique posterieur," which linked headache with a supposed posterior sympathetic deficiency.[1] The term cervicogenic headaches was finally coined in 1983 by Sjaastad after he presented a case series of 22 patients with a uniform clinical presentation of headache in the setting of evidence suggesting a cervical etiology.[2] In 1988, the International Headache Society amended its diagnostic classification system to include a category for headaches associated with disorders of the neck. Finally, after various iterations, clinical diagnostic criteria were published in 1998 by the Cervicogenic Headache International Study Group (CHISG) and remain the most current clinical criteria for the entity.[3]

EPIDEMIOLOGY

There is considerable variability in the reporting of the prevalence of cervicogenic headaches. The reason for this is that prevalence studies have been undertaken on heterogenous patient populations, the study methodologies have been varied, and several different diagnostic criteria have been used. In the general population, cervicogenic headache has an estimated prevalence of between 0.4% and 2.5%.[4] Cervicogenic headaches seem more common among women. The average age of presentation is 42.9 years with an average duration of symptoms lasting 6.8 years.[5,6]

PATHOPHYSIOLOGY

The most common source implicated in cervicogenic headache is upper cervical joint dysfunction. It is thought that pain in cervicogenic headache results from the interaction of nociceptive afferents of the descending trigeminal nerve (trigeminal nucleus caudalis) and the C1 to C3 segments of the cervical spine. Accordingly, pain signals can travel bidirectionally between the neck and regions of the face and head that receive innervation from the trigeminal nerve (**Fig. 1**).[7] The best studied and most implicated joint in cervicogenic headache is the C2–3 zygapophyseal joint. Stimulation of C2–C3 intervertebral disc or cervical facet joints produced characteristic and distinguishable pain in normal subjects.[8] The second commonly implicated joint is the C1–2 atlanto-axial joint, which innervates the posterior fossa and portions of the dens.[7] In one study, head pain was demonstrated in human subjects following electrical stimulation of C1 or noxious stimulation of the greater occipital nerve.[9] The C2 nerve root is the main contributor to the greater occipital nerve and due to the unique anatomy of C1 and C2 relative to other vertebrae, the greater occipital nerve is not protected by a cervical facet leaving it prone to injury. This is supported by a study in which direct compression of the greater occipital nerve was achieved in cadavers between the atlas and axis in extension and by adjacent muscles and connective tissue traveling to the occipital scalp in flexion.[10] Less commonly, cervicogenic headache may result from involvement of the C3–C4 zygapophyseal joint,

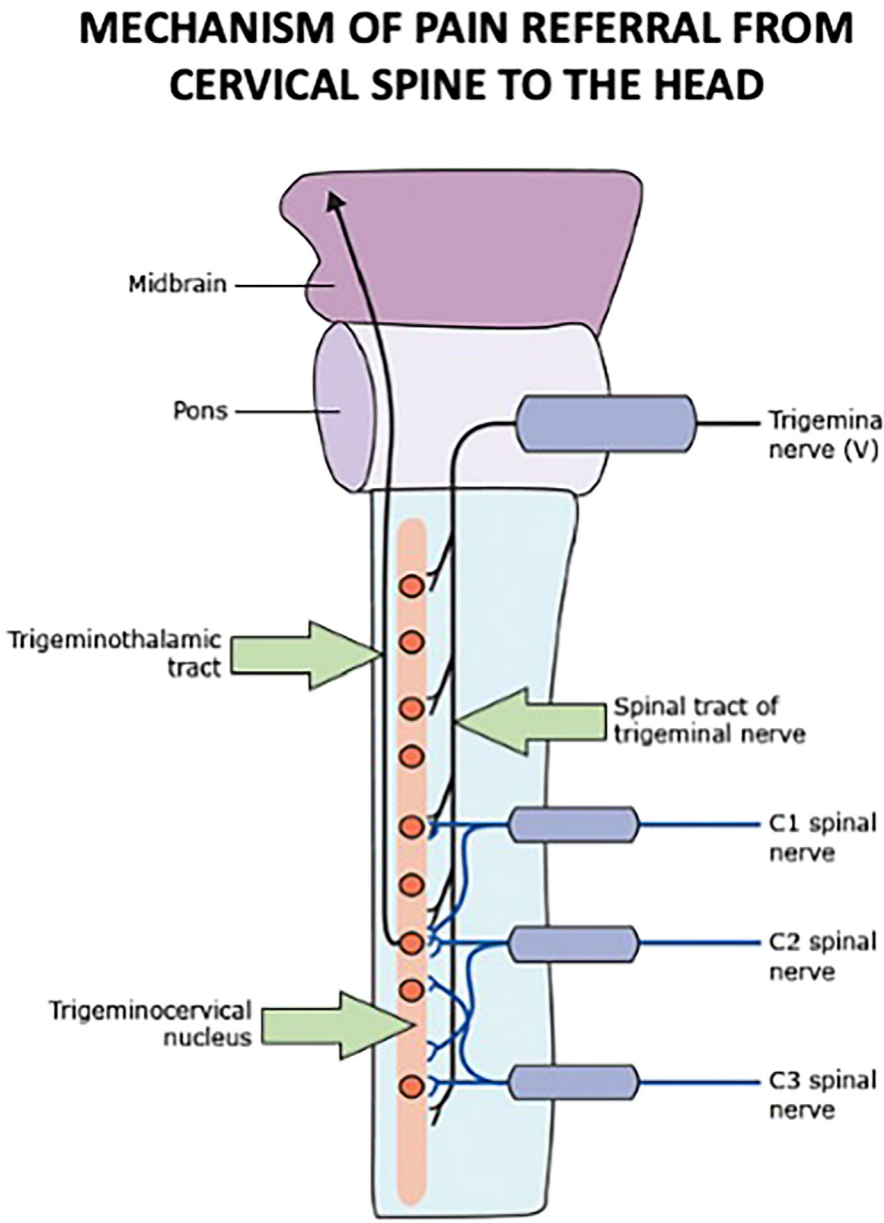

Fig. 1. Mechanism of pain referral from the cervical spine to the head showing nociceptive afferents of the trigeminal and upper 3 cervical spinal nerves converge onto second-order neurons in the trigeminocervical nucleus in the upper cervical spinal cord. This convergence mediates the referral of pain signals from the neck to regions of the head innervated by cervical nerves or the trigeminal nerve. (*Reprinted with permission from* Elsevier. The Lancet Neurology, 2009, 8 (10), October 2009, 959-968.)

upper cervical intervertebral discs, or intraspinal processes affecting the upper cervical roots.[10]

CLINICAL PRESENTATION

Cervicogenic headaches have a varied clinical presentation, particularly because the cervical cause of these headaches is heterogeneous. The most salient and specific features of cervicogenic headaches are concerning the involvement of the neck. Neck pain is typically described as constant, dull, nonthrobbing. It is typically localized to the occiput and is ipsilateral to the side of the headache. The headache is similarly unilateral at the onset, although it may progress to bilateral. It is typically occipital but there have been reports of periorbital, temporal, frontal, and parietal distributions. Myofascial trigger point tenderness may be present in the ipsilateral suboccipital, cervical, or shoulder regions and may refer pain to the head with stimulation.[11] There have been rare reports of radicular symptoms on the side of the headache and sensory dysfunction of the occipital scalp if the greater occipital nerve is involved.

The first diagnostic criterion based solely on clinical features was formalized by the CHISG in 1990 and further amended in 1998 to improve specificity (**Box 1**).[3] The revised criteria were evaluated for interrater reliability and showed fair-to-good agreement with higher scores obtained by expert neurologists and anesthesiologists with more experience diagnosing cervicogenic headache.[11] The criteria prioritize clinical features pertaining to neck involvement, such as precipitation of pain with awkward head positioning of external pressure over the upper cervical/occipital region. Patients may describe protective behaviors to limit neck motion or avoid certain precipitating neck positions.[11] These protective behaviors may also be visible in the examination room before attempting provocative maneuvers. They may present as abnormal posture at baseline or awkward head movements. Restriction of neck range of motion is a critically important clinical feature of cervicogenic headache. Patients with cervicogenic headache typically show at least 10° of restriction on the affected side, and this restriction correlates to headache frequency and associated disability.[12,13]

The characteristics of the head pain itself as well as the additional, although less important, clinical characteristics such as nausea, photophobia, phonophobia, and neck pain are not specific to cervicogenic headaches. Thus, when relying solely on clinical criteria, there exists a substantial possibility of overdiagnosis of cervicogenic headaches.

Box 1
Diagnostic criteria for cervicogenic headache per CHISG

Diagnostic Criteria
- I. Symptoms and signs of neck involvement
  - A. Precipitation of head pain, similar to the usually occurring one
    1. By neck movement and/or sustained awkward head positioning, and/or
    2. By external pressure over the upper cervical or occipital region on the symptomatic side
  - B. Restriction of the range of motion in the neck
  - C. Ipsilateral neck, shoulder, or arm pain of a rather vague nonradicular nature or, occasionally, arm pain of a radicular nature
- II. Confirmatory evidence by diagnostic anesthetic blockades
- III. Unilaterally of the head pain, without side shift

Important notations added to the diagnostic criteria are as follows: points 1A suffices as the sole criterion for the diagnosis whereas points 1B and 1C do not; provisionally, the combination of 1B and 1C is satisfactory; and "unilaterality on two sides" may be acceptable

INTERNATIONAL CLASSIFICATION OF HEADACHE DISORDERS-3 CRITERIA AND CAUSATION

The third edition of the International Classification of Headache Disorders (ICHD-3) is a hierarchical classification system and is widely accepted as the current gold standard diagnostic classification for headache disorders. Cervicogenic headaches are coded under Part II of the ICHD-3 classification, secondary headaches, which, by definition, are headaches that develop as a secondary symptom due to another disorder that is known to cause headaches. The diagnosis is specifically listed under subsection 11.2, headaches attributed to disorders of the neck. It is 1 of 3 headache diagnoses in this subsection, along with headache attributed to retropharyngeal tendonitis and headache attributed to craniocervical dystonia (HACCD).

Per the ICHD-3, cervicogenic headaches are headaches caused by a disorder of the cervical spine and its component bony, disc, and/or soft tissue elements, usually but not invariably accompanied by neck pain and have the following diagnostic criteria (**Box 2**).[14] The diagnosis requires the presence of a headache with or without neck pain (criterion A), the presence of a cervical source for the headache (criterion B), and evidence of causation (criterion C). Causation in cervicogenic headaches is, however, a hotly debated concept because neck involvement in the form of pain or restricted mobility can be a component of primary headache versus a nidus for secondary headache. The ICHD-3 lists causes that certainly and possibly fulfill the criteria of causation, although this list is not exhaustive, and care must be taken to investigate causes outside this list (**Box 3**).[14]

INTERVENTIONAL PROCEDURES: A ROUTE TO DIAGNOSIS, PROBABLE CAUSATION, AND TREATMENT

Interventional procedures can be useful in the treatment and diagnosis of cervicogenic headaches. Here we present 2 clinical vignettes as an avenue to narrow the differential diagnoses and decipher the most reasonable treatment plan.

Vignette #1

A 55-year-old woman presents with a 6-month history of left occipital-temporal headaches, which are dull and aching in quality. She sought care with her primary care doctor who initially treated her for presumed tension headache with a nonsteroidal anti-

Box 2
Diagnostic criteria for cervicogenic headache per ICHD-3

Diagnostic Criteria
A. Any headache fulfilling criterion C
B. Clinical, laboratory, and/or imaging evidence of a disorder or lesion within the cervical spine or soft tissues of the neck, known to be able to cause headache
C. Evidence of causation demonstrated by at least 2 of the following:
   1. Headache has developed in temporal relation to the onset of the cervical disorder or appearance of the lesion
   2. Headache has significantly improved or resolved in parallel with improvement in or resolution of the cervical disorder or lesion
   3. Cervical range of motion is reduced, and headache is made significantly worse by provocative maneuvers
   4. Headache is abolished following diagnostic blockade of a cervical structure or its nerve supply
D. Not better accounted for by another ICHD-3 beta diagnosis

Box 3
Etiologies of causation in cervicogenic headaches per ICHD-3

Certainly fulfill
1. Tumor
2. Fracture
3. Infection
4. Rheumatoid arthritis of the upper cervical spine

May fulfill
1. Cervical spondylosis
2. Osteochondritis

inflammatory drug and a muscle relaxant. She did not improve. On returning, she also complains of bilateral neck pain, although worse on the left side. Radiograph of the cervical spine is obtained, and she is noted to have multiple levels of spondylosis from C2 to C6. She then receives 6 weeks of physical therapy during which she worsens with persistent headaches ranging from moderate to sometimes severe intensity. MRI of the cervical spine confirms similar findings from C2 to C6—spondylosis with mild-to-moderate lateral recess narrowing and facet hypertrophy but no root impingement. MRI of the brain is obtained, and it is normal.

Given the presentation of occipital and temporal headache with ipsilateral neck pain, a provocative maneuver is attempted and produces accentuation of pain without radicular spread. A reasonable next step is a diagnostic and therapeutic block. The highest likely target is the C2–3 facet joint or the third occipital nerve. This assertion is based on the distribution of pain and noted prevalence of cervicogenic headache being most common in the C2–3 area followed by C1–2, then C3–4.[10] A zygapophyseal joint injection at the left C2–3 facet can be achieved via C-arm guided localization and injection with either bupivacaine or a combination of bupivacaine and a nonparticulate steroid such as preservative-free dexamethasone. If the facet injection provides 2 weeks of 70% to 80% relief, then the next therapeutic goal is to achieve more enduring relief with a medial branch block at the superior articular pillar of C3 subjacent to the C2–3 facet. There, the third occipital nerve courses onward along a medial and cephalad course to the occipital scalp (**Figs. 2** and **3**). If at least 90% relief from local anesthetic injection at the medial branch is achieved, then a medial branch radiofrequency ablation can be performed. Relief is apt to last 6 to 12 months with repeat radiofrequency ablation providing similar relief.[15–17] Some clinicians rely on greater than 50% relief but false-positive results increase with a lower bar.

If there is no relief from a facet or medial branch block an atlanto-occipital (C0–1) or atlanto-axis (C1–2) joint injection are potential targets. Lee and colleagues provided data from a study of 29 patients, 20 of whom had at least 50% relief from C0–1 joint injection.[18] This relieved pain in the posterior neck and occipital area but in some patients, there was referred pain extending to the lower neck and scapula that disappeared after the injection. Atlanto-axial involvement causes pain in the upper neck radiating to the suboccipital zone and laterally. However, similar to all the upper cervical joints, referred pain patterns can vary and spread to the ear or scapula. Bogduk and Govind sites referral patterns for the C0–1-related and C1–2-related cervicogenic pain patterns. Volunteers were given injections to expand the joint to induce pain, which mapped the distribution of pain referral.[8,19] Given the varied presentation, a tailored approach to injections for patients' refractory to facet or medial branch blocks is warranted.

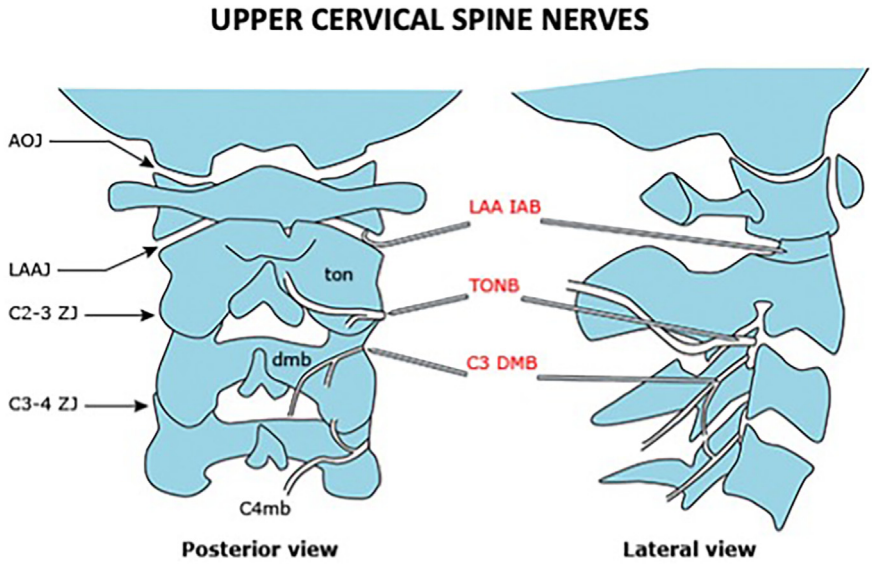

Fig. 2. Posterior and lateral views of the upper cervical spine nerves, showing the leading articular sources of cervicogenic headache, the related nerves, and where needles are placed for diagnostic blocks of these structures.[7] Red labels and needles point to target sites for diagnostic blocks. AOJ, atlanto-occipital joint; C3 DMB, C3 deep medial branch block; C4mb, medical branch of the C4 dorsal ramus; DMB, deep medial branch of the C3 dorsal ramus; LAA IAB, intra-articular block of the lateral atlanto-axial join; LAAJ, lateral atlanto-axial joint; ton, third occipital nerve; TONB, third occipital nerve block; ZJ, zygapophysial joint. (*Reprinted with permission from* Elsevier. The Lancet Neurology, 2009, 8 (10), October 2009, 959-968.)

Vignette #2

A 44-year-old man presents with a history of acute onset severe neck pain associated with lancinating pain along the anterior frontotemporal region extending down the preauricular region, posterior jaw on the left side, and shoulders bilaterally but significantly worse on the left side. There is no numbness or weakness in the upper limbs and cervical spasm is noted. His pain is refractory to treatment with 1 week of scheduled naproxen and methocarbamol. Given the presence of lancinating facial pain and severe neck pain, MRI of the internal auditory canal with and without contrast is obtained and does not reveal vascular compression in the trigeminal region of the pons or ambient cistern. However, MRI of the cervical spine reveals a C4–5 broad disc protrusion with more leftward extension compressing the cord at this level. Neurosurgical consultation is obtained, and although there is no myelopathic physical finding, anterior cervical decompression and fixation is entertained given the trigeminal neuralgic pain and concurrent neck pain. A course of oral steroids helps partially but the severe pain returns as the steroid dosing is tapered and discontinued.

In this patient, possible interventions include epidural steroid injection versus neurosurgical intervention. Given his presentation with spinal stenosis created by a broad disc protrusion with left-sided deviation, it may be appropriate to proceed directly to neurosurgical intervention. However, some interventionalists would first try a less-invasive approach with either interlaminar or transforaminal epidural steroid injection.

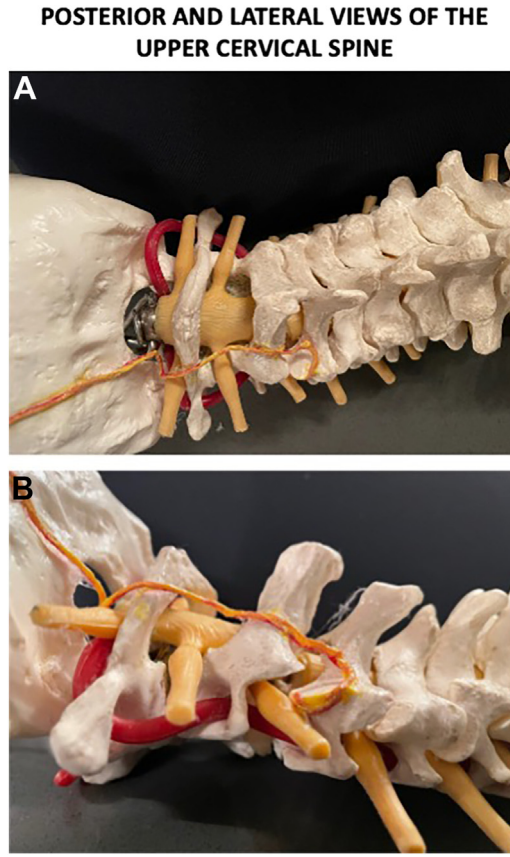

Fig. 3. Posterior and lateral anatomic views of the occipital nerve. (*A*) Posterior view of the upper cervical spine with the third occipital nerve coursing around the superior facet endplate at C3 then cephalad to the occipital skull. The facet joint is purposely separated from the inferior endplate of C2 facet to emphasize the course of the third occipital nerve from its inception as the superficial branch of the dorsal ramus of C3 root-a medical branch path. (*B*) Lateral view of the upper cervical spine demonstrating the location of the vertebral artery and the lack of a true foramen transversarium cephalad to the C2 vertebra. The root and the vertebral artery are relatively nakedly exposed.

Some risks do still exist, including nerve injury particularly with injectate volumes greater than 1 to 2 mL or quick injection causing a pressure surge. Interlaminar injection has less of a risk compared with transforaminal epidural injection with the latter being closer to the vertebral artery increasing the risk of arterial insult. In this case, pursuing surgery will be necessary if conservative measures fail to mitigate this patient's pain. Unrelenting severity of pain would typically prompt surgical intervention sooner rather than later.

COMPLICATIONS FROM CERVICAL INTERVENTIONAL PROCEDURES

Machikanti and colleagues conducted a prospective, nonrandomized study examining the records looking for adverse events for 7500 episodes related to 43,000 facet injections. About 11.4% of the adverse events were due to intravascular penetration,

with 20% cervical, 4% lumbar, and 6% thoracic injections.[20] A meta-analysis study performed by Ekhator and colleagues revealed superior reduction in pain and duration of relief in radiofrequency ablation procedures versus epidural steroid injections with a complication rate of 12%.[21] Only a few anecdotal reports have noted serious side effects such as intravascular injection into the vertebral artery due to error in localization of its trajectory causing vertebral dissection or stroke. Injection of an anesthetic intraarterially could cause vertigo, ataxia, seizure, stroke, or death. If the needle penetrates the facet joint and the tip advances beyond the facet joint anterior margin, inadvertent epidural injection will occur. Local anesthetic spread could cause respiratory depression. Proximity of the dorsal root and thecal envelope of the nerve root at that level could be violated at the distal lateral recess or the foramen resulting in root injury or intrathecal spread of the injectate. In **Fig. 3**, the lateral view of the spine shows the proximity of the vertebral artery to the facet region. The occipital-axial joint and C1–2 are especially notable for vulnerable vessels and exposed nerve roots. Given this anatomy, the risk factor for interventional measures increases at these upper cervical spine levels. This region requires caution and exceptional skills to avoid adverse events. There is also a risk of inadvertent intrathecal entry at the nerve root level. The C7/T1 space is the most capacious epidural space, and it is the most favored level to do an interlaminar epidural injection. A more proximate site near the area of interest can be considered if there is concern for impedance from a stenotic region, fibrosis, or other obstructions. Transforaminal epidural injection in the cervical region accounts for only 2.4% of epidural injections performed, and it has a higher complication rate.[22] Scanlon and colleagues performed a survey study of US physician members of the American Pain Society with 287 respondents.[23] Complications were noted in 78 patients out of 287 who received transforaminal epidural steroids. There were 12 cervical spine infarcts; 13 fatalities from posterior circulation infarcts, combined brain, and cervical infarcts with seizure; and 5 unspecified adverse events. Inamasu and Guiot reviewed the incidence of vertebral artery injury from diagnostic and therapeutic cervical procedures including embolic events, arterial dissection, or inadvertent intraarterial injection of anesthetic causing seizures, respiratory depression, or other serious events.[24] Recognition of the presentation of arterial dissection requires vigilance especially since some patients' symptoms may evolve subacutely. Neck pain and occipital pain is commonly seen and if unrecognized, occlusion of the vertebral artery could be devastating. Serious complications are reported as notably rare, nonetheless, it should be stressed that appropriate measures should be implemented to minimize risk. There is a likelihood that adverse events are underreported. Further studies and mandatory reporting mechanisms should be instituted to truly reflect the procedural risk and adverse events.

A MULTIFACETED APPROACH TO DIAGNOSIS

The successful diagnosis of cervicogenic headache relies on a careful history including thorough review of systems, as well as a physical examination. We recommend a combined approach to diagnosis that includes careful consideration of clinical criteria, assessment of causation, and evaluation of response to treatment to most accurately diagnose cervicogenic headaches. Here, we propose a diagnostic algorithm that combines the most pertinent and specific clinical features per the CHISG, the causative recommendations of the ICHD-3 criteria, and the response to validated treatment options (**Fig. 4**).

Per the CHISG, the presence of characteristic head pain precipitated by neck movement and/or sustained awkward head positioning and/or external pressure over the

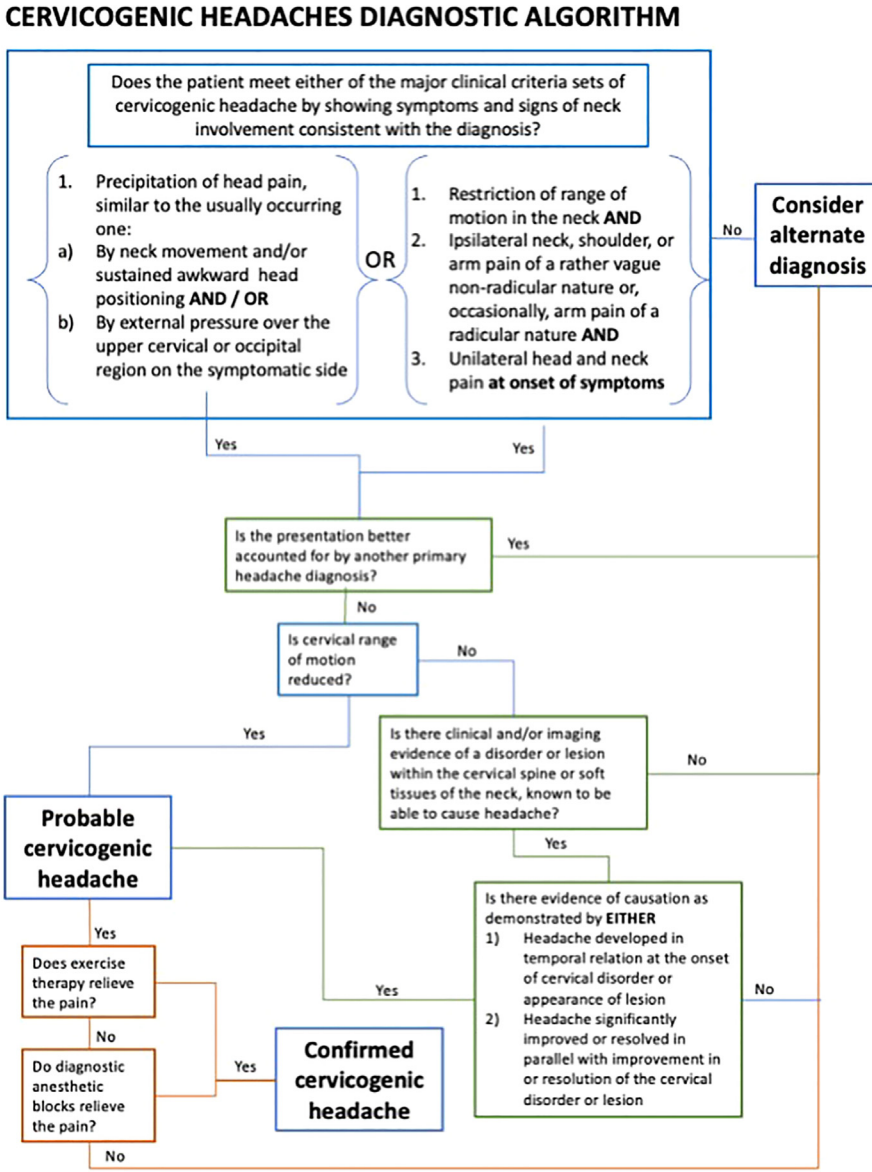

Fig. 4. A proposed diagnostic algorithm for cervicogenic headaches incorporating clinical features (*blue boxes*), causation criteria (*green boxes*), and intervention-based confirmation (*red boxes*). Endpoints are either confirmed cervicogenic headache, probable cervicogenic headache, or alternate diagnosis. A confirmed diagnosis of cervicogenic headache will, at a minimum, meet ICHD-3 criteria.

upper cervical or occipital region on the symptomatic side secures a diagnosis of cervicogenic headache. It is also proposed that a combination of (1) restriction of the range of motion in the neck, (2) characteristic ipsilateral neck, shoulder, or arm pain and (3) unilaterality of the headache at onset of symptoms secures the diagnosis. For research

purposes, CHISG recommends that the combination of these clinical features must be supported by confirmatory evidence with diagnostic anesthetic blockades. For our clinical diagnostic purposes that aim to be practical and prioritize noninvasive measures, diagnostic anesthetic blockades are not required to fulfill the initial clinical diagnostic criteria and are only introduced later.

Although the CHISG suggests that the clinical points outlined are sufficient to provide a diagnosis, the ICHD-3 criteria require additional proof of causation, as well as a concerted effort to rule out common mimicking entities. Thus, if clinical criteria are fulfilled per the CHISG criteria and the presentation is not better accounted for by an alternate ICHD-3 diagnosis, the algorithm moves on to establish causation, specifically by introducing criterion B, followed by criterion C1 and C2 which are 2 clinical features that both help establish causation and are not accounted for by the CHISG. At this stage in the algorithm, workup will likely be expanded to include laboratory testing to rule out alternate systemic sources of pain, and imaging of the head and neck with either MRI or computed tomography (CT). Cervical spine MRI or CT do adequately demonstrate cervical facet hypertrophy when examined in the sagittal or axial plane and so may be useful in establishing a cervical source of pain.[11] Imaging also aids in ruling out sources of pain that may warrant alternate intervention such as vertebral artery dissection, aneurysm, or myelopathy.

Fulfillment of the most salient clinical features per the CHISG as well as establishment of causation per the ICHD-3 result in a diagnosis of probable cervicogenic headache, which warrants intervention in a stepwise manner with priority given to the most noninvasive treatment options. To further solidify and confirm the diagnosis of cervicogenic headache, the algorithm offers therapeutic diagnostic modalities. We recommend intervention with physical therapy followed by or concurrently with diagnostic anesthetic blocks at the discretion of the provider based on degree of pain and disability. Substantial improvement in pain or full resolution results in a diagnosis of confirmed cervicogenic headache.

This diagnostic algorithm is novel in that it provides a more practical and stepwise manner for the diagnosis of cervicogenic headaches, an entity often clouded by alternate contributing sources of pain such as primary headaches, alternate secondary headaches, or nonheadache neurologic diagnoses. It first establishes clear clinical criteria per the most validated criteria available, and priority is given to features that have a higher degree of specificity for cervicogenic headaches. This is followed by a rigorous evaluation of causation per ICHD-3 consensus criteria. This step often requires laboratory testing and imaging to rule out alternate causes and possibly establish a cervical source of pain. Only after both these criteria are met are therapeutic diagnostic modalities offered. This approach is important because simultaneous treatment of the various etiologies on the differential for cervicogenic headaches is usually underway. It becomes difficult to tease out the true cause of headache when multiple interventions are performed simultaneously. A high clinical and causative degree of suspicion is required before the initiation of therapeutic intervention targeted to treat cervicogenic headaches, especially when considering invasive options.

This approach may also reduce the burden of health-care costs associated with jumping straight to anesthetic blocks. There are multiple checkpoints in the algorithm that lead the practitioner to consider alternate diagnoses and halt their progress down the cervicogenic headache diagnostic and therapeutic path. In addition, invasive therapeutic interventions are often not immediately covered by insurance companies. Insurance companies often rely on evidence-based metrics or a preponderance of expert consensus to finance treatment, both of which are lacking in the research and literature surrounding cervicogenic headache. Adherence to this stepwise path

may also reduce unnecessary invasive measures and subsequently reduce the financial burden on the patient.

DIFFERENTIATING FROM PRIMARY OR OTHER SECONDARY HEADACHES

The definitive diagnosis of cervicogenic headaches is complicated by the fact that patients may not present purely with one type of headache. Thus, treating a patient without considering wider differential diagnoses and management options can be detrimental and ineffective for the patient. Often it is useful to treat the headache phenotype depending on the presentation.[25] It is helpful to categorize headaches based on whether it is a primary headache disorder or a secondary headache disorder. Primary headaches that may be confused with or coexist with cervicogenic headaches include migraine and tension-type headaches.

Migraines are characterized by paroxysmal moderate-to-severe headaches associated with nausea and/or vomiting or photophobia and phonophobia. Neck pain may be present in up to 76% of patients with migraines.[26] The pathophysiology of cervicogenic headaches through the activation of the trigeminal nerve may also consequently activate nociceptive afferents involved in migraines resulting in a mixed clinical picture such that it is difficult to differentiate between the 2 headache types.

Similarly, patients with primary tension-type headaches often have neck pain and may be present in up to 88% of these patients.[26] Tension-type headaches are characterized by mild-to-moderate headaches often bilateral and nonpulsating and are not associated with nausea or vomiting, although phonophobia or photophobia may exist.[14] Cervical myofascial pain is common in patients with tension-type headaches and cervicogenic headaches. Thus, similar pathophysiology is noted with peripheral sensitization through the trigeminal nucleus caudalis causing increased pain in both headache types. Fortunately, management options for the primary headache types often overlap but do not aid in differentiating the type of headache.

DIFFERENTIATING FROM OTHER NEUROLOGIC DIAGNOSES

Disorders such as cervical or vertebral artery dissection, posterior fossa tumor, multiple sclerosis, or retropharyngeal abscess may present with similar symptoms as cervicogenic headache but the associated symptoms including focal neurologic signs, fever, meningismus, and acuity would effectively lower the suspicion for cervicogenic headaches. Other neurologic conditions to consider when diagnosing cervicogenic headaches include occipital neuralgia and cervical dystonia.

Cervicocerebral artery dissection is an important differential diagnosis to consider for cervicogenic headaches. Extracranial or intracranial dissections can present with acute severe unilateral head and neck pain typically ipsilateral to the side of the dissection accompanied by focal neurologic symptoms. Dissections involving the anterior circulation often present with temporal or frontal pain, whereas vertebrobasilar dissections involve occipital and nuchal pain thus mimicking cervicogenic headaches.[27] Persistent headache and neck pain after cervicocerebral artery dissection can also occur and in many studies reveal that this affects about 20% of patients 6 months to 3 years after the dissection.[28] Posterior circulation dissections and a history of primary headaches could predispose to persistent headache and neck pain after a cervicocerebral artery dissection.

Occipital neuralgia is characterized by unilateral or bilateral severe paroxysmal shooting, stabbing, or sharp pain around the greater, lesser, or third occipital nerves.[14] The pain is associated with allodynia and tenderness on applying pressure to the area. The greater occipital nerve is most often affected, 90% of the time, compared with

10% of patients with lesser occipital nerve involvement, and rare involvement of the third occipital nerve.[29] Occipital nerve blocks can provide sustained relief for occipital neuralgia but have also been shown to alleviate both migraine and cervicogenic headaches.

Cervical dystonia is an idiopathic condition causing involuntary muscle contractions of isolated muscle groups in the cervical region resulting in twisting movements and abnormal postures. HACCD is characterized by neck pain that is in the location of the dystonic muscle, has either worsened or developed around the time of the cervical dystonia, and/or has improved as the cervical dystonia improved. In one study, pain and disability were significantly worse in patients with HACCD compared with patients with cervical dystonia without headaches and can be seen in up to 29% of patients with cervical dystonia.[30]

SUMMARY

Cervicogenic headache is a unique clinical entity, which presents with head and neck pain due to a cervical source and affects a significant proportion of the population. Since its recognition as a secondary headache disorder, there has been debate regarding the most sensitive and specific diagnostic measures, and this has posed a dilemma for diagnosing practitioners and researchers. This is further clouded by the many neurologic and nonneurologic mimickers of cervicogenic headache pain.

We provide a systematic algorithm for diagnosis that serves as a practical approach that prioritizes ruling in patients based on a critical assessment of their clinical presentation and physical examination, followed by the establishment of causation, and finally confirmation of suspicion with interventions that are simultaneously therapeutic. Further research is required to better define treatment options for this patient population. This, and other studies of cervicogenic headache, will be facilitated by this consistent and standardized approach to diagnosis.

CLINICS CARE POINTS

- Given the varied presentations of cervicogenic headaches, it is important to utilize a standardized algorithm for diagnosis and treatment.
- Interventional procedures are useful for the treatment and diagnosis of cervicogenic headaches and require a careful targeted approach.
- Treatment and confirmation of cervicogenic headaches includes diagnostic anesthetic blockade and exercise therapy to relieve pain.
- The differential diagnosis of cervicogenic headaches, although extensive, can often involve similar management strategies such as with migraine, tension-headache, and occipital neuralgia.

DISCLOSURE

The authors have no conflicts of interest or relevant financial disclosures.

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