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Cervicogenic Headache: Diagnostic Evaluation and Treatment Strategies

By David M. Biondi, DO

Neck pain and cervical muscle tenderness are common and prominent symptoms of primary headache disorders.[1] Less commonly, head pain may actually arise from bony structures or soft tissues of the neck, a condition known as "cervicogenic headache."[2] Cervicogenic headache can be a perplexing pain disorder that is refractory to treatment if it is not recognized. The pathophysiology and source of pain in this condition have been debated,[3-5] but it is believed to be referred from one or more muscular, neurogenic, osseous, articular, and vascular structures in the neck.[6] The trigeminocervical nucleus is an area of the upper cervical spinal cord where sensory nerve fibers in the descending tract of the trigeminal nerve (trigeminal nucleus caudalis) are believed to interact with sensory fibers from the upper cervical roots. This functional convergence of upper cervical and trigeminal sensory pathways allows the bidirectional referral of painful sensations between the neck and trigeminal sensory receptive fields of the face and head.[6] This issue of *Pain Management Rounds* presents an overview of cervicogenic headache, focusing on its causes, diagnosis, and treatment.

NECK PAIN AS A MANIFESTATION OF MIGRAINE

Neck pain and muscle tension are common symptoms of a migraine attack.[1,7-9] In a study of 50 migraine patients, 64% reported neck pain or stiffness associated with their migraine attack, with 31% experiencing neck symptoms during the prodrome, 93% during the headache phase, and 31% during the recovery phase.[1] In this study, 7 patients reported that pain referred into the ipsilateral shoulder and 1 patient reported that their pain extended from the neck into the lower back region. In another study of 144 migraine patients from a university-based headache clinic, 75% of patients reported neck pain associated with migraine attacks.[8] Of these patients, 69% described their pain as "tightness," 17% reported "stiffness," and 5% reported "throbbing." The neck pain was unilateral in 57% of respondents, 98% of whom reported that it occurred ipsilateral to the side of headache. The neck pain occurred during the prodrome in 61%, the acute headache phase in 92%, and the recovery phase in 41%. Recurrent, unilateral neck pain without headache was found to be a variant of migraine.[10] Careful history gathering in cases of recurrent neck pain discerned that previously overlooked symptoms were either similar or identical to those associated with migraine.

Differences in neck posture, pronounced levels of muscle tenderness, and the presence of myofascial trigger points were observed in subjects with migraine, tension-type headache, or a combination of both, but not in a non-headache control group.[1,11,12] A comparison between the headache groups demonstrated no significant differences in myofascial symptoms or signs, dispelling the common belief that tension-type headache is associated with a greater degree of musculoskeletal involvement than migraine.[12]

HEADACHE AS A MANIFESTATION OF NECK DISORDERS

Head pain that is referred from the bony structures or soft tissues of the neck is commonly called "cervicogenic headache." It is often the sequela of a head or neck injury, but may also occur in the absence of trauma. The clinical features of cervicogenic headache may mimic those commonly

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TABLE 1: The Cervicogenic Headache International Study Group Diagnostic Criteria

Major criteria of cervicogenic headache

(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 (ROM) in the neck
- (c) ipsilateral neck, shoulder, or arm pain of a rather vague nonradicular nature or, occasionally, arm pain of a radicular nature.

Points (I) (a through c) are set forth in a surmised sequence of importance. It is obligatory that one or more of the phenomena in point (I) are present. Point (a) suffices as the sole criterion for positivity within group (I); points (b) or (c) do not. Provisionally, the combination of (I) (b and c) has been set forth as a satisfactory combination within (I). The presence of all three points (a, b, and c) fortifies the diagnosis (but still point (II) is an additional obligatory point for scientific work).

(II) Confirmatory evidence by diagnostic anesthetic blockades. Point (II) is an obligatory point in scientific work.

(III) Unilaterality of the head pain, without sideshift. For scientific work, point (III) should preferably be adhered to.

Head pain characteristics

(IV) (a) moderate-severe, nonthrobbing, and non-lancinating pain, usually starting in the neck
- (b) episodes of varying duration, or
- (c) fluctuating, continuous pain

Other characteristics of some importance

(V) (a) only marginal effect or lack of effect of indomethacin
- (b) only marginal effect or lack of effect of ergotamine and sumatriptan
- (c) female sex
- (d) not infrequent occurrence of head or indirect neck trauma by history, usually of more than only medium severity

None of the single points under (IV) and (V) are obligatory.

Other features of lesser importance

(VI) Various attack-related phenomena, only occasionally present:
- (a) nausea
- (b) phonophobia and photophobia
- (c) dizziness
- (d) ipsilateral "blurred vision"
- (e) difficulties on swallowing
- (f) ipsilateral edema, mostly in the periocular area

TABLE 2: Clinical characteristics of cervicogenic headache

- Unilateral head or face pain without sideshift; the pain may occasionally be bilateral
- Pain localized to the occipital, frontal, temporal, or orbital regions
- Moderate-to-severe pain intensity
- Intermittent attacks of pain lasting hours to days, constant pain or constant pain with superimposed attacks of pain
- Pain is generally deep and nonthrobbing in character; throbbing may occur when migraine attacks are superimposed
- Head pain is triggered by neck movement, sustained or awkward neck postures; digital pressure to the suboccipital, C2, C3, or C4 regions or over the greater occipital nerve; valsalva, cough, or sneeze might also trigger pain
- Restricted active and passive neck range of motion; neck stiffness
- Associated signs and symptoms can be similar to typical migraine accompaniments including nausea, vomiting, photophobia, phonophobia, and dizziness; others include ipsilateral blurred vision, lacrimation, and conjunctival injection or ipsilateral neck, shoulder, or arm pain

associated with primary headache disorders such as tension-type headache, migraine, or hemicrania continua and, as a result, distinguishing among these headache types can be difficult. The prevalence of cervicogenic headache in the general population is estimated to be 0.4% – 2.5%, but is as high as 20% in patients with chronic headache.[13] The mean age of patients with this condition is 42.9 years and it is 4 times more prevalent in women. The Cervicogenic Headache International Study Group developed diagnostic criteria that provide a detailed, clinically useful description of the condition (Table 1).[14] The diagnosis of cervicogenic headache can often be made without resort to diagnostic neural blockade by completion of a careful history and physical examination (Table 2).

DIAGNOSTIC TESTING FOR SUSPECTED CERVICOGENIC HEADACHE

Diagnostic imaging (eg, x-ray, magnetic resonance imagine [MRI], and computed tomography [CT] myelography) cannot confirm the diagnosis of cervicogenic headache, but can lend support to its diagnosis.[15] A comprehensive history, review of systems, and physical examination, including a complete neurological assessment, will often identify the potential for an underlying structural disorder or systemic disease.[16] Imaging is primarily used to search for secondary causes of pain that may require surgery or other more aggressive forms of treatment.[17] The differential diagnoses in cases of suspected cervicogenic headache include posterior fossa tumor, Arnold-Chiari malformation, cervical spondylosis or arthropathy, herniated intervertebral disc, spinal nerve compression or tumor, arteriovenous malformation, vertebral artery dissection, and intramedullary or extramedullary spinal tumors. Laboratory evaluation may be necessary to search for systemic diseases that may adversely affect muscles, bones, or joints (ie, rheumatoid arthritis, systemic lupus erythematosus, thyroid or parathyroid disorders, primary muscle disease, etc).

Zygapophyseal joint, cervical nerve, or medial branch blockade are used to confirm the diagnosis of cervicogenic headache and predict the treatment modalities that will most likely provide the greatest efficacy. The first 3 cervical spinal nerves and their rami are the primary peripheral nerve structures that can refer pain to the head.

• The ***suboccipital nerve (dorsal ramus of C1)*** innervates the atlanto-occipital joint; therefore, pathology or injury affecting this joint is a potential source of head pain that is referred to the occipital region.

• The ***C2 spinal nerve and its dorsal root ganglion*** have a close proximity to the lateral capsule of the atlantoaxial (C1-2) zygapophyseal joint and innervate the atlantoaxial and C2-3 zygapophyseal joints; therefore, trauma to or pathologic changes around these joints can be a source of referred head pain. C2 neuralgia is typically described as a deep or dull pain that usually radiates from the occipital to parietal, temporal, frontal, and periorbital regions. A paroxysmal sharp or shock-like pain is often superimposed over the constant pain. Ipsilateral eye lacrimation and conjunctival injection are common associated signs. Arterial or venous compression of the C2 spinal nerve or its dorsal root ganglion have been suggested as a cause for C2 neuralgia in some cases.[11,19,20]

• The ***third occipital nerve (dorsal ramus C3)*** has a close anatomic proximity to and innervates the C2-3 zygapophyseal joint. This joint and the third occipital nerve appear most vulnerable to trauma from acceleration-deceleration ("whiplash") injuries of the neck.[21] Pain from the C2-3 zygapophyseal joint is referred to the occipital region, but is also referred to the frontotemporal and periorbital regions. Injury to this region is a very common cause of cervicogenic headache. The majority of cervicogenic headaches occurring after whiplash resolve within a year of the trauma.[22] Of interest are reports that patients with chronic headache have experienced substantial pain relief after diskectomy at spinal levels as low as C5-6.[23,24]

Diagnostic anesthetic blockade for the evaluation of cervicogenic headache can be directed to several anatomic structures such as the greater occipital nerve (dorsal ramus C2), lesser occipital nerve, atlanto-occipital joint, atlantoaxial joint, C2 or C3 spinal nerve, third occipital nerve (dorsal ramus C3), zygapophyseal joint(s), or intervertebral discs based on the clinical characteristics of the pain and findings of the physical examination.[25] Fluoroscopic or interventional magnetic resonance imaging (iMRI)-guided blockade may be necessary to assure accurate and specific localization of the pain source.[26-28]

Occipital neuralgia is a specific pain disorder characterized by pain that is isolated to sensory fields of the greater or lesser occipital nerves.[29] The classical description of occipital neuralgia includes the presence of constant deep or burning pain with superimposed paroxysms of shooting or shock-like pain. Paresthesia and numbness over the occipital scalp are usually present. It is often difficult to determine the true source of pain in this condition. In its classical description, the pain of occipital neuralgia is believed to arise from trauma to, or entrapment of, the occipital nerve within the neck or scalp, but the pain may also arise from the C2 spinal root, C1-2 or C2-3 zygapophyseal joints, or pathology within the posterior cranial fossa. Occipital nerve blockade, as it is typically performed in the clinic setting, often results in a nonspecific regional blockade rather than a specific nerve blockade and this may result in the misidentification of the occipital nerve as the source of pain. This "false localization" might lead to unnecessary interventions aimed at the occipital nerve (eg, surgical transection or other neurolytic procedures).[5]

A regional myofascial pain syndrome (MPS) affecting cervical, pericranial, or masticatory muscles can be associated with referred head pain. Sensory afferent nerve fibers from upper cervical regions have been observed to enter the spinal column by way of the spinal accessory nerve before entering the dorsal spinal cord.[30,31] It is believed that the close association between sensorimotor fibers of the spinal accessory nerve and spinal sensory nerves allows for a functional exchange of somatosensory, proprioceptive, and nociceptive information from the trapezius, sternocleidomastoid and other cervical muscles to converge in the trigeminocervical nucleus, ultimately resulting in the referral of pain to trigeminal sensory fields of the head and face. Muscular trigger points, a hallmark of MPS, are discreet hyperirritable areas of contracted muscle that have a lowered pain threshold and refer pain to distant sites in predictable and reproducible patterns.[32,33] Anesthetic injections into trigger point regions can assist in the diagnostic evaluation and therapeutic management of referred head or face pain from cervical muscular sources.[32]

TREATMENT OF CERVICOGENIC HEADACHE

The successful treatment of cervicogenic headache usually requires a multifaceted approach using pharmacological, non-pharmacological, manipulative, anesthetic, and occasionally, surgical interventions [Table 3]. Medications alone are often ineffective or provide only modest benefit for this condition. Anesthetic injections can temporarily reduce pain intensity, but their greatest benefit is allowing greater participation in physical treatments. The success of diagnostic cervical spinal nerve, medial branch, or zygapophyseal joint blockade can predict response to radiofrequency thermal neurolysis.[34] Developing an individualized treatment plan enhances successful outcomes.

Pharmacological treatment

Pharmacological treatments for cervicogenic headache include many medications used for the preventive or palliative management of migraine and neuropathic pain syndromes. The listed medications have neither been approved by the Food and Drug Administration (FDA), nor rigorously studied in controlled clinical trials for the treatment of cervicogenic headache. They are only suggested as potential treatments based on the anecdotal experiences of clinicians treating this condition or similar pain disorders. The side effects and laboratory monitoring guidelines provided are not intended to be comprehensive and consultation of standard references or product package inserts are recommended prior to prescribing any of these medications.

TABLE 3: Potential treatment interventions for cervicogenic headache

Pharmacological*
- tricyclic antidepressants (amitriptyline, nortriptyline, doxepin, desipramine, and others)
- antiepileptic drugs (gabapentin, carbamazepine, topiramate, divalproex sodium, and others)
- muscle relaxants (tizanidine, baclofen, cyclobenzaprine, metaxalone, and others)
- nonsteroidal, anti-inflammatory drugs
  - nonselective COX inhibitors (indomethacin, ibuprofen, naproxen and others)
  - COX-2 selective inhibitor (celecoxib)

Non-pharmacological
- manipulative or manual therapies
- physical therapy
- transcutaneous electrical nerve stimulation (TENS)
- biofeedback/relaxation therapies
- individual psychotherapy

Interventional
- anesthetic blockade
  - spinal roots, nerves, rami, or branches
  - zygapophyseal joints
  - muscular trigger points
- neurolytic procedure
  - radiofrequency thermal neurolysis
- botulinum toxin injections
- occipital nerve stimulator

Surgical
- neurectomy
- dorsal rhizotomy
- microvascular decompression
- nerve exploration and "release"
- zygapophyseal joint fusion

* None of the listed medications are given an indication for this condition by the FDA

Many patients with cervicogenic headache overuse or become dependent on analgesics. Medication – when used as the only treatment for cervicogenic headache – does not generally provide substantial pain relief in most cases. Despite this observation, the judicious use of medications can provide enough pain relief to allow greater patient participation in a physical therapy and rehabilitation program. To improve compliance, medications are initially prescribed at a low dose and increased over 4 to 8 weeks as necessary and tolerated. The cautious combining of medications from different drug classes or with complementary pharmacological mechanisms may provide greater efficacy than using individual drugs alone (ie, an antiepileptic drug combined with a tricyclic antidepressant). Frequent follow-up visits for medication dosage adjustments, monitoring of serum drug levels, and evidence of medication toxicity are recommended.

Antidepressants

The tricyclic antidepressants (TCAs) have long been used for management of various neuropathic, musculoskeletal, head, and face pain syndromes. Analgesic dosages are typically lower than those required for the treatment of depression. The norepinephrine and serotonin reuptake inhibitors (SNRIs) such as venlafaxine and duloxetine have been anecdotally observed to be helpful in the prophylactic management of migraine. Similar observations have been reported for venlafaxine in the treatment of painful diabetic neuropathy, fibromyalgia, and regional myofascial pain syndromes, while duloxetine is indicated for the management of painful diabetic neuropathy. The selective serotonin reuptake inhibitors (SSRIs) are generally ineffective for pain control.

Antiepileptic drugs (AEDs)

The antiepileptic drugs (AEDs) are believed to be modulators or stabilizers of peripheral and central pain transmission and are commonly used for the management of neuropathic, head and face pain syndromes. ***Divalproex sodium*** is indicated for the preventive management of migraine headache and may be effective for cluster headaches, as well as other neurogenic pain syndromes. Serum drug levels can be used as a therapeutic dosing guide. Monthly monitoring of liver transaminases and a complete blood count for evidence of toxicity is recommended, especially during the first 3 to 4 months of treatment or whenever dosages are escalated.
Gabapentin is indicated for the management of postherpetic neuralgia and has been used for managing other neuropathic pain syndromes and migraine. No specific laboratory monitoring is usually necessary.
Topiramate is indicated for migraine prophylaxis and has been anecdotally reported to be effective in the management of painful diabetic neuropathy and cluster headaches. Intermittent monitoring of serum electrolytes might be needed because of its diuretic effect through carbonic anhydrase inhibition.
Carbamazepine is an effective medication in the treatment of trigeminal neuralgia and central neuropathic pain. Serum drug levels can be used as a therapeutic dosing guide. Monthly monitoring of liver transaminases and a complete blood count is recommended, especially during the first 3 to 4 months of treatment or whenever dosages are increased. Several of the other newer AEDs might be used when other treatments are ineffective.

Analgesics

Non-steroidal anti-inflammatory drugs (NSAIDs) may be used as regularly scheduled medications for round-the-clock management of chronic pain or as needed for the management of acute pain. The COX-2 selective antagonist, celecoxib, appears to have less gastrointestinal toxicity than nonselective NSAIDs, but renal toxicity after long-term use remains a concern.

Narcotic analgesics have not generally been effective in the long-term management of cervicogenic headache, but may be cautiously prescribed for temporary pain relief. Migraine-specific abortive medications such as ergot derivatives or triptans are not effective for the chronic head pain of cervicogenic headache, but may relieve the pain of episodic migraine attacks that can occur in some patients.

Other medications

Muscle relaxants, especially those with central activity such as tizanidine and baclofen may provide some analgesic efficacy. Botulinum toxin, type A, injected into pericranial and cervical muscles is a promising treatment for migraine and cervicogenic headache,[35,36] but further clinical and scientific study is needed.

Physical and manual therapies

Physical therapy is an important therapeutic modality for the rehabilitation of cervicogenic headache.[37] The intensity of headache might initially worsen during or after physical therapy especially if it is vigorously applied. Physical treatment is better tolerated when initiated with gentle muscle stretching and manual cervical traction. Therapy can be slowly advanced, as tolerated, to include strengthening and aerobic conditioning. Using anesthetic blockade and neurolytic procedures for temporary pain relief can enhance the efficacy of physical therapy.

Psychological and behavioral treatment

Psychological and non-pharmacological interventions such as biofeedback, relaxation, and cognitive-behavioral therapy are important adjunctive treatments in the comprehensive management of pain.[38] Ongoing intensive, individual psychotherapy is often required if the chronic pain patient has a prominent affective or behavioral component and the pain persists despite aggressive treatment.

Anesthetic blockade and neurolysis

Cervical epidural steroid injections may be indicated in cases of multilevel disc or spine degeneration.[39] Greater and lesser occipital nerve blockade may provide temporary, but substantial pain relief in some cases.[40] Trigger point injections with a local anesthetic may also provide temporary pain relief and relaxation of local muscle spasm. If diagnostic blockade of the cervical nerve, medial branch, or zygapophyseal joint is successful in providing substantial, but temporary pain relief, the treatment algorithm can then proceed to consideration of a longer-acting neurolytic procedure (eg, radiofrequency thermal neurolysis).[34,41,42] After anesthetic blockade and neurolytic procedures, a course of physical therapy and rehabilitation is recommended to enhance functional restoration and affect a longer lasting analgesic benefit.

Surgical treatment

A variety of surgical interventions have been performed for presumed cases of cervicogenic headache.[3] Surgical procedures such as neurectomy, dorsal rhizotomy, and microvascular decompression of nerve roots or peripheral nerves are not generally recommended without compelling radiological evidence for a surgically-correctable pathology or a history of symptoms that are refractory to all reasonable nonsurgical treatments. Surgical liberation of the occipital nerve from "entrapment" in the trapezius muscle or surrounding connective tissues can provide substantial, but temporary pain relief in some cases.[43] Similarly, only temporary pain relief is observed after surgical transection of the greater occipital nerve.[43] Intensification of pain or anesthesia dolorosa are potential adverse outcomes to consider when contemplating the use of surgical interventions.

SUMMARY

Cervicogenic headache is a relatively common cause of chronic headache that is often misdiagnosed or unrecognized. Its presenting symptom complex can be similar to that of the more commonly encountered primary headache disorders such as migraine or tension-type headache. Early diagnosis and management by way of a comprehensive, multidisciplinary pain treatment program can significantly decrease the protracted course of costly treatment and disability that is often associated with this challenging pain disorder.

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