Cervical Facet Arthropathy and Occipital Neuralgia: Headache Culprits

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Abstract Cervicogenic headache (CH) is pain referred from the neck. Two common causes are cervical facet arthropathy and occipital neuralgia. Clinical diagnosis is difficult because of the overlying features between primary headaches such as migraine, tension-type headache, and CH. Interventional pain physicians have focused on supporting the clinical diagnosis of CH with confirmatory blocks. The treatment of cervical facet arthropathy as the source of CH is best approached with a multidimensional plan focusing on physical therapy and/or manual therapy. The effective management of occipital neuralgia remains challenging, but both injections and neuromodulation are promising options.

Keywords Cervicogenic headache · Occipital neuralgia · Cervical facet arthropathy · Occipital nerve stimulation · Occipital nerve block

Introduction

The recognition of cervical spine pathology as a source of headache has grown over the last 10 years. Cervicogenic headache (CH) is pain referred to the head from the cervical region. The prevalence of cervicogenic headache ranges from 0.4% to 2.5% of the general population, but in pain-management clinics, it represents 15% to 20% of patients with chronic headache [1]. The combined interest of both neurologists and interventional pain-management physi-
cians in the treatment of CH has led to multiple ways of diagnosing and treating it. This article focuses on two common sources of CH: cervical facet arthropathy and occipital neuralgia.

Anatomy and Pathophysiology

The facet joint is a synovial-lined joint that links two vertebral bodies. These joints allow the spine to flex, extend, and rotate. The wear and tear on the body from movement over time can cause these joints to become arthritic and painful. Over and above our intervertebral discs shrink as part of the normal aging process. Up to 70% of the compressive force usually applied to the discs transfers to the facet joints [2]. Flexion/extension injuries also are a common cause of cervical facet pathology. Facet pathology after whiplash injuries has been extensively studied, with focus on the C2–C3 joint [3]. The pathophysiology of whiplash injuries remains unclear. It is hypothesized that there is excessive facet flexion/extension caused by a sudden acceleration–deceleration force. This results in the release of inflammatory cytokines.

Sensory innervation from the facet joints is provided by small medial branch nerves that transport afferent impulses to the brain for interpretation via their respective nerve roots (Fig. 1).

The cervical facet joints long have been suspected as a possible source of neck pain and headache. To test this theory, Dwyer et al. [4] injected the cervical facet joints of normal volunteer subjects with contrast to extend the joints. They then asked the volunteers if they had pain, and if so, to identify the location. Volunteers did report pain on extension of the joint; the referred-pain location of each facet joint then was mapped out, similar to the
Fig. 1 Medial branch innervations of the facet joints. Each facet joint is innervated by two medial branch nerves. Both medial branch nerves need to be blocked in order to denervate a facet joint.

dermatome map. This experiment supported the hypothesis that cervical facet joints could be a source of pain, because in fact there was a specific referral pattern of pain from each individual facet joint (Fig. 2).

The cervical joints that have been implicated in CH are innervated by the first three cervical roots, C1, C2, and C3. The anatomy of the first three cervical facet joints are as follows. The occiput articulates with the C1 vertebral body (the atlas), forming the occiput-atlas (OA) joint and the C1 body (the axis) articulates with the C2 body (the axis), forming the atlantoaxial (AA) joint. The OA and AA joints are innervated by the ventral rami of the first and second cervical spinal nerves. There is no vertebral disc between the OA and AA joints. Another source of pain implicated in CH is the C2–C3 facet joint, which is innervated by two branches of the dorsal ramus of the third cervical spinal nerve.

The upper cervical nerve roots also are the origin of the greater and lesser occipital nerves (Fig. 3). The greater occipital nerve arises from the ventral ramus of the second cervical nerve as it appears between the atlas and the lamina of the axis. The lesser occipital nerve also arises here and can be a branch of the second cervical nerve root or originate from a combination of the second and third cervical nerve root. The occipital nerves penetrate the nuchal fascia at the base of the skull and then spread cephalad.

Fig. 2 Cervical facet referral pattern

Nociceptive afferents from C1, C2, and C3 synapse in close proximity to the trigeminocervical nucleus. The trigeminocervical nucleus also receives afferents from the first division of the trigeminal nerve via the

Fig. 3 Origin of the greater occipital nerve. GON—greater occipital nerve
trigeminal nerve spinal tract [5–9]. This convergence is indicated in the referral transmission of pain originating in the cervical spine via C1, C2, and C3 to the divisions of the trigeminal nerve. Because the trigeminocephalic ganglion also interconnects the trigeminal nerve with afferents from the occipital nerve, it is possible that occipital neuralgia can refer to any of the branches of the trigeminal nerve, with a propensity for the retro-orbital region [10]. Stimulation of the greater occipital nerve will increase excitability of dural inputs to the trigeminocephalic nucleus [7].

Clinical Diagnosis

The clinical appearance of primary headache disorders, such as tension-type headache and migraine, often overlap with CH, making it difficult to accurately distinguish them from each other. The clinical distinction between primary and cervicogenic headache is difficult because tenderness over the cervical spine and neck pain are common symptoms in primary headache disorders [11]. The second edition of the International Classification of Headache Disorders recommends classifying headache with myofascial tender points in the neck as tension-type headache [12]. There is debate about whether it is possible to diagnose CH accurately based on history and physical examination alone. Observers generally agree that the two most reliable indicators of CH include pain that starts in the neck, radiating forward to the frontotemporal region of the head, and provocation of pain by neck movement [13, 14]. There is mixed opinion as to the reliability of restricted range of motion, pressure pain on palpation, and unilateral headache as indicators. The Cervicogenic Headache International study group did establish guidelines that they believe support the clinical diagnosis of CH [15]. Major criteria include precipitation of head pain by neck movement or external pressure; restricted range of motion of the neck, ipsilateral neck, shoulder, or arm; pain of a rather vague, nonradicular nature; or occasional arm pain of a radicular nature. These guidelines remain up for debate.

Once suspected that a headache is cervicogenic, it is difficult to determine which cervical level is the source of pain via history, physical exam, and imaging. The referral pattern of pain originating from the OA joint is poorly described. Pain originating from the AA joint (C1–C2) has been described to be focused in the occipital region and referred to the orbit and ears. Pain originating from the C2–C3 facet joint also is focused in the occipital region and referred through the parietal to the frontal region and orbit. Imaging frequently is incapable of providing an exact anatomic source of the pain. Magnetic resonance imaging (MRI) scans done on 24 patients with clinical features of CH were compared with 20 control patients in one study [16]. The results showed no demonstrable difference in the appearance of cervical spine structures on MRI, suggesting that the diagnosis of CH requires more than simply the presence of radiologic abnormalities. In another study of 252 patients with arthritis (peripheral osteoarthritis or degenerative disease of the spine), 27 (11%) had AA arthritis on standard x-ray, and all of these patients had occipital and neck pain. Of the 75 patients without any neck or occipital pain, none had AA arthritis [17], contributing to the hypothesis that if one has occipital headache and AA arthritis on imaging, cervicogenic headache is the likely diagnosis, with the AA joint as the pain generator.

Occipital neuralgia is characterized by pain that is isolated to sensory fields of the greater and lesser occipital nerve. Classically, the description of occipital nerve pain is a deep or burning pain with shock-like features over the occipital region. Injury to the occipital nerves can occur anywhere along their course from the cervical spinal roots to the tip of the nerve endings.

Interventional Diagnosis

The source of a suspected pain generator within the cervical spine frequently can be anesthetized via a wide array of interventional procedures that, if successful at relieving the pain, frequently support the diagnosis of CH. If AA (C1–C2) arthropathy is the suspected trigger of a cervicogenic headache, a needle can be advanced under fluoroscopic guidance to the AA joint. A local anesthetic can be delivered to test whether there is a distinct resolution of the pain after anesthetizing the joint. This also can be done for the OA joint. Based on the anatomy of the lower cervical facet joint (below C1–C2) rather than intra-articular injections, the medial branch nerves, which transmit sensory information about the joint via the nerve roots for interpretation by the brain, are anesthetized. The diagnostic utility of performing confirmatory blocks is that, if positive, they protect the patient from the futile pursuit of other and competing diagnoses, and protect them from undergoing presumptive treatment or treatments that are not appropriate [18].

With any diagnostic test, a percentage of patients may have a false-positive or false-negative response. One way to corroborate a positive response to the primary block is to perform a second, confirmatory block. The most accurate results would be obtained by a follow-up placebo injection containing normal saline or local anesthetic under double-blind conditions. However, this is not ethical because of the inherit risk of cervical injections. Thus, while there is
validity to diagnostic injections, their accuracy is not without unavoidable false-positive and false-negative responses.

The occipital nerve block is traditionally administered at the point where the greater occipital nerve crosses the superficial nuchal line. Rather than just injecting a local anesthetic, the block usually includes a mixture of corticosteroid plus local anesthetic, which makes it both diagnostic and therapeutic at the same time. The injection is characteristically performed as a nonspecific regional blockade rather than a specific nerve blockade; because there is a normal anatomic variation to the exact nerve path and no fluoroscopic landmarks, the injection must be done “blindly” to the general path of the occipital nerve. Ultrasound is now being investigated for a more targeted approach.

Treatment Options

The treatment of cervical facet arthropathy and occipital neuralgia as a source of headache is challenging. Both are best treated with a multimodal approach to pain utilizing physical therapy, medication, and injection therapy. Treatment is especially challenging when the diagnosis has been made clinically and not proven interventional because the diagnosis and identification of the source of the pain only can be definitive through interventional means.

There are no specific medications with approval from the US Food and Drug Administration or convincing evidence base for the treatment of CH resulting from pathology within the cervical facet joints. Frequently, by the time the diagnosis of CH is made, the patient usually has tried and failed multiple medications. These usually include nonsteroidal anti-inflammatory drugs (NSAIDS), antidepressants, antiepileptic drugs, muscle relaxants, and opioids. Both physical therapy and manual manipulation have been the subjects of many studies on the treatment of cervicogenic pain and seem to be of benefit. Jull et al. [19] investigated using specific exercise, manual therapy, or a combination of the two for the treatment of CH, comparing these three study groups to patients receiving nonspecific care from a general practitioner. The study showed a significant reduction in headache frequency and intensity in all three groups as compared to those receiving nonspecific care. Furthermore, it was noted that 76% of the patients in all three study groups achieved more than a 50% decrease in headache frequency, and 35% had complete relief at the 7-week follow-up period. At 1 year, 72% continued to have a 50% decrease in headache frequency. Interestingly, there were no statistically significant differences between the physical therapy group, the manual therapy group, and the combination therapy group.

One of the utilities of anesthetic injections is short-term pain relief, which allows the patient to more fully participate in physical therapy, ultimately resulting in a better outcome. It allows for a break in the pain cycle. Numerous anecdotal reports have identified injections of cortisone into the OA, AA, or C2-C3 joint as being effective in the treatment of CH, but this hypothesis has not been tested in controlled trials and is not a proven treatment. Radiofrequency ablation (using heat to cauterize painful nerves) is an option, but is a risky procedure in this instance, with so many vital anatomic structures in such a small space. In Freund and Schwartz’s study [20], Botulinum toxin injections were shown to be beneficial for patients with chronic headache after a whiplash injury. In this study, patients who received Botox injections at five cervical trigger points showed significant improvement in pain and range of motion at 4 weeks follow-up as compared to patients who received placebo (saline injections) at the same points. These patients receiving Botox had no preinjection imaging, no anesthetic blocks, and could not be unequivocally differentiated from tension-type headache, making the preprocedure diagnosis of CH unclear. Surgical options for CH exist as well; if the pain is proven to stem from a particular facet joint, it can be fused with its adjacent joint (arthrodesis) [21, 22]. The fusion of painful facet joints is a common procedure for spine surgeons. A multimodal approach to CH comprised of biofeedback, relaxation, and cognitive-behavioral therapy is important in the comprehensive management of pain [23].

The occipital nerve is classically blocked at the nuchal line with a combination of a steroid and local anesthetic. It is thought that occipital nerve blocks have a neuromodulatory effect on pain transmission. In one study, 94% of patients obtained pain relief with occipital nerve blockade containing a steroid and local anesthetic, but only for a duration of 10 to 77 days [24]. The study included patients suffering from CH of multiple etiologies. More aggressive treatments have included the surgical release of the occipital nerve on the theory that the nerve is entrapped. Occipital nerve release has not been shown to provide long-lasting effective relief [5], perhaps because in some cases the origin of the occipital nerve, the C2 spinal nerve, is the root cause. One surgical study selected patients who had CH, treated with positive blockade of the C2 spinal nerve. Patients then underwent surgical decompression and microsurgical neurolysis of the C2 spinal nerve. Upon follow-up at 16 months, 14 of 31 patients were pain free [25]. With the evolution of pain management, some reports describing spinal cord stimulator technology for CH from occipital neuralgia have entered the literature. Rather than placing
the cylindrical electrodes in the epidural space, they are placed under the skin next to the occipital nerve. Subcutaneous cylindrical electrodes are placed next to the distal branches of the C1–C2–C3 spinal nerves as they converge to create the occipital nerve. By using controlled low-dose electricity, it is suspected there is a neuromodulating effect on the occipital nerve. Positron emission tomography (PET) imaging studies after occipital nerve stimulation (ONS) have shown modulation of activity in the thalamus and anterior cingulated cortex, suggesting that ONS may have a central role to play here [26**, 27*]. In one study of 13 patients, 66% achieved greater than 75% pain relief and 33% achieved 50% to 75% pain relief [28]. Like most forms of pain, a multimodal approach encompassing physical therapy and medications is most effective.

Conclusions

Cervical facet arthropathy and occipital neuralgia are two common causes of CH. Both are frequently responsible for pain in the occipital region. However, because of their close anatomic relationship to the trigeminocephalalgic nucleus, pain may be referred to any branch of the trigeminal nerve, resulting in clinical presentations similar to tension-type headache or migraine.

Diagnosis and treatment of cervical facet joint arthropathy as a cause of CH are difficult, and a reliable set of diagnostic criteria remains elusive. Although cervical facet arthropathy may occur at any age, the number of patients with wear-and-tear cervical pathology will increase with the baby boomers reaching their later years. There is great debate regarding the clinical accuracy of the CH diagnosis without the support of a diagnostic injection. Diagnostic injections are not without risk and are not without rates of false-positive and false-negative response. No single treatment of CH has emerged as the gold standard. At this time, best practice is a multimodal approach that includes physical therapy or manual therapy as the focal point.

The upper cervical nerve roots that innervate the cervical facet joints also are the origin of the greater and lesser occipital nerves. Occipital neuralgia can be caused by pathology anywhere along its course. Occipital nerve blocks are typically both diagnostic and therapeutic, but do not confirm the cause of occipital nerve pathology. The injections may be short lived, but newer pain management techniques with occipital nerve neuromodulation show promise.

Disclosures No conflicts of interest relevant to this article were reported.

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