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Tracking patients with chronic occipital headache after occipital nerve decompression surgery: A case series

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Abstract

Background: The therapeutic benefit of nerve decompression surgeries for chronic headache/migraine are controversial.

Aim: To provide clinical characteristics of headache type and treatment outcome of occipital nerve decompression surgery.

Methods: A retrospective review of clinical records. Inclusion criteria were evidence of chronic occipital headache with and without migrainous features and tenderness of neck muscles, occipital allodynia, and inadequate response to prophylactic drugs.

Results: Surgical decompression of the greater and lesser occipital nerves provided complete and extended (3–6 years) relief of new daily persistent headache in case 3 (46 year old female), and of chronic post-traumatic headache in cases 4 and 6 (35 and 30 year old females, respectively), partial relief of chronic headache/migraine in cases 1 and 2 (41 year old female and 36 year old male), and no relief of episodic (cases 3 and 4) or chronic migraine (case 5, 52 year old male), or chronic tension-type headache (case 7, 31 year old male).

Conclusions: As a case series, this study cannot test a hypothesis or determine cause and effect. However, the complete elimination of new daily persistent headache and post-traumatic headache, and the partial elimination of chronic headache/migraine in two patients – all refractory to other treatment approaches – supports and justifies the effort to continue to generate data that can help determine whether decompression nerve surgeries are beneficial in the treatment of certain types of chronic headache.

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Keywords

Chronic migraine; occipital allodynia; inflammation; muscle tenderness; pain; trigeminal

Introduction

Migraine and non-migraine headaches are commonly associated with tenderness of pericranial and neck muscle and soft tissue (1–5). When the muscle tenderness appears after the onset of migraine, it is considered an associated symptom that is secondary to the development of sensitization in central trigeminovascular neurons that process sensory information from the intracranial meninges and pericranial and neck muscles (6,7). Such neurons reside in the upper cervical spinal cord. In contrast, when the muscle tenderness is chronic or appears before the onset of migraine, it is considered primary and postulated to originate in activation of pericranial nociceptors (8,9). The receptive fields of such nociceptors may include the muscles themselves, the tendons that attach the pericranial muscles to the bones of the calvaria, and the periosteum. The periosteum is the part of the calvarial bone to which muscles are attached.

In the past 50 years, nearly all studies on the pathophysiology of migraine have focused on its brain origin and nearly all studies on the pathophysiology of the associated headaches have focused on the role played by activation of nociceptors that innervate the meningeal sinuses and the dura mater (10). Consequently, much knowledge exists to support both notions. In contrast, only a small number of studies had attempted to understand the pathophysiology of migraines that could originate extracranially, and virtually no study had attempted to understand the role played by pericranial nociceptors in such headaches (10). We attribute the latter mainly to lack of an appropriate animal model. Regardless of the reason, in the absence of data, it is easy to refute the possibility that some migraines may originate extracranially. It is also easy to adhere to the belief that muscle tenderness is a symptom rather than a cause.

To date, lack of data is the major weakness in the hypothesis that in some headaches, tenderness of pericranial and neck muscles may be the cause rather than the symptom. In keeping faith with the view that, like all hypotheses, this one should be accepted or refuted by data rather than opinions, we have initiated a series of studies in which attempts are made to generate new knowledge on physiological, cellular, molecular and genetic events and processes which occur in pericranial tissues that are chronically tender in a subset of migraine patients. In the first of such studies, we analyzed discarded periosteum tissues of CM patients whose pericranial and neck muscles were chronically tender and found that in comparison to subjects who are not migraine patients, their periosteum expressed a significantly larger number of genes that promote inflammation and significantly smaller number of genes that suppress inflammation (9). Because the up-regulated proinflammatory genes were linked to activation of white blood cells, production of cytokines, and inhibition of NF κ B activation, whereas the down-regulated anti-inflammatory genes were linked to repression of macrophage activation and prevention of cell lysis, we interpret the findings as suggesting that the molecular environment in which periosteal pain fibers exist is inflamed

and that this localized inflammation can irritate, activate, or sensitize trigeminovascular nociceptors that reach the affected periosteum through somatic branches of the occipital nerve as well as through suture branches of intracranial meningeal nociceptors (8,9). While the findings of the periosteum biopsies cannot be considered sufficient evidence to prove or disprove a hypothesis, they constitute a first step along this novel path.

The analyzed tissues were obtained during occipital nerve decompression surgeries performed between 2011 and 2013 under the premise that such procedures can ease disease burden by reducing migraine and headache days per month in chronic migraine patients who were unresponsive to any other form of treatment. In the current study, we examined the effects that these surgeries had on selected patients whose biopsies we analyzed – those whose medical records were reliable and available to us for at least 3 years post-operatively.

The purpose of this case report is to share our clinical experience with the success and failure of occipital nerve decompression surgeries for the relief of chronic headache with and without migrainous features using “real-life” documentations from follow up visits. The seven cases we chose for this report represent the challenges and the promises of this controversial approach. While each of the seven patients whose data are included in this paper provided informed consent for both the surgery and the record review, the information presented here was obtained from a retrospective review of medical records. As such, this report is not a prospective study and the strength of the data and the conclusions must be judged accordingly.

Results

All seven patients whose cases are summarized below fulfilled the following criteria for nerve decompression surgery: a) daily or almost daily pain in the distribution of the occipital nerves (suboccipital neck, the occiput, and frontal radiation to retro-orbital area, temples, forehead); b) some degree of occipital allodynia; c) spasm and discomfort of neck and shoulder muscles; d) aggravation of pain with engagement of cervical and shoulder muscles; e) tenderness on occiput; f) pain present for more than 6 months; g) lack of adequate response to preventive medications (other than Botox); h) pain resulting in disability.

Nerve decompression surgeries are conducted under general endotracheal anesthesia, with the patient placed in the prone position with careful padding to support the head and protect the face and eyes. A midline vertical occipital incision of approximately 5 cm length is made, followed by dissection of underlying soft tissue using electrocautery. Once the dorsal fascia is identified and dissected, micro-tip bipolar cautery is used to expose branches of the dorsal and greater occipital nerves and the semispinalis capitis muscles, bilaterally. The nerves are then decompressed throughout their length by removal of surrounding muscle and/or fascia tissues. Branches of the greater occipital nerves commonly travel through the semispinalis capitis muscles. A partial resection of the semispinalis capitis muscle is thus performed. For the decompression of the lesser occipital nerve, a 3 cm horizontal incision is made over the mastoid area on each side of the head, and branches of the lesser occipital nerves are released from the fascia attachments of the nuchal musculature, where they usually seem constricted. At the end of these procedures, a pain catheter is placed in the

operative field to provide continuous local anesthesia for five days following surgery. A drain is placed in the base of the midline wound and is brought out through a separate stab incision. The entire surgical procedure generally requires four to five hours, depending on the number of nerve branches, the degree of compressive tissue, and the BMI of the patient. All nerve decompression surgeries were performed by the same surgeon.

Case 1: Partial response in a chronic migraine patient

A 41-year old woman with a history of chronic migraine for 7 years presented in September 2012. The onset of migraine was not associated with any inciting event and she had no family history of headache. The headaches were episodic at first and had gradually increased in frequency until they became almost daily in 2011. These headaches were described as bilateral vice-like pressure pain that started in the suboccipital area, where the pain was constant and daily, and radiated to the occiput and temple on one side of the head on 4–6 days a week. When the headaches radiated to the occiput and temple, they were mostly unilateral and accompanied by photophobia, phonophobia, nausea, vomiting, dizziness, tingling and stabbing (thus fulfilling migraine criteria of > 15 days/month). There was marked occipital allodynia, such that occipital pain would cause night-time awakening, leading the patient to have to get out of bed, remove her head from the pillow, and spend the rest of the night in a chair, avoiding contact with the occiput. Physical examination revealed marked tenderness on the right and left occiput. Previous evaluations by neurologists indicated chronic migraine. Preventive medications including anticonvulsants (AED) were mildly effective, and the patient was on topiramate and levetiracetam with unclear benefit. OnabotulinumtoxinA had been administered twice according to the CM protocol of 155 units in 31 locations with no significant reduction in migraine days. Anxiety was ultimately diagnosed and successfully treated with escitalopram and prn hydroxyzine. Occipital nerve blocks (attempted three times) resulted in reduction of pain severity by about 50% for less than 2 weeks. Nerve decompression surgery was performed in December 2012. In 2013, there was a gradual reduction in the constant suboccipital pain and the frequency of the radiating migraines. The remaining migraine attacks had the tendency to occur on days in which her anxiety was high. By January 2015, the patient reported nearly 90% reduction in her previously constant suboccipital pain. In contrast, her occipital/temple migraines continued to occur about 15 days a month, usually associated with stress, photophobia, nausea and dizziness. OnabotulinumtoxinA injections (155 units per CM protocol) were administered for CM beginning May 2015. Although onabotulinumtoxinA injections had been ineffective prior to surgery, following surgery they were highly effective, reducing the occipital/temple migraine days from 15 to 2.5 days a month.

Outcome: In March 2018, 5.5 years after her surgery, her constant suboccipital headache and allodynia were reduced by 90%, and onabotulinumtoxinA injections, which had been ineffective in reducing migraine days prior to surgery, became effective in reducing frontal headaches following surgery.

Case 2: Partial response in a chronic migraine patient

A 36-year old man presented in October 2011 with a 17-year history of headaches that had been episodic at first but gradually increased in frequency and had been daily since 2001.

There was no inciting event for the onset of headaches. The pain was mild-moderate in intensity and described as pressure and throbbing. It involved the right occiput and trapezius muscle, with occasional radiation to the front. There was moderate tenderness and allodynia in the occipital region, and pain level increased with resting the back of the head on the pillow, to the extent that he would often arise from bed during the night. Associated symptoms included photophobia, phonophobia, nausea and rare vomiting; about eight days a month were migraine days. Physical and neurological examination were normal, albeit with a medical history of depression. Preventive medications including AEDs, TCA and Ca channel blockers were ineffective. Occipital nerve blocks reduced the pain insignificantly for up to 2 days. Occipital nerve decompression was performed in April 2013. The daily head and neck pain gradually reduced, and by January 2014, headache frequency was significantly reduced, while neck muscle tightness and spasm continued to occur at varying frequency. Daily occipital pain stopped by 2015. Depression worsened over the years in relation to life events, and episodic frontal headaches with photophobia could occur at varying frequencies in response to stress triggers. Zonisamide was started for episodic migraine prevention and sumatriptan was effective for acute episodes. Psychiatric care and psychotherapy have been consistently recommended and sporadically undertaken for depression and anxiety, which had varying effects on headaches. Foam rollers have been used with benefit for the trapezius muscle spasm.

Outcome: 3.5 years following surgery (most recent contact in November 2016), occipital pain remained absent and frontal headache frequency was 3–4 times a month on zonisamide.

Case 3: Complete elimination of new daily persistent headaches (NDPH) with no effect on low-frequency episodic migraine

A 46-year old woman presented in October 2011 with eight years of low-frequency episodic migraine and seven years of NDPH that did not fulfil migraine criteria. The NDPHs involved the left occiput, radiated to the left retro-orbital area, were of moderate intensity and were not associated with common migraine symptoms. The low-frequency episodic migraines were bilateral, frontal headache accompanied by photophobia, phonophobia, osmophobia, nausea and vomiting. The NDPH was more problematic for the patient as it occurred daily and was felt as burning behind the left eye and tightness in the occiput, suboccipital neck and trapezius muscle (which increased by physical activity). Physical examination revealed tenderness on the bilateral occiput overlying the lesser and greater occipital nerves, more on the left than on the right. Vital signs and neurological examination were normal. Preventives including AEDs, tricyclic antidepressant (TCA), and verapamil were all ineffective. Left occipital nerve blocks provided significant relief for the NDPH, which lasted for only 2 weeks. Past medical history was positive for fibromyalgia, which did not limit function. Nerve decompression surgery was conducted in December 2011. By January 2012, the NDPH were reduced in frequency and intensity and occurred three days a week. By March 2012, NDPHs were completely resolved and she resumed normal physical activities. By January 2013, one year post surgery, low-frequency episodic migraine (which occurred twice a month) remained unchanged.

Outcome: Six years following occipital nerve decompression, all NDPHs were gone whereas low-frequency episodic migraines continue to occur two times per month.

Case 4: Complete elimination of chronic post-traumatic headache fulfilling migraine criteria, with no effect on low-frequency episodic migraine

A 35-year old woman with a history of low-frequency episodic migraine (occurring 6 times a year, responding to rizatriptan, affecting right or left temple, and pulsating in character) presented in October 2012 with a 10-month history of daily headaches that started following a head injury she sustained in a car crash in which her head struck the passenger window (CT scanning revealed an occipital lobe hemorrhage which cleared without surgical intervention). These post-traumatic headaches, which started a few weeks after the injury and remained unchanged since, fulfilled migraine criteria; they were moderate to severe in intensity and associated with daily photophobia, and occasional phonophobia and nausea. At the time of the initial evaluation, the headaches were daily and lasted 4–12 hours. Her post-traumatic headaches started in the occiput, felt as if someone was pressing a thumb against the back of her head, radiated to the temples, and were unresponsive to rizatriptan. In general, they were more painful on the right than on the left side. Along this line, there was daily tightness in the right suboccipital cervical muscles and the shoulder portion of the right trapezius and occipital allodynia necessitated sleeping on the left side of the head. It often awakened her at night, requiring her to leave the bed. The headaches prevented the patient from participating in her competitive horseback riding combined with shooting, as the muscular activity involved would increase her headache severity. Physical examination revealed significant tenderness over the lesser and greater occipital nerves. Preventive medications including AEDs, TCA and Lyrica were ineffective. Occipital nerve blocks provided significant relief for 3–4 months. Occipital nerve decompression surgery was performed in January 2013. Nine months after surgery her daily post-traumatic headaches were reduced to 8–12 a month. In mid-2015, two and a half years after the surgery, her post-traumatic headaches were completely resolved whereas her low-frequency episodic migraine remained unchanged, occurring twice a month.

Outcome: By August 2017, 4.5 years after nerve decompression surgery, all post-traumatic headaches (all occipital, with pressure in the back of the head and radiating forward) were resolved. Complete elimination of occipital allodynia and muscle tightness allow her to return to competitive horseback riding and sleep comfortably on both sides of her head. On the other hand, her low-frequency migraine attacks, affecting mainly the right or left temple areas and responsive to rizatriptan, were still present.

Case 5: No response in a chronic migraine patient

A 52-year old man presented in May 2012 with a history of headaches that had been present for 22 years, with family history of migraine and no inciting events. The frequency had gradually increased over the years and they had become daily several years earlier. Thirty days of the month fulfilled migraine criteria. The daily headache usually began in the sub-occipital and occipital areas and radiated to both temples. The pain was a constant, dull ache, described as “like someone is trying to push a softball out of my head,” and was rated as 7/10 on a visual analogue scale. There was constant tightness of cervical muscles and

turning the head to the side would increase his pain. There was mild occipital allodynia, as pain would awaken him at night for which he would arise from bed specifically to remove his head from the pillow, and he could also have pain from resting the occiput against the back of a chair. Physical examination revealed marked tenderness on the occiput at the lesser and greater occipital nerves. Preventive medications including AED and BP (metoprolol, lisinopril, amlodipine) agents were ineffective. Occipital nerve blocks as well as cervical muscle trigger point injections eliminated the headache briefly (up to 2 days); after which the headaches gradually increased to pre-nerve block level within 2 weeks. A peripheral neurostimulator trial had not been effective, so a permanent device was not placed. Occipital nerve decompression surgery was performed in June 2012. At subsequent visits, there was no significant reduction in pain, although the episodes of most severe headaches reduced in frequency from several times a month to once a month.

Outcome: In 2018, 5.5 years after surgery, there was no change in daily occipital pain radiating to the temples.

Case 6: Complete response in a patient with post-traumatic occipital headache

A 30-year old woman with family history of migraine and occasional frontal headaches was presented in September 2011 with a history of occipital headaches that had been severe and daily for 10 years, following a fall in which she struck the back of her head against a brick wall. The onset of occipital head pain and pain in the posterior neck was immediate, and it had never subsided. These occipital headaches ranged from moderate to severe in intensity and felt like a vice tightening around the back of her head. The pain in the neck also felt like a vice grip, not muscular tightness. There was minimal occipital allodynia or nausea and no photophobia or phonophobia; 30 days a month were headache days, not migraine. Physical examination revealed moderate-marked tenderness on the bilateral lesser and greater occipital nerves. Trials of preventive medications including AEDs and antidepressants had been ineffective. An occipital nerve block worsened the headache for two days. Occipital nerve decompression was performed on December 10, 2011. The occipital headaches diminished rapidly post-surgically, and by six months they were all but gone. Occasional frontal headaches that were responsive to OTC analgesics continued to occur, as they had prior to the head injury. At the last contact in January 2015, three years following surgery, the response was maintained, with no return of occipital headaches and occasional mild frontal headaches.

Outcome: complete elimination of daily occipital headache and no change in occasional frontal headaches that responded to OTC analgesics.

Case 7 – Reduced opioid consumption but no elimination of chronic tension type headache (CTTH)

A 31-year old man presented in November 2011 with chronic tension-type headache with pericranial tenderness for the past 12 years. These TTHs, which gradually become more painful over the years, were daily, constant, starting in the occiput and radiating to the vertex. They were described as moderate tightness and pressure in the suboccipital area and squeezing pain in the occipital and vertex regions. There was mild-moderate allodynia, but

no nausea, photophobia, or any migraine associated symptom. Occipital nerve blocks were either minimally helpful for one day, or not helpful at all. A trial of peripheral nerve stimulation was completed and did not provide benefit; the permanent device was not placed. Numerous preventives including AEDs and antidepressants were not helpful. As the patient was managed with opiate analgesics with a dose of 90 morphine milligram equivalents (MME) prior to surgery, he was admitted to the hospital and an opioid analgesia withdrawal protocol was administered. The patient remained off the opioid medications for one month, during which his CTTH got much worse, he was unable to work and consequently went back on the opioids. Following the opioid withdrawal failure, occipital nerve decompression was performed in December 2011. On March 2012, after seeing only mild benefit from the surgery and consequently continuing to use high doses of opioids, the patient was admitted again for discontinuation of opiates. In December 2012, after being opioid free for 9 months, the intensity but not frequency of his CTTH decreased significantly. In June 2013, still opioid free, the intensity of his headaches as well as his functionality continue to improve. In September 2014, his headache intensity increased again, and he was put on tramadol (10–20 MME) by a pain management physician. In July 2017, the patient agreed to take a psychological evaluation that revealed a diagnosis of Somatic Symptom Disorder. In February 2018, while continuously taking tramadol (20 MME), the patient continues to report daily headache of moderate to severe intensities. While on opioids, his level of function is good, he is working full time in a position that requires international travel and is an involved father of two children.

Outcome: While the surgery was not beneficial for the CTTH, it led to a reduction in opioid use, from 90 MME before surgery to 20 MME after surgery. Due to the complexity of the case, we cannot comment with certainty on whether the mild reduction in headache intensity is driven by the reduced opioid consumption or the surgery itself. This case also raises the possibility that comorbid Somatic Symptom Disorder may play a key role in the ongoing TTH.

Discussion

Surgical decompression of the greater and lesser occipital nerves provided complete and extended (3–6 years) relief of NDPH in case 3 (46 year old female), and of chronic PTH in cases 4 and 6 (35 and 30 year old females, respectively), partial relief of CM in cases 1 and 2 (41 year old female and 36 year old male), and no relief of episodic (cases 3 and 4) or chronic migraine (case 5, 52 year old male), or chronic TTH (case 7, 31 year old male).

Given the many limitations of the presented case reports, we acknowledge that the results do not allow us to draw any conclusions about the effectiveness of the described occipital nerve surgeries for the treatment of chronic and episodic migraine as well as PTH, TTH and NDPH, especially since we do not have a (control) group of patients from which we may be able to determine if in some of these patients the reduction or elimination of the headache would have occurred on its own over time. It also does not allow us to identify the symptoms or procedures that can help us identify responders and non-responders. However, given that in three cases the daily headaches were resolved after the nerve decompression surgery,

efforts to generate more data that can help us prove or disprove the efficacy of nerve decompression surgery for daily headache seems justified.

While it may be premature to discuss, we noted that the surgical decompression procedure provided complete elimination of NDPH and PTH but not episodic or chronic migraine, or chronic TTH. Theoretically, one can use these results to support the notion that the pathophysiology of migraine differs from the pathophysiology of PTH or NDPH. More specifically, one can speculate on the possibility that the pathophysiology of PTH and NDPH does not trigger the long-term changes in the excitability of neurons in the sensory cortices and along the trigeminovascular pathways (i.e. in the thalamus and spinal trigeminal nucleus) – one of the hallmarks of migraine. Alternatively, it is also reasonable to suggest that continuous input from affected peripheral nerves and their nociceptors play more important role in PTH and NDPH (where the activity of the central neurons depends on the peripheral drive) than in migraine (where the activity, hyper-responsiveness and hyper-sensitivity of the central neurons may become independent of the peripheral drive). Finally, while we have no data to try to explain the slow pace at which patients improve after surgery, we speculate that it may take time for the peripheral nerves to heal and for the central neurons to recover from their chronic activation state.

Perspective

Decompression surgeries are intended to relieve symptoms caused by pressure on, or compression of, nerve fibers and their roots, most commonly in the spinal cord where bulging or collapsed disks, thickened joints, loosened ligaments, and bony growths can narrow the spinal canal and the spinal nerve foramen (11). These pathologies often lead to the development of symptomatic degenerative spondylosis (spinal stenosis), low back pain and sciatica – pain conditions commonly manifested as deep muscle pain and associated with numbness, tingling, and weakness in corresponding distributions (11). Mechanistically, these pains are thought to be neuropathic and inflammatory; to involve joints, ligaments, periosteum, blood vessels and paravertebral muscles; to be worsened by extension; and to be associated with spasticity (12–14).

Somewhat similarly, the paravertebral muscle pain features of spinal pathologies may also be present in migraine patients whose headaches involve the back of the head and chronic tenderness of neck muscles. This symptomatic association gave rise to the notion that compressed nerves may be the underlying pathophysiology of the headache in those patients whose migraine attacks begin with muscle tenderness and affect the occipital area first.

The number of spinal cord or nerve root decompression surgeries performed annually around the world continues to increase (15). In spite of the growing popularity of these surgeries, the surgical management of spinal stenosis and narrowing of the spinal nerve foramen remains controversial due to lack of controlled trials demonstrating superior effectiveness in comparison to nonsurgical approaches (16,17). Nevertheless, because treatments are tailored to the individual patient, and because some patients prefer the surgical over a nonsurgical approach, it is likely that physicians will continue to perform decompression surgeries for years to come.

Adapting the nerve decompression surgical approach, a group of plastic surgeons have been reporting for almost 20 years that decompression of branches of sensory nerves of the calvaria, including branches of the occipital nerve, reduce migraine and non-migraine headaches in patients whose headaches are tightly associated with chronic tenderness of neck muscles (18–21). Before then, much of the rationale for targeting a treatment approach to the occipital nerve was borne in reports on therapeutic effects of occipital nerve blocks. In fact, two meta-analyses that assessed the efficacy of greater occipital nerve block versus placebo for the treatment of migraine showed that, compared with control intervention, GON blocks reduced pain intensity significantly more (22,23).

The controversy over decompression spinal surgeries for back pain and sciatica did not spare the upper cervical spine-derived nerve decompression surgeries for occipital headache and migraine (24). In an email sent to members of the American Headache Society (AHS) in 2013, the AHS issued a statement that urged “patients, healthcare professionals and migraine treatment specialists themselves, to exercise caution in recommending or seeking such therapy”. This statement was later adopted by the American Board of Internal Medicine Foundation’s Choosing Wisely Task Force (25). But as with all debates and controversies in medicine, the ongoing discussion on whether or not nerve decompression approaches are effective for migraine pain treatment must be supported exclusively by data, rather than notions or theoretical rationalizations. The data presented in this series of case reports justify efforts to continue and pursue the effectiveness (or lack thereof) of this treatment approach.

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References

1. Jensen R, Rasmussen BK, Pedersen B, et al. Cephalic muscle tenderness and pressure pain threshold in a general population. *Pain* 1992; 48: 197–203. [PubMed: 1589238]
2. Jensen R, Rasmussen BK, Pedersen B, et al. Muscle tenderness and pressure pain thresholds in headache. A population study. *Pain* 1993; 52: 193–199. [PubMed: 8455967]
3. Tfelt-Hansen P, Lous I and Olesen J. Prevalence and significance of muscle tenderness during common migraine attacks. *Headache* 1981; 21: 49–54. [PubMed: 7239900]
4. Selby G and Lance JW. Observations on 500 cases of migraine and allied vascular headache. *J Neurol Neurosurg Psychiatr* 1960; 23: 23–32. [PubMed: 14444681]
5. Aaseth K, Grande RB, Lundqvist C, et al. Pericranial tenderness in chronic tension-type headache: The Akershus population-based study of chronic headache. *J Headache Pain* 2014; 15: 58. [PubMed: 25193401]
6. Burstein R Deconstructing migraine headache into peripheral and central sensitization. *Pain* 2001; 89: 107–110. [PubMed: 11166465]
7. Nosedá R and Burstein R. Migraine pathophysiology: Anatomy of the trigeminovascular pathway and associated neurological symptoms, cortical spreading depression, sensitization, and modulation of pain. *Pain* 2013; 154(Suppl 1): S44–S53. [PubMed: 23891892]
8. Burstein R, Blake P, Schain A, et al. Extracranial origin of headache. *Curr Opin Neurol* 2017; 30: 263–271. [PubMed: 28248698]
9. Perry CJ, Blake P, Buettner C, et al. Upregulation of inflammatory gene transcripts in periosteum of chronic migraineurs: Implications for extracranial origin of headache. *Ann Neurol* 2016; 79: 1000–1013. [PubMed: 27091721]

10. Olesen J, Burstein R, Ashina M, et al. Origin of pain in migraine: Evidence for peripheral sensitisation. *Lancet Neurol* 2009; 8: 679–690. [PubMed: 19539239]
11. de Campos TF. Low back pain and sciatica in over 16s: Assessment and management NICE Guideline [NG59]. *J Physiother* 2017; 63: 120. [PubMed: 28325480]
12. Weisman MH. Inflammatory back pain: The United States perspective. *Rheum Dis Clin North Am* 2012; 38: 501–512. [PubMed: 23083751]
13. Vardeh D, Mannion RJ and Woolf CJ. Toward a mechanism-based approach to pain diagnosis. *J Pain* 2016; 17: T50–T69. [PubMed: 27586831]
14. Baron R, Binder A, Attal N, et al. Neuropathic low back pain in clinical practice. *Eur J Pain* (London, England) 2016; 20: 861–873.
15. Cherkin DC, Deyo RA, Loeser JD, et al. An international comparison of back surgery rates. *Spine* 1994; 19: 1201–1206. [PubMed: 8073310]
16. Weinstein JN, Lurie JD, Tosteson TD, et al. Surgical versus nonsurgical treatment for lumbar degenerative spondylolisthesis. *N Engl J Med* 2007; 356: 2257–2270. [PubMed: 17538085]
17. Zaina F, Tomkins-Lane C, Carragee E, et al. Surgical versus non-surgical treatment for lumbar spinal stenosis. *Cochrane Database Syst Rev* 2016; 29: CD010264. doi:10.1002/14651858.CD010264.pub2.
18. Lee M, Brown M, Chepla K, et al. An anatomical study of the lesser occipital nerve and its potential compression points: Implications for surgical treatment of migraine headaches. *Plast Reconstr Surg* 2013; 132: 1551–1556. [PubMed: 24005368]
19. Kurlander DE, Punjabi A, Liu MT, et al. In-depth review of symptoms, triggers, and treatment of temporal migraine headaches (Site II). *Plast Reconstr Surg* 2014; 133: 897–903. [PubMed: 24675192]
20. Kurlander DE, Ascha M, Sattar A, et al. In-depth review of symptoms, triggers, and surgical deactivation of frontal migraine headaches (Site I). *Plast Reconstr Surg* 2016; 138: 681–688. [PubMed: 27556607]
21. Ascha M, Kurlander DE, Sattar A, et al. In-depth review of symptoms, triggers, and treatment of occipital migraine headaches (Site IV). *Plast Reconstr Surg* 2017; 139: 1333e–1342e.
22. Tang Y, Kang J, Zhang Y, et al. Influence of greater occipital nerve block on pain severity in migraine patients: A systematic review and meta-analysis. *Am J Emerg Med* 2017; 35: 1750–1754. [PubMed: 28844531]
23. Zhang H, Yang X, Lin Y, et al. The efficacy of greater occipital nerve block for the treatment of migraine: A systematic review and meta-analysis. *Clin Neurol Neurosurg* 2018; 165: 129–133. [PubMed: 29421172]
24. McGeeney BE. Migraine trigger site surgery is all placebo: A response. *Headache* 2016; 56: 779–781. [PubMed: 27092536]
25. Loder E, Weizenbaum E, Frishberg B, et al.; American Headache Society Choosing Wisely Task Force. Choosing wisely in headache medicine: The American Headache Society's list of five things physicians and patients should question. *Headache* 2013; 53: 1651–1659. [PubMed: 24266337]

Clinical implications

- The ongoing controversy over the role of nerve decompression surgery for the treatment of chronic headache is based on opinions, rather than data.
- The presented case series justifies an effort to continue to generate data that may help us determine whether or not these procedures decrease the burden of headache in selected patients.
- The origin of chronic occipital headache may differ from the one driving chronic migraine.