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Occipital Neuralgia as a Sequela of Sports Concussion: A Case Series and Review of the Literature

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Introduction

Sports and recreation-related concussions are common, with an annual incidence in the United States estimated to be between 1.6 and 3.8 million (19,20,22). The majority of these injuries are considered to be self-limited; however prolonged symptoms of greater than 3 months are not uncommon (5,10,25,27). In particular, delayed headache is associated with delayed recovery from concussion and increased risk of postconcussion syndrome (3,8,10).

The pathophysiology of postconcussive headache is likely complex and multifactorial, involving both local injury as well as the activation of peripheral and central pain pathways. Occipital neuralgia (ON) is a known etiology of headaches and may stem from trauma to the neck, such as with a concussion or whiplash injury (23). ON also may feature symptoms such as nausea, dizziness, and photosensitivity that are associated commonly with concussion. Despite these features, ON may be an underappreciated cause of headache in sports-related concussion; a PubMed search returned only one reference when using the terms “sports” and “occipital neuralgia” (26).

Due to the prominence of headache as a symptom in concussion, the potential for concomitant conditions such as ON arising from the same traumatic injury, and the overlapping symptomatology between these conditions, it is important that clinicians be able to recognize ON in the management of concussion. In this case series, we will report on our observation of ON as a significant factor in the postconcussion headaches and symptomatology of three patients. We also will present a brief review of the available

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literature on ON, with attention to its relevant anatomy, presentation, examination, and treatment.

Cases

Case #1

A 16-year-old female volleyball player was diving for a ball when she hit the right side of her head on the floor. There were no transient neurological symptoms or cervical or occipital pain. The next morning, she awoke with severe headache at the back of her head. A Sports Concussion Assessment Test (SCAT) only revealed one symptom (headache). Neuropsychological computerized testing demonstrated excellent performance in all categories; however immediately thereafter, the patient developed severe headache and nausea. At 12 d after the injury, the patient was reassessed and reported a headache with severity of 1 out of 6 localized at the right posterior aspect of her head as her only symptom. Physical examination demonstrated decreased range of motion and tenderness throughout the cervical spine, most prominently at the right greater than left occiput with reproduction of her headache upon palpation. Trigger point injections with 1% lidocaine were performed at eight locations over the bilateral superior trapezius and achieved 50% reduction of her headache for 2 h. This prompted a referral to an interventional anesthesiologist for a diagnostic block of the right greater occipital nerve (GON) and lesser occipital nerve (LON), after which the patient expressed complete relief of all symptoms for 6 h. The patient then was prescribed a multimodal approach to symptom management, including massage therapy, myofascial release, and a right-sided pulsed radiofrequency stimulation of the GON and LON. The patient achieved temporary resolution of her headaches shortly following this last procedure at 166 d after the concussion. While her headaches significantly improved at rest and without rotational movement, any rotational movement during exertion creates headaches that impede participation in sports. Thus the patient has decided to voluntarily retire from volleyball.

Case #2

A 28-year-old professional baseball player sustained a concussion from a collision at home plate. The patient complained of anterograde amnesia and feeling significant mental foginess. After essentially maintaining bed rest for 2 d, he experienced severe cervical pain and a bout of emesis during ambulation. Subsequent computed tomography (CT) imaging of the cervical spine was normal. On examination, his cervical range of motion was limited to the left more than the right and his neck pain and limited range of motion gradually resolved over 2 months. Despite this, he was unable to resume any physical activity due to the development of a severe sharp, stabbing, and throbbing headache located in the left occiput. Triggers included the resumption of any athletic activity, but he found that he could avoid triggering headaches by looking straight ahead. A follow-up physical examination was significant for tenderness in the left occiput at the craniocervical junction. He was diagnosed with ON and was referred to interventional pain management for a left cervical medial branch block of C2 to C3 and third occipital nerve (TON) blocks. These procedures provided 100% pain relief and greatly increased cervical range of motion. He was then able to complete a graded return to play protocol successfully and resumed baseball.

Case #3

A 17-year-old female soccer player was hit in her forehead during a challenged header and experienced immediate symptoms, most prominently including headache, fogginess, dizziness, and photophobia. No neck pain was noted on the initial physical examination. Her initial SCAT2 symptom number was 17 (which included headache) with a symptom severity score of 67. Several trials of medications including acetaminophen, ibuprofen, amitriptyline, and zonisamide failed to reduce her headache symptoms. Five weeks after the concussion, she developed neck pain, and on repeat examination, she had a positive Tinel sign at the left craniocervical junction with significant worsening of her headache. She was referred to interventional pain management for a GON and LON block. During a phone follow-up, she reported 100% relief of her headache and neck pain for 3 d and noted significant improvement of her other concussion symptoms as well. She then received a pulsed radiofrequency stimulation of the left GON and LON, which resulted in 50% relief from her headache and neck pain for 3 wk. Due to the partial and short duration of the effect, a nerve block was then performed on the C2 and C3 medial branches and TON. The patient received 100% relief of her headache and neck pain for 5 d, again with concomitant improvement of her other symptoms. Given this response, a radiofrequency denervation of these same nerves was performed. She received complete relief of her headache and neck pain with a SCAT2 symptom number of 3 with a symptom severity score of 10. After return of her symptoms in 4 months, the procedure was repeated with the same results. She continues to work with physical therapy and speech and language pathology to address her remaining lingering symptoms.

Discussion

What Is ON?

The International Headache Society describes ON as recurring paroxysmal attacks of severe nonthrobbing, shooting, or stabbing pain of the posterior scalp in the distribution of the GON, LON, or TON (14). These paroxysms last from seconds to minutes, but there also can be persistent pain between the paroxysms (14,28). The pain is associated with diminished sensation or dysesthesia and/or marked tenderness and pain elicited by stimulation of the hair or scalp over the affected nerve branches and/or trigger points at the emergence of the GON or in area of innervation by C2 (14). This clinical variability may be attributed to the fact that ON can involve any of the three branches of the occipital nerve (9).

The GON most commonly is involved in up to 90% of cases, whereas involvement of the LON is much less common, and the TON rarely is involved (12). Involvement of both the GON and LON is seen in 9% of cases. The GON originates from the dorsal ramus of C2, medial branch, and then travels superiorly between the inferior oblique capitis and semispinalis capitis muscles. It then extends rostralaterally deep to the trapezius muscle and pierces the aponeurosis of the trapezius just inferior to the superior nuchal ridge. This is where it becomes subcutaneous, lying medial to the occipital artery, and most often is constricted (9,28). It provides sensory innervation from the external occipital protuberance to the vertex of the posterior scalp (9). The LON is composed of branches of C2 and C3 from the cervical plexus and travels superiorly along the posterior border of the

sternocleidomastoid muscle (9). It innervates the lateral scalp and the area just posterior to the auricle (9). The TON is a branch of C3, which emerges inferiorly (7) and innervates the upper neck and lower occipital scalp (9).

History and Examination

History Precipitation of headache with movement or palpation of the neck is a common feature of ON (15). Shooting or stabbing pain in the neck radiating over the cranium is also a common complaint (28). Pain and decreased sensation is typically but not always unilateral and is most prominent in the occipital region (15). Tinnitus, scalp paresthesia, nausea, dizziness, and visual disturbances also are common complaints due to connections of the C2 dorsal root portion of the occipital nerve with cranial nerves VIII, IX, X and the cervical sympathetic (4,18,28). Pain may radiate to the parietal or temporal scalp, and it is even possible for the patient to experience pain in the fronto-orbital area due to connections in the trigeminal spinal nuclei (4,14,26,28).

ON commonly results from head trauma such as that associated with concussion and/or whiplash injuries seen in sports, motor vehicle accidents, and war zones (7). However, ON also may stem from compression due to degenerative spine changes, malignant growths, extra and intracranial vessels, giant cell arteritis, bony callus formation after vertebral fracture, or chronically contracted spinal muscles (13,17). It is important to screen for these pathologies during the history, particularly in the absence of trauma (15). Key historical information includes 1) mechanism, 2) initial symptoms (such as headache, neck pain, and stiffness), and 3) associated injuries such as fractures or impairments of vision or of the vestibular system (15). If related to concussion, headaches and other symptoms of ON typically manifest subacutely after the concussion (such as was typical in the presented cases); if they appear long after the concussive event, one should consider other etiologies (15).

Differentiating ON headaches from other types of headaches can be challenging. Typically ON does not produce an aura, which may help differentiate from migraine headache without aura or tension-type headache (15). ON should be distinguished from referred pain from atlantoaxial or upper zygapophyseal joints or from trigger points in neck muscles (as described in the first presented case) (14). Additionally, understanding patterns of referred pain can assist in the diagnosis of which nerve(s) are impaired (16).

Examination Tenderness along the course of the occipital nerves and a positive Tinel sign are common findings (9). Palpation of the GON and LON and the areas of their likely entrapment is the most important examination component. The most common location to palpate and percuss the GON is 2 cm lateral and 2 cm inferior to the external occipital protuberance (21). The LON is lateral to the GON and may be palpated/percussed along the superior nuchal ridge one-third of the way from the external occipital protuberance toward the mastoid (9).

Diagnostic testing Cervical spine x-ray images may show abnormal anatomy and demonstrate instability. Arthritic change at the second cervical vertebra may be evident on an open-mouth view (28). A CT scan of the craniocervical junction may indicate a

degenerative process and help rule out neoplastic pathology (28). Magnetic resonance imaging can show soft tissue pathology (28) such as infiltrative lesions (9), and electromyography and nerve conduction studies are useful to evaluate for radiculopathy (15). Local anesthetic injection blocks of occipital nerves are used frequently to ease pain temporarily (14,18) and provide diagnostic information (13,18,28). Ultrasound evaluation of the GON in patients with unilateral symptoms has shown an increase in cross-sectional area and circumference in the GON on the symptomatic side and shows promise as a diagnostic modality (6).

Treatment of ON

The manner in which ON is treated depends on the severity of the pain and the resultant disability. If the patient's pain and disability have not improved with conservative measures such as short-term immobilization with bracing, massage, physical therapy, anti-inflammatory medications, acetaminophen, muscle relaxants, gabapentinoids, tricyclic antidepressants, and selective serotonin and norepinephrine reuptake inhibitors, then procedural management may be appropriate. The distribution of the pain (unilateral vs bilateral, GON vs LON vs TON, or combinations thereof) and qualities of the pain will determine the approach that can be used. ON pain is most commonly unilateral and in the distribution of the GON but can be associated with TON involvement when the second and third cervical vertebrae are disturbed also (as was the case with the third patient that was presented) (12).

Traditional ON can be treated with multiple interventional or procedural strategies. The primary treatment strategy is the attempted abolition of nerve conduction of the occipital nerve at a peripheral location. This can be performed with pharmacological agents, heat, cold, and electrical stimulation. The most common starting point is the blockade of the affected nerve with a local anesthetic. This blockade will provide rapid pain relief and can be used as a diagnostic guide for future therapies. While local anesthetic blockade pharmacologically is short-lived, pain relief may be of much longer duration (as was with the presented cases); however this dissociation between the duration of pharmacological effect and the duration of symptomatic relief is not well understood (1).

The addition of other pharmacological agents to the injectate such as steroid, alpha-adrenergic modulating agents, or opioids has not been found to provide any additional analgesic benefit (1,2) and may increase the likelihood of harm; therefore these agents should be used with caution and with a specific rationale. Although the appropriate volume of the injectate has not been examined rigorously, small volumes (<1 mL per nerve) are likely to decrease the false positive rate, although this risks greater false negative results (13). A positive diagnostic block of GON, LON, or both establishes them as being involved in the pain generation. If the pain relief or duration is not sufficient, then modulation of the affected nerves can be performed with pulsed radiofrequency stimulation (Fig. 1) (13). In those who have a diagnosis of ON supported by a positive diagnostic block but did not respond to pulsed radiofrequency stimulation, occipital nerve stimulation with implanted stimulating leads and associated implantable pulse generator may be performed (Fig. 2) (24).

Occipital nerve stimulation uses a surgically implanted device to provide long-lasting relief; however it also is associated with risks including infection, migration of the leads, and lead failure. An alternative approach is to perform a local anesthetic blockade of the TON at the level of the C2 and C3 facet joint (as was performed in the third presented case). If this is positive (50% or greater relief), a radiofrequency denervation or lesioning can be performed with success rates of up to 88% (11).

Conclusions

There is currently minimal literature on ON after sports-related concussion; however due to sharing a common possible mechanism of injury and the overlapping symptomatology between these conditions, ON should be incorporated into clinicians' approach to concussion as a possible associated injury, particularly in cases that feature an appropriate distribution of symptoms in the occiput and posterior cervical spine. It is also important to realize that prolonged headache after concussion may be ON and not lingering concussion symptoms. Thus further research is needed regarding the epidemiology, recognition, diagnosis, and treatment of this entity in the setting of sports-related concussion.

References

1. Ashkenazi A, Blumenfeld A, Napchan U, et al. Peripheral nerve blocks and trigger point injections in headache management — systematic review and suggestions for future research. *Headache*. 2010; 50:943–52. [PubMed: 20487039]
2. Ashkenazi A, Matro R, Shaw JW, et al. Greater occipital nerve block using local anaesthetics alone or with triamcinolone for transformed migraine: a randomised comparative study. *J. Neurol. Neurosurg. Psychiatry*. 2008; 79:415–7. [PubMed: 17682008]
3. Babcock L, Byczkowski T, Wade SL, et al. Predicting postconcussion syndrome after mild traumatic brain injury in children and adolescents who present to the emergency department. *JAMA Pediatr*. 2013; 167:156–61. [PubMed: 23247384]
4. Baron EP, Cherian N, Tepper SJ. Role of greater occipital nerve blocks and trigger point injections for patients with dizziness and headache. *Neurologist*. 2011; 17:312–7. [PubMed: 22045281]
5. Bazarian JJ, Atabaki S. Predicting postconcussion syndrome after minor traumatic brain injury. *Acad. Emerg. Med*. 2001; 8:788–95. [PubMed: 11483453]
6. Cho JC, Haun DW, Kettner NW. Sonographic evaluation of the greater occipital nerve in unilateral occipital neuralgia. *J. Ultrasound Med*. 2012; 31:37–42. [PubMed: 22215767]
7. Cohen SP, Plunkett AR, Wilkinson I, et al. Headaches during war: analysis of presentation, treatment, and factors associated with outcome. *Cephalalgia*. 2012; 32:94–108. [PubMed: 21994113]
8. De Kruijk JR, Leffers P, Menheere PP, et al. Prediction of post-traumatic complaints after mild traumatic brain injury: early symptoms and biochemical markers. *J. Neurol. Neurosurg. Psychiatry*. 2002; 73:727–32. [PubMed: 12438478]
9. Dougherty C. Occipital neuralgia. *Curr. Pain Headache Rep*. 2014; 18:411. [PubMed: 24737457]
10. Faux S, Sheedy J. A prospective controlled study in the prevalence of posttraumatic headache following mild traumatic brain injury. *Pain Med*. 2008; 9:1001–11. [PubMed: 18266807]
11. Govind J, King W, Bailey B, Bogduk N. Radiofrequency neurotomy for the treatment of third occipital headache. *J. Neurol. Neurosurg. Psychiatry*. 2003; 74:88–93. [PubMed: 12486273]
12. Hammond SR, Danta G. Occipital neuralgia. *Clin. Exp. Neurol*. 1978; 15:258. [PubMed: 756019]
13. Headache Classification Committee on the International Headache Society (IHS). The International Classification of Headache Disorders, 3rd edition (beta version). *Cephalalgia*. 2013; 33:629–808. [PubMed: 23771276]

14. Hecht JS. Occipital nerve blocks in postconcussive headaches: a retrospective review and report of ten patients. *J. Head Trauma Rehabil.* 2004; 19:58–71. [PubMed: 14732831]
15. Huang JH, Galvagno SM Jr, Hameed M, et al. Occipital nerve pulsed radiofrequency treatment: a multi-center study evaluating predictors of outcome. *Pain Med.* 2012; 13:489–97. [PubMed: 22390409]
16. Johnston MM, Jordan SE, Charles AC. Pain referral patterns of the C1 to C3 nerves: implications for headache disorders. *Ann. Neurol.* 2013; 74:145–8. [PubMed: 23424170]
17. Kapural L, Stillman M, Kapural M, et al. Botulinum toxin occipital nerve block for the treatment of severe occipital neuralgia: a case series. *Pain Pract.* 2007; 7:337–40. [PubMed: 17986166]
18. Kuhn WF, Kuhn SC, Gilberstadt H. Occipital neuralgias: clinical recognition of a complicated headache. A case series and literature review. *J. Orofac. Pain.* 1997; 11:158–65. [PubMed: 10332322]
19. Langlois JA, Rutland-Brown W, Wald MM. The epidemiology and impact of traumatic brain injury: a brief overview. *J. Head Trauma Rehabil.* 2006; 21:375–8. [PubMed: 16983222]
20. Lincoln AE, Caswell SV, Almgvist JL, et al. Trends in concussion incidence in high school sports: a prospective 11-year study. *Am. J. Sports Med.* 2011; 39:958–63. [PubMed: 21278427]
21. Loukas M, El-Sedfy A, Tubbs RS, et al. Identification of greater occipital nerve landmarks for the treatment of occipital neuralgia. *Folia Morphol. (Warsz).* 2006; 65:337–42. [PubMed: 17171613]
22. Marar M, McIlvain NM, Fields SK, Comstock RD. Epidemiology of concussions among United States high school athletes in 20 sports. *Am. J. Sports Med.* 2012; 40:747–55. [PubMed: 22287642]
23. Olesen, J. International Headache Society. 2nd. Blackwell Science Ltd; Oxford (UK): The International Headache Classification.
24. Palmisani S, Al-Kaisy A, Arcioni R, et al. A six year retrospective review of occipital nerve stimulation practice—controversies and challenges of an emerging technique for treating refractory headache syndromes. *J. Headache Pain.* 2013; 14:67. [PubMed: 23919570]
25. Ponsford J, Cameron P, Fitzgerald M, Grant M. Long-term outcomes after uncomplicated mild traumatic brain injury: a comparison with trauma controls. *J. Neurotrauma.* 2011; 28:937–46. [PubMed: 21410321]
26. Rifat SF, Lombardo JA. Occipital neuralgia in a football player: a case report. *Clin. J. Sport Med.* 1995; 5:251–3. [PubMed: 7496851]
27. Stulemeijer M, van der Werf S, Borm GF, Vos PE. Early prediction of favourable recovery 6 months after mild traumatic brain injury. *J. Neurol. Neurosurg. Psychiatry.* 2008; 79:936–42. [PubMed: 17951281]
28. Vanelderden P, Lataster A, Levy R, et al. Occipital neuralgia. *Pain Pract.* 2010; 10:137–44. 8. [PubMed: 20415731]

Figure 1. Location of pulsed RF stimulating probe. (Reprinted from Huang JH, Galvagno SM Jr, Hameed M, *et al.* Occipital nerve pulsed radiofrequency treatment: a multi center study evaluating predictors of outcome. *Pain Med.* 2012;13:489–97. Used with permission.)

Figure 2.
Occipital nerve stimulator. Permission granted from RW Hurley (co-author).