Ozone Injection Treatment for Unremitting Head and Neck Pain Post Motor Vehicle Accident: A Case Report

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ABSTRACT

Background: An 18-year-old Caucasian female presented for treatment 8 months after a rear impact car crash. She complained of cervical spine pain, pain at the base of the skull, and headaches which began at the base of the skull and radiated bilaterally to the back of the skull and above the ears.

Methods: The patient was treated with ozone injections to the suboccipital and upper cervical paraspinal musculature.

Results: The patient reported a 90% decrease in headache frequency and intensity over a 90-day period following the injections.

Conclusion: Properly diagnosed, unremitting head and neck pain can respond favorably to ozone injection therapy when other standard conservative treatment options have failed.

INTRODUCTION

An understanding of the anatomy and physiology of the suboccipital and upper cervical spine musculature (SUCM) (Semispinalis Capitus, Inferior Oblique, Splenius Capitus, Trapezius, Sternocleidomastoid) with relation to the greater and lesser occipital nerves is required to understand the pathogenesis of headaches secondary to compression and

irritation of these nerves in the suboccipital region. The SUCM can lead to compression of the Greater and Lesser Occipital Nerves (GLON) as they course through the juncture of the skull and upper cervical area as seen in **Figure 1**.[1]

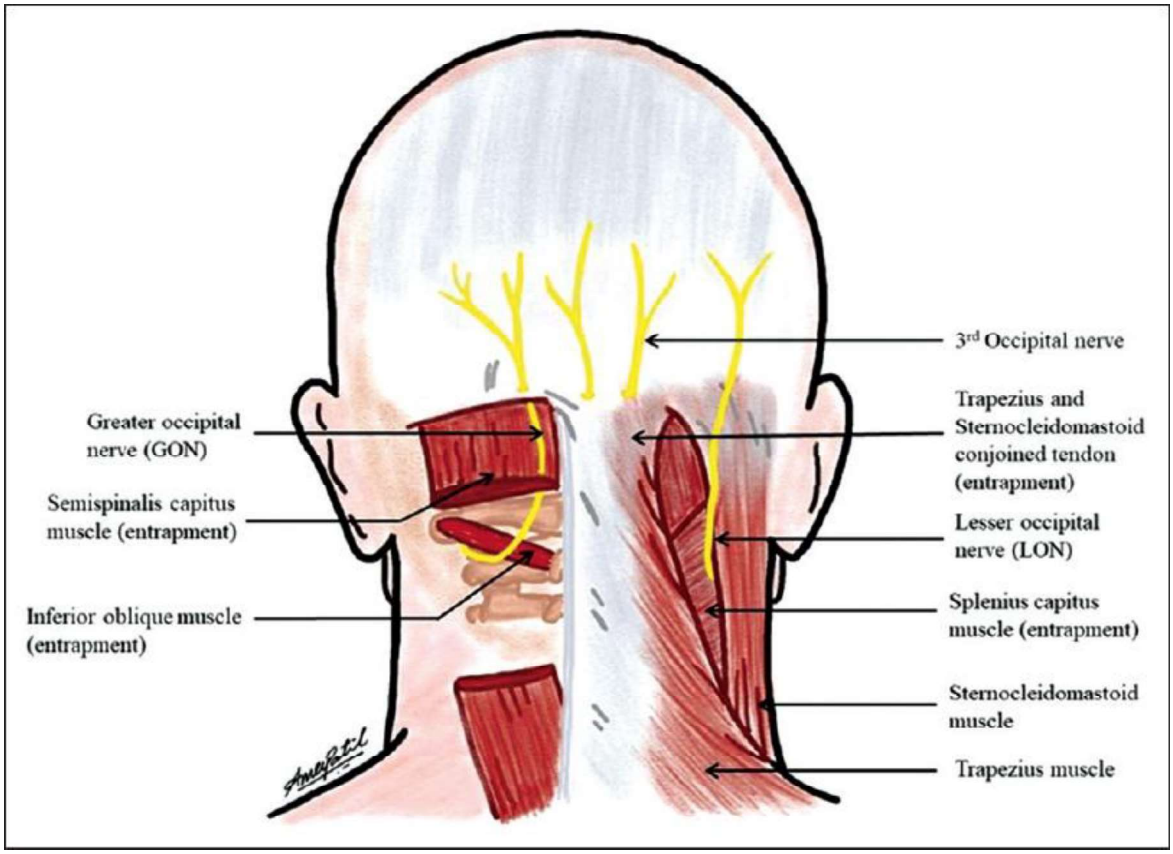

Figure 1

Detailed cadaver studies have shown several areas for possible compression of the occipital nerves along their paths through the posterior cervical muscles and fascia. The GLON may be compressed at one or more of many locations: between the semispinalis and the inferior oblique muscle; at the entry to the semispinalis capitis muscle; at the exit from the semispinalis capitis muscle; at the entrance to the trapezius muscle; the exit from the trapezius; at the fascial insertion of the trapezius at the occipital ridge; and by the occipital artery at the distal location of the nerve above the skull base. The lesser occipital nerve may be compressed by the fascial attachment of the sternocleidomastoid muscle, by branches of the occipital artery, and by fascial bands.[2]

At this time, there are not adequate imaging techniques available to image the full scope of the compression sites of the lesser and greater occipital nerves; while segments of the occipital nerves may be visualized with ultrasound, as well as the morphologic changes caused by compression within the semispinalis capitis muscle, it is not possible to visualize the nerve in its entirety to allow determination of all the sites of compression.[3]

The mechanism by which occipital nerve compression may cause headaches may lie in the resulting inflammation that has been observed in both animal and human studies of nerve compression. Experiments in rat models have demonstrated inflammation both local to and remote from the site of peripheral nerve compression.[4] Similar findings of local inflammation due to nerve compression in human subjects were demonstrated in a 2014 study. The authors reviewed the pathology of resected sections of the lateral femoral cutaneous nerve in seven subjects with meralgia paresthetica who underwent neurectomy for chronic, disabling pain. Histology of the resected nerves showed perineural thickening, demyelination, axonal degeneration, and varying degrees of endoneurial and epineural inflammation. Collections of inflammatory cells of mild to moderate size were observed in five of the seven subjects, and the degree of inflammation correlated with the degree of axonal injury. Review of the same nerve from autopsy controls did not reveal perineural thickening, axonal loss, or inflammation.[5]

It is important and helpful to distinguish the symptoms of unremitting head and neck pain associated with occipital nerve compression as defined by the International Headache Society.[6] Occipital Neuralgia (Greater and Lesser) is defined as pain that has two of the following three characteristics: 1) recurring in paroxysmal attacks lasting from seconds to minutes; severe in intensity; shooting, stabbing or sharp in quality, 2) The pain is associated with dysesthesia and/or allodynia, and 3) either tenderness to palpation of the nerve or its branches, or the presence of tender trigger points at the emergence of the occipital nerves. The pain must also be temporarily eased by an anesthetic block of the occipital nerves.

Proper diagnosis of occipital nerve compression as the cause of the patient's unremitting head and neck pain is paramount. The Headache Classification Committee of the International Headache Society (IHS) in the International Classification of Headache Disorders, 3rd edition clarifies some of the differential diagnoses with regards to unremitting head and neck pain and occipital neuralgia. The presence of unremitting head and neck pain in no way excludes the presence of clearly central and brainstem processes being instrumental in the pathophysiology of other headache types, such as low-frequency migraine with aura. One may, in fact, conceptualize headache disorders existing along a continuum of pathophysiology, reflecting the anatomic continuum of nerves from intra- to extracranial. Strong central processes such as low-frequency episodic migraine with aura lie at one end, the "central end," of the spectrum, while unremitting, solely sub-occipital and occipital pain lies at the other, "peripheral end," of the spectrum. Most patients will reside at a point between the ends of the spectrum, and some patients may have two completely different headache types, and thus reside at two places on the spectrum. The clinical characteristics of the separate headaches will differ, as will effective treatments for each headache type. The concept of a spectrum for the pathophysiology of headache disorders is, in fact, illustrated by the range of responses following nerve decompression. Some patients with the purely peripheral factor of nerve compression may have a significant reduction in

pain, similar to the reduction of lumbar radicular pain seen after a lumbar microdiscectomy, while those individuals with additional centrally mediated headaches such as menstrual migraine, chemically induced headaches, dehydration headaches, or migraine with aura will likely continue to experience these headaches. Correct identification of patients who have predominant peripheral factors such as occipital nerve compression as the cause of the unremitting head and neck pain is the most important step in determining if occipital nerve decompression is an appropriate intervention. It is important to note, however, that elimination of the posterior peripheral trigger for unremitting head and neck pain *may* additionally reduce frontal headaches that are consistent with migraine, again, likely reflecting the anatomic spectrum of the neural networks that span both the extracranial and intracranial spaces, and the elimination of activation of extracranial trigeminal nociceptors by the removal of the inflammation-causing compression. It is also of note that indirect results of nerve decompression may result in additional improvement that extends beyond the elimination of unremitting head and neck pain, such as improved sleep from the reduction in occipital allodynia, proper hydration, or dependency on caffeine.[6]

Ozone aids in the inhibition of pain receptors as well as a decrease in myofascial hypertonia. Several authors have described the anti-inflammatory, analgesic, and anti-edema properties of injected medical ozone, and propose that the oxidation of the algogenic receptors would inhibit the pain signal and activate the antinociceptive system.[7] This is supported in a preclinical study in which the authors induced sciatic damage in mice and confirmed the corticofrontal activation of genes of caspase 1, 8, and 12 (pro-inflammatory, pro-apoptotic, and responsible for allodynia) caused by the injury; this expression was normalized with a single peripheral injection of O_2/O_3 around the damaged area, which also reduced mechanical allodynia.[8]

These properties would favor a muscle relaxant effect, as well as improved mobility of the treated area that can be observed clinically. This effect is very important in muscle recovery with O_2/O_3 injections.[9] The utility of ozone therapy in the treatment of painful muscle hypertonia highlights the tremendous muscle relaxant effect that is produced.[10]

Headaches after a car crash are common and are recognized as one of the bigger pain complaints among patients who have sustained a hyper acceleration/deceleration injury.[11] However, ozone injections are not commonly utilized as a successful treatment option. This case indicates that ozone injections should be considered.

CASE PRESENTATION

An 18-year-old Caucasian female presented for care and treatment for her persistent neck and head pain following a rear impact motor vehicle collision 8 months prior.

Following the crash, the patient received multiple conservative treatment options at other facilities. These included chiropractic manipulation, physiotherapy modalities, deep tissue

myofascial release therapy, and cervical spine specific rehabilitation. The patient reported a slight decrease in her pain for a short period of time with each treatment. However, the head and neck pain would return within hours. She also underwent myofascial trigger point injections to the parascapular musculature. She reported only a few hours of pain relief with these injections.

At her initial visit, the patient described the pain as a 3/10 at its best and a 7/10 at its worst. The headaches were described as severe, sharp, shooting, constant, daily, and started at the base of the skull then radiated to the side of her head and over her ears.

Examination of the cervical spine revealed a decrease in active cervical spine range of motion with pain. Significant tenderness in the bilateral cervical paraspinal musculature and bilateral suboccipital musculature was noted. When palpated, the suboccipital myofascial hypertonicity reproduced the patient's sharp and shooting head pain. The location of the head pain was described by the patient as starting at the back of the head/base of the skull with radiating head pain above the ears.

The patient had no history of migraine headaches or migraine with aura. She did describe periodic headaches prior to the crash associated with dehydration, caffeine consumption, and stress. These headaches were infrequent and occurred with physical activity, dietary changes, and stressful situations.

Following the examination and subsequent diagnosis of unremitting head and neck pain associated with occipital nerve compression/irritation, a discussion of mechanism of injury and etiology of the condition were explained to the patient, and intramuscular ozone injections to the upper cervical musculature and suboccipital musculature were recommended.

After obtaining consent for treatment, the patient was taken to a procedure room where she was instructed to lay prone on the procedure table. The upper cervical/suboccipital area was exposed. The most tender musculature in the upper cervical and suboccipital regions were palpated and identified. The patient confirmed the areas to be injected by indicating a reproduction of her head pain upon palpation of the subject musculature. Once the areas for injection were identified and marked (2 in the suboccipital musculature and 2 in the upper cervical spine musculature), a local anesthetic spray was applied to the injection sites. The skin was then prepared with standard sterile preparation techniques. A 30-gauge ½ needle was then utilized to inject 5ml of 19 gamma ozone into each of the target muscles for a total of 20ml of ozone. The injection sites were then covered with a small bandage and the patient underwent 15 minutes of interferential therapy to the region. The patient was given instructions to avoid soaking the affected area in water for 24 hours. She was given a handout with home based cervical spine stretches to perform and was instructed to perform these stretches several times a day for the next week.

Within one week the patient reported a significant decrease in the frequency and intensity of her headaches. She reported experiencing only 2 headaches in the 7-day period. It is important to note that one of those headaches, according to the patient, was due to dehydration as she hiked a lot the day that the headache appeared. Two weeks after the injections, the patient was no longer experiencing headaches.

90 days after the intramuscular upper cervical and suboccipital ozone injections, the patient reported a 90% decrease in the frequency and intensity of her headaches. According to the patient, her post injection headaches now coincided with increased stresses associated with schooling and/or a lack of caffeine consumption. This would indicate a return to her normal degree of headaches in frequency and causative factors.

An updated palpatory examination of the upper cervical and suboccipital musculature was now negative for reproduction of the patient's head pain. This, in conjunction with the patient's description of her changes in symptoms, indicated a successful decompression of the occipital nerves and relief of the unremitting head and neck pain with intramuscular ozone injections.

CONCLUSION

Unremitting head and neck pain is common following an injury to the upper cervical spine and suboccipital musculature. While conservative treatment options such as spinal manipulation, physiotherapy modalities, deep tissue therapy, trigger point injections to the parascapular musculature, and spine specific rehab are often effective in treating head and neck pain, some patients do not respond to traditional treatment options, as was the case with this patient. After proper diagnosis and treatment with intramuscular ozone injections, decompression of the occipital nerves was achieved thus eliminating the patient's injury related head and neck pain.

With a good history, examination, and proper diagnosis, injury induced unremitting head and neck pain can be treated successfully with intramuscular ozone injections. If a patient does not respond to physical medicine modalities, traditional chiropractic care, conservative treatment options, or ozone injections, additional investigation or referrals may need to be considered.

CONSENT

Written informed consent was obtained from the patient for publication of this case report.

COMPETING INTERESTS

The author declares no competing interests.

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