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MEDICINE

Orofacial Neuralgia Following Whiplash-Associated Trauma: Case Reports and Literature Review

Firoozeh Samim [1] · Joel B. Epstein [2,3,4,5]

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Abstract

Whiplash-associated disorder (WAD) has a close relationship to and functional linkage with the neck and jaw. Studies show an association between orofacial pain and cervical pain and confirm intersegmental nociceptive connections between the trigeminal regions and the cervical spine. We present six cases of WAD-associated orofacial neuralgia. The objective of this study is to present a series of cases of neuralgia-like pain following WAD. Six cases of WAD-associated orofacial neuralgic pain are presented. Neuralgia was identified based upon history, examination, and successful pain management with specific medications for neuralgia. Five out of the six cases were initially diagnosed and treated with limited efficacy as temporomandibular joint disorder (TMD) with continuing pain and were subsequently diagnosed and treated with efficacy as trigeminal neuralgia. MRI and CT scan may suggest cervical spine changes mostly at C2–3, C3–4, and C5–6. Traditional therapy for musculoskeletal pain had no impact upon orofacial pain in patients with neuralgia-like pain following WAD. These cases show that development of neuralgia-like pain may have delayed onset following WAD. Clinical presentation and diagnosis of neuralgia were confirmed upon control of pain with use of carbamazepine or oxcarbazepine. To our knowledge, these cases represent the first report of WAD-related orofacial neuralgia and suggest the potential for neuralgia-like pain to occur following WAD and that clinical diagnosis is needed to lead to effective therapy.

Keywords Orofacial pain · Trigeminal neuralgia · Whiplash-associated disorder · Temporomandibular disorders · Post-traumatic trigeminal neuropathy

Introduction

Whiplash-associated disorder (WAD) is a collection of symptoms that can affect the neck, back, and jaw, triggered by an accident with an acceleration-deceleration mechanism, most often a motor vehicle collision [1]. WAD has been a controversial and debated condition in part due to the compensable nature of the injury, the lack of precise pathological diagnosis, and lack of specific diagnostic tests [2]. The current understanding of WAD is that it is caused by an initial peripheral injury to the affected regions including the back, neck, and jaw following whiplash injury with or without head injury [3], although both peripheral and central sensitization may also occur. Cervical WAD may be reported as soft tissue injury and, with more severe injury, may include cervical spine injury. Regardless of whether a peripheral lesion is present, it is important to consider the processes that may be initiated by whiplash-associated pain, dysfunction, and related symptoms,

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✉ Firoozeh Samim
firoozeh.samim@mcgill.ca

Joel B. Epstein
joel_epstein@hotmail.com

[1] Faculty of Dentistry, McGill University, Montreal, QC, Canada

[2] Department of Surgery, Cedars-Sinai Health System, Los Angeles, CA, USA

[3] Division of Otolaryngology and Head and Neck Surgery, City of Hope National Medical Center, Duarte, CA, USA

[4] Department of Oral Medicine, University of Washington, Seattle, WA, USA

[5] Attending Oral Medicine, Seattle Cancer Care Alliance, Seattle, WA, USA

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particularly in those who develop chronic symptoms following the trauma of an accident [4].

Although most individuals recover from WAD, a substantial number may develop chronic symptoms. A review of 50 chronic WAD patients reported that 94% had shoulder pain, 90% headache, 88% jaw-face pain, and 72% back pain [5]. A variety of additional symptoms are also reported, including disturbances in concentration, memory, sleep, hearing, vision, and mood, and dizziness. Female patients are often predominant in WAD cases [5].

Neuropathy associated with WAD is rarely reported in the literature. Cases with neuralgia-like pain may be included in a broad definition of neuropathy, but specific reports of WAD-associated, neuralgia-like orofacial pain has not been specifically described [4]. A total of six cases of WAD-related orofacial neuralgia are reviewed here. All six patients contacted individually and completed a signed consent form. Our literature review summarizes the current understanding, symptoms, and management of WAD-related neuropathy with a focus on orofacial neuralgia-like pain.

Case Reports

Case 1

A 50-year-old female was involved in a motor vehicle collision and diagnosed with WAD grade II of the neck, upper back, and lower back, and with headache. Direct facial impact in the accident and prior history of pain in the face, neck, or head were denied. Facial pain was diagnosed 1 month post-accident and became severe after 1 year, when it was described as sharp and electric pain in the right lateral tongue and triggered sharp pain with light touch of the tongue and the right lower lip. Descriptors of tongue pain included sharp, stabbing, and stinging with aching pain between severe episodes. Scored on a pain scale of 0 to 10, the pain was rated as 6 to 8.

The patient was given a diagnosis of temporomandibular pain and initial therapy for TMD, including an oral appliance and physiotherapy. Headache occurred primarily in the right temple. Mild tenderness was present in the lateral pole of the left temporomandibular joints (TMJ) and the left posterior masseter muscle. Magnetic resonance imaging (MRI) of the neck suggested possible bulging disks at C3–4 and C5–6 without foraminal narrowing.

In addition to TMD, the patient was diagnosed with trigeminal neuralgia based upon tongue pain triggered by eating, speaking, and brushing teeth. Facial pain was controlled with oxcarbazepine (300 mg, twice a day [BID]). After 3 years, the patient continued on oxcarbazepine to control trigeminal neuralgia and used an oral appliance to control mild TMD.

Case 2

A 34-year-old female was involved in a motor vehicle collision and diagnosed as WAD grade II with pain in the neck, upper back, and low back, and headache. No direct trauma occurred, and loss of consciousness was denied. One year post-accident, jaw pain was reported following physical manipulation of the neck. The jaw pain was reported to be present in the left lower molar region and extended to the first bicuspid area. The jaw pain was scored as 10 out of 10, with characteristics of sharp, throbbing, stabbing, and piercing pains. The pains forced her off work, prevented mouth care, and affected her diet. Because the pain was triggered by eating, her diet was limited to liquids taken through a straw.

MRI of the neck and central nervous system (CNS) ruled out lesions and demyelinating changes; however, possible mild cervical disk displacement at C2–3 and C5–6 was noted. Instability of C2–3 was reported by physical therapy. Multiple dental evaluations did not identify any dental concerns. The patient was diagnosed with TMD with mild tenderness in temporalis, trapezius muscle in the neck, and at posterior skull insertion. Mild tenderness of the lateral capsule in the right TMJ and intraoral temporalis tendon was seen. Examination revealed that the cranial sensory nerves were intact; the patient reported occasional triggering of pain in the lower molar gingiva with light touch.

The patient was treated with gabapentin, pregabalin, ibuprofen, naproxen, and Toradol with minimal effect upon pain. However, carbamazepine (100 mg, BID) led to rapid pain relief with the jaw pain, which decreased to 2 to 3 out of 10 after 1 week of carbamazepine. At follow-up, the triggered facial pain was resolved, neck and back symptoms were controlled, headache was controlled with a residual sensation of tightness, and mild aching occurred only a few times per month.

Case 3

A 56-year-old female was diagnosed with WAD with pain in the back, neck, and bilateral face following a motor vehicle collision. When seen, she reported a 4-month history of sharp pain in the anterior maxilla that was scored as 10 out of 10. A history of jaw joint clicking or parafunctional jaw habit was denied. Sharp and excruciating pains occurred when her anterior teeth came into contact. The patient avoided chewing and had lost weight.

No oral or dental findings were noted on clinical exam and imaging. Physiotherapy, acupuncture, and osteopathy were performed for at least eight sessions with no improvement. Jaw manipulation under intravenous sedation did not show limitation, and no benefit was seen following jaw manipulation. There was no effect on pain when local anesthetic infiltration was administered in the anterior maxilla, and no benefit

was reported following a trial of clonazepam. The patient was then considered to have post-traumatic trigeminal neuralgia and prescribed with carbamazepine (400 mg, BID) as a treatment trial, which decreased pain by an estimated 75%.

Case 4

A 36-year-old female was involved in a motor vehicle collision, diagnosed with WAD, TMD, and post-traumatic trigeminal-like neuralgia pain. One year following the motor accident, she reported painful bilateral TMJ clicking, with sharp pain in the left side of the face and associated headaches. Jaw movement, palpation, and cold weather triggered the sharp pain. Other pains were reported in the eyes and temples. She had history of migraine headaches, which were more severe and frequent since the accident.

Unassisted jaw opening was limited to 28 mm and assisted was 42 mm, causing bilateral TM joint and masseter pain. Bilateral TM joint reciprocal clicking was present. Masticatory muscles (temporalis and masseter) and TMJ tenderness were identified. Occipital tenderness was noted with unknown cause. Light touch of the left cheekbone triggered paroxysmal sharp pain that lasted for a few seconds. MRI and computed tomography (CT) scan did not show any fracture or lesions in the CNS, along the trigeminal nerve and no abnormality was seen in the cervical spine.

No effect on pain was seen following treatment trials of muscle relaxants, massage therapy, and physiotherapy. Oral appliance with anterior repositioning and an oral stabilizing appliance were provided with no improvement at a 2-month follow-up. Prescription of carbamazepine (400 mg, BID) improved facial pain by an estimated 70%, with resolution of sharp pain on the left face, and touching the left face no longer triggered sharp pain.

Case 5

A 36-year-old female was involved in a motor vehicle accident that involved a severe left front vehicular impact leading to seatbelt bruising, but no direct head or facial trauma. She was diagnosed with cervical and back WAD grade II, TMD post-MVA, but was upgraded to cervical WAD grade III due to neurologic signs in the legs and left arm. Concussive symptoms with headache, pain, tingling, and numbness in the arm were reported. She also developed altered sleep patterns and memory loss. Jaw pain occurred within a few weeks of the accident, with left TMJ clicking, left jaw pain, and left-sided headaches, all of which occurred daily. When headaches were severe, she developed pulsing pain, visual sensitivity, and occasional nausea. She also noted a hot sensation in the neck and left face.

She developed additional new facial pain approximately 4 years following the MVA. Pain was described as sharp, stabbing, and electric, and was triggered with light touch over the left upper lip and with tooth brushing of the upper left teeth. Examination identified a pain trigger point in the left infraorbital nerve where hyperesthesia was present adjacent to the left ala of the nose, and increased sensitivity was reported at the vermillion border of the left upper lip. Light touch triggered the severe stabbing jaw pain. MRI suggested possible vessel loop on the cisternal portion of the trigeminal ganglion, but did not identify any lesions in the CNS or cervical spine.

Treatment of jaw symptoms following the MVA included an oral appliance with physical therapies. A short-term trial with amitriptyline and gabapentin for pain was discontinued due to side effects and lack of significant benefits. Following the onset of the severe sharp, stabbing, electric pain (10/10) that extended to the upper left canine tooth and left eye, she was provided carbamazepine (100 mg BID) with significant pain improvement. The dose of carbamazepine was increased (200 mg BID) and complete pain relief was reported. While she had excellent response to carbamazepine, she had mild side effects and was anxious to be off medication. Neurosurgical decompression of the ganglion was completed and symptoms remain resolved.

Case 6

A 48-year-old female was involved in a severe motor vehicle accident with no direct head or facial trauma, and was diagnosed with WAD, TMD, and post-traumatic trigeminal-like neuralgia pain. Constant pain was reported on the left face with limited mouth opening. The pain was sharp for a few seconds and then became dull; its intensity was 6 out of 10, subsiding gradually over 3 to 4 days. Pain started in the TMJ region and radiated to the cheek and ear with headaches. Open jaw locking occurred several times and was released by self-maneuver. Chewing, yawning, talking, smiling, and bending the head forward aggravated the pain, and she limited her diet, which resulted in weight loss of almost 10 lb.

Jaw opening was limited to maximum 19 mm, and maximum assisted opening was 26 mm. Light touch on the right and left masseter and temporalis caused moderate pain. MRI showed the presence of vessel on the trigeminal ganglion, but no changes were identified in the CNS or cervical spine.

The early treatment included an oral appliance and self-management. Massage and physical therapy were not helpful. Over-the-counter pain medications were helpful in pain management. Three years after the accident, additional symptoms presented with severe sharp, stabbing, and electric pain that extended to the upper left canine tooth and left eye. Carbamazepine at 400 mg BID was associated with significant pain improvement. Carbamazepine dose was increased to treat recurred pain with benefits.

Discussion

We present six cases of neuralgia-like pain following motor vehicle accident trauma and WAD, which were managed successfully with specific medications for neuralgia.

WAD-related orofacial pain with neuralgia characteristics may have a functional linkage between the jaw and neck regions as shown in experimental and clinical studies [6–8]. Trigeminal nerve and CNS input is immediately adjacent to cervical spine sensory input, and crossover and sensory amplification may occur, potentially affected by head/neck movement in whiplash movement [9]. It is possible that trigeminal-cervical reflexes may be responsible for WAD-related orofacial neuralgia-like pain. Studies show the association between orofacial pain and neck pain [10, 11], and confirm the intersegmental nociceptive connections between the trigeminal regions and the cervical spine [12]. In addition, the fusimotor muscle spindle system may play a critical role in chronic cervical and masticatory muscle pain related to the onset, spread, and perpetuation of regional pain [13, 14].

In the cases reported here, five out of the six WAD cases were treated for TMD prior to diagnosis of neuralgia-like pain. TMD has been reported to occur in patients post WAD, related to accidents with alterations in muscles and sometimes in TMJ function in patients [15]. A review of 125 articles assessing the incidence of TMD pain in WAD shows that the median prevalence of TMD pain after WAD is 23%, ranging from 2.4 to 52%, compared with 3% in healthy controls [16]. However, it remains unknown if structural or functional changes in cervical muscles are related to TMD in WAD subjects with neck pain. Furthermore, the roles of peripheral and central sensitization are not fully understood. MRI studies that explore the cervical extensor musculature have identified fatty tissue infiltration in the cervical extensor muscles, which could be associated with sensory, motor, and physical dysfunction in chronic WAD [17–19]. Additional research is required to clarify the relation between TMD occurrence, persistence, and its mechanism in WAD patients [20, 21]

All six of the cases followed WAD due to a motor vehicle collision, and all cases had cervical involvement and TMD. The patients were subsequently diagnosed with trigeminal neuralgia-like pain, which was effectively treated as neuralgia, confirming the working diagnosis. Neuralgia-like symptom is likely a rare cause of pain in chronic WAD [4]. These six cases suggest that trigeminal neuralgia may arise in WAD cases and may be delayed in diagnosis or onset [22]. All cases had TMD, neck pain, and headache; three had complex headaches (myofascial/vascular symptoms, autonomic signs). Among these six cases, one patient refused imaging, MRI in one case failed to show any changes, MRI identified cervical spine changes in two cases, and two had a blood vessel identified on the trigeminal ganglion. Four cases had delayed onset, and three cases had delayed diagnosis while undergoing treatment of WAD and TMD.

The development of neuralgia associated with WAD, as reported here and discussed in previous publications, may upgrade the classification of WAD to grade III because patients with disturbed trigeminal nerve function fulfill the WAD grade III criteria [4, 23]. Accordingly, the examination of the trigeminal nerve function should be included in WAD patient diagnosis to improve the WAD classification, and health care providers should be aware of the potential for delayed onset of neuralgia symptoms.

Diagnosis of WAD-related orofacial neuralgia needs to be differentiated from traumatic orofacial neuropathy with direct traumatic or surgical damage to the nerves in the orofacial region, which can result in trigeminal neuropathy [24]. Patients with traumatic orofacial neuralgia may have traumatic histories involving the trigeminal nerve, but without WAD history. For the majority of traumatic orofacial neuropathy patients (about 54%), the condition may be caused by mandibular third molar surgery, and in others, traumatic orofacial neuralgia may be associated with other dental surgeries and procedures. However, less than 5% of cases are associated with orofacial trauma [25]. The differentiation between WAD-related orofacial neuralgia and TMD can be achieved by the WAD history, symptom report, clinical examination findings, in some cases imaging of the head and neck, and by confirmation during a therapeutic trial with appropriate medication. Patient-completed screening questionnaire such as PD-Q (painDETECT screening questionnaire) can be used as a pre-consultation tool to facilitate the diagnosis. However, the specificity and sensitivity of this questionnaire have not been tested in this cohort of patients. The results of previous studies on the validity and accuracy of PD-Q in assessing neuropathic pain and post-traumatic trigeminal nerve injuries and distinguishing these conditions from non-neuropathic pain are variable. PD-Q has the potential to assist in triage of patients [26].

The current limited understanding of WAD-related orofacial neuralgia-like pain may lead to delayed diagnosis based on symptoms of pre-trigeminal neuralgia and classical neuralgia. When dental conditions can be ruled out, clinical diagnosis of WAD-associated orofacial neuralgia can be made based on WAD history and according to the symptoms of trigeminal neuralgia evolving over time, and confirmed by response to specific medication for neuralgia. MRI and CT scan may or may not indicate cervical injury mostly at C2–3, C3–4, and C5–6, as seen in our cases, or by vascular compression of the trigeminal ganglion. Continuing follow-up is needed to rule out the potential of demyelinating diseases. It is important to note that, based on the cases presented here and suggestions in the literature [3], processes that may initiate or enhance whiplash-related pain and related symptoms may be

overlooked in WAD patients, given the common soft tissue effects in the affected region.

Post-traumatic trigeminal neuropathy refers to pain after trauma such as fracture that develops usually 3–6 months after the incident which may affect all three divisions of the trigeminal nerve. Patients with post-traumatic trigeminal neuropathy might complain of allodynia, hypo- or hyperalgesia, and often associated psychological components such as depression and anxiety [27]. Although WAD-associated orofacial pain neuralgia might mimic the same pathophysiology, it may be more complex but clinical diagnosis supported by response to medications such as carbamazepine.

Even though we found no research related to specific mechanisms of WAD-associated orofacial neuralgia, research data demonstrate that sensory disturbance may be associated with pain in chronic WAD [4]. Other than potential cervical spine change, the absence of tissue damage in local body parts suggests sensitized peripheral and central nociceptive pathways may cause the pain hypersensitivity. This can be seen as decreased pain thresholds to various stimuli such as thermal, electrical, vibration, and mechanical pressure stimulation [28–30]. These mechanisms may be relevant in the six cases reported here, as none of the cases of WAD-associated orofacial neuralgia was found to have tissue damage at the site of pain. Altered thermal sensitivity in facial skin of chronic WAD caused by motor accident is detectable using quantitative sensory testing [31]. Additionally, minimal stimulation by light touching, and associated with eating, which represents mechanical stimulation, triggered severe pain in these six cases. Jaw movement and cold weather also triggered severe pain in one case. The pain complaints were severe and not controlled until clinical diagnosis of trigeminal neuralgia-like pain was made and medication for neuralgia was prescribed.

These cases show that complex pain, including jaw pain, may be associated with WAD and that details of history and examination are necessary to identify probable conditions and begin effective treatment. In the reported six cases, delay in effective management may have occurred due to lack of defined differential diagnosis, lack of awareness of differences in common musculoskeletal pain in WAD and neuralgia, and lack of reports in the literature. While it is possible that these cases of neuralgia-like pain may have arisen spontaneously, unrelated to WAD, the timing and onset suggest an association with WAD. In any event, the nature of symptoms required specific management for neuralgia to control the severe orofacial pain.

These cases likely represent uncommon presentation of pain following trauma and WAD. In patients with TMD pain and WAD, conventional TMD treatment, such as jaw exercises and occlusal appliances, has less effect with an improvement rate of 48%, ranging from 13 to 68%, compared with 75% in TMD patients without trauma and WAD [16]. In clinical care, it is critical that no invasive or irreversible treatment is provided for TMD in complex settings of TMD and trauma unless specific diagnosis is made; appropriate management should be directed at the clinical pain presentation. Clinical diagnosis is critical in receiving intervention for pain. Without proper clinical diagnosis, inappropriate or unnecessary dental care may be performed, and appropriate therapy may be delayed.

This report presents the first case series in the English literature of WAD-associated orofacial neuralgia caused by motor accident. The literature includes several related publications [5, 16, 30] and discussions of possible mechanisms of pain in WAD, but it does not specifically address trigeminal neuralgia-like symptoms. For example, a report of 50 cases of jaw-face pain in chronic WAD was made based on patient questionnaires; the report focused on patients' symptoms without clinical data [5]. Another report explored 65 cases of TMD in patients with motor vehicle accidents, late development of WAD, and orofacial pain, but excluded patients with whiplash injury [32]. An additional report reviewed 125 articles about TMD pain after whiplash trauma from 1966 to 2012 without focusing on or indicating motor vehicle collision involvement [16].

Conclusion

We describe six cases of orofacial neuralgia in subjects with WAD. These cases suggest that the symptoms of orofacial neuralgia may be identified from a couple of months to a few years post-accident accompanied by pains in different body parts such as the neck, upper or lower back, and head. Orofacial neuralgia symptoms may overlap those of TMD, but all cases had specific symptoms of trigeminal neuralgia-like pain and responded to specific medication trials. MRI and CT scan may or may not indicate cervical injury mostly at C2–3, C3–4, and C5–6. Differential diagnosis needs to exclude TMD and trigeminal neuralgia, and dental examination is necessary to exclude dental conditions. Currently, management relies primarily upon medications used to treat neuralgia, as described in this report.

Authors' Contributions Both authors share the cases anonymously and contribute in the cases. Dr. Samim is the main author.

Compliance with Ethical Standards

Conflict of Interest The authors declare that they have no conflict of interest.

Ethical Approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee (REB).

Informed Consent Signed informed consent was obtained from all individual participants in the study.

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