Review

The whiplash injury phenomenon: a review of the literature

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Abstract

A single traumatic injury to the head, neck and temporomandibular joint that occurs during a sudden motor vehicle collision, is known as whiplash. In spite of the fact that whiplash injury is undefined and does not reflect the biomechanical events of motor vehicle accidents, temporomandibular symptoms may be associated with or occur independently of whiplash-associated disorders. The purpose of this review is to clarify whether a true correlation between temporomandibular joint (TMJ) dysfunction and whiplash injury exists. To this aim, a PubMed/Medline search was conducted using the terms “temporomandibular dysfunction”, “jaw pain”, “temporomandibular joint”, “whiplash”, “motor-vehicle accidents” and “motor-vehicle collisions”. Over 200 related articles were reviewed. The incidence of TMJ dysfunction resulting from whiplash varies from low to moderate and the mechanism of injury is poorly understood. Oral health care providers should be aware of the possible influence of litigation following motor vehicle accidents and its association with the so-called late whiplash syndrome. To date, there is no direct correlation between whiplash injury and TMJ dysfunction. The effect of whiplash on TMJ function is limited in terms of duration and often disappears without complications. A systematic and careful approach is needed when treating TMJ symptoms after whiplash and a differential diagnosis should be considered when temporomandibular joint disorder manifestations occur long after the whiplash incidence.

Keywords

Whiplash; Temporomandibular joint; Jaw pain

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1. Introduction

A single traumatic injury to the head, neck and temporomandibular joint that occurs during a sudden motor vehicle collision, is known as whiplash (TMJ). It is thought to be caused by whip-like motions of the head, neck and the lower jaw of the occupants of a car undergoing a sudden impact, typically a motor vehicle accident (MVA), where the front end of one vehicle (bullet vehicle) strikes the rear end of another vehicle (target vehicle).

The injury is not a result of the direct physical force or direct trauma to the occupants of the target vehicle does not cause the injury, but rather due to the indirect trauma released through acceleration/deceleration of the occupants’ head, neck and mandible. In spite of the fact that whiplash injury is unclear and does not reflect the biomechanical events of MVA, it seems to obtain recognition in pseudo-scientific speculations and facts regarding MVA. Additional confusion results from the fact that the diagnosis of TMJ whiplash seems to include all symptoms affecting the mandibular locomotor apparatus [1, 2].

Christensen & McKay [3] concluded that the concepts of the postulated motion of the head, neck and TMJ are unspecific, speculative and scientifically wrong. When the bullet vehicle impacts the target vehicle, the occupant of the target vehicle experiences excessive backward rotation of the head i.e. cervical hyperextension, followed by excessive forward rotation i.e. cervical hyperflexion. As the head undergoes backward rotation, the mandible is depressed and anterior hyper-translation of the mandibular condyles occurs. As the head undergoes forward rotation, the mandible is elevated and posterior hyper-translation of the mandibular condyles occurs. The final injury is thought to be an anterior displacement of the TMJ disc, and damage to the posterior attachment of TMJ disc [1, 2]. On the other hand, these events do not appear to affect the occupants of the bullet vehicle.

2. Review

To date, no documented jaw whiplash injuries that have been observed in real-life and in MVA-related experiments have been reported. The injury claims are based on speculations arising from a history of TMJ pain and TMJ magnetic resonance images (MRI). TMJ history must be demonstrated by scientific physical instrumental evidence and a distinction between subjective and objective biomedical observations. Objective scientific observations are made by means of accurate measuring instruments, while subjective pseudo-scientific observations are made by ones’ interpretation without the use of objective measuring instruments [4]. Differences between objective and subjective observations appear in clinical documentation of complaints on pain in the TMJ apparatus i.e. joints and muscles. These facts lead to several diagnoses and classifications based on pseudoscientific measurements [5–7].
Any diagnostic method that based on subjective pain is open to criticism because pain and other subjective symptoms are defined by their subjective properties, and there is no necessary correlation between objective and subjective observations [4]. Pain represents a subjective and psychological state rather than expression of physiological stimulation. Some authors utilize reports on pain that are based on mailed questionnaires using analog scales to record pain intensity, as evidence of the subjective complaints [7]. Only appropriate neuroimaging, electrophysiological and neuroanatomical techniques can reveal the nerve action potentials and neural pathways [8]. Any biomedical diagnosis or classification of any somatic disease or dysfunction must rely on independent and objective diagnostic techniques, and not only on patients’ subjective complaints and recollections. Physical events of MVA, usually rear end collisions, occur within a second, and the occupants’ recollections of his bodily movements do not always reflect the actual events [9].

Some clinicians invoke the patient’s history of whiplash injury as circumstantial evidence of cause and effect between somatic TMJ dysfunction and MVA[1]. An estimated 40% of subjects involved in MVAs will complain of TMJ dysfunctions[1, 10]. On the other hand, some clinicians have opposing conclusions, i.e. there is no correlation between MVA and TMJ dysfunction[11, 12]. These studies found that 95% of patients involved in MVA did not display any TMJ diseases/dysfunction. The relative risk of the absence of TMJ symptom is 19-fold the relative risk of the presence of TMJ symptoms[13].

According to a survey of 172 patients with TMJ dysfunction, litigating patients complain of more severe symptoms than non-litigating patients[14]. Therefore, the symptomatology of litigating patients contains a pronounced mendacious component. Therefore, it is scientifically wrong to conclude that anamnestic and symptomatic data can constitute the only evidence for any valid and objective diagnosis or classification of somatic TMJ dysfunction/diseases.

Clinical reports that rely on anamnestic and or symptomatic data in order to establish cause or effect relationship between somatic TMJ dysfunction and MVAs are unreliable. A study conducted on 180 accident victims in Greece reported that four weeks following the accident, victims reported neck pain, headache, shoulder pain and dizziness[15]. However, following 4 weeks from the accident, more than 90% had recovered from these symptoms and returned to pre-accident state of health. There were no cases of chronic disability. The question arises why in Greece, acute whiplash injury symptoms are self-limiting and brief and do not turn into late whiplash syndrome. Another study conducted on 210 victims of MVA in Lithuania reported that 2.4% of subjects suffered from jaw pain for 1 day per month, compared with 3.3% of the control patients[16]. A low prevalence of jaw sounds, pain, jaw locking, tinnitus and facial pain was found in both groups. Unlike whiplash affected individuals in Western societies, in Lithuania, accident victims with acute whiplash injury do not report chronic symptoms of TMJ dysfunction.

Acute whiplash injury symptoms are self-limiting, and brief when there is no preconceived notion of chronic pain or long term disability arising from MVA, and no involvement of the therapeutic community, insurance companies or litigation[17]. In a study conducted on 19 acute whiplash patients and 20 control patients, Kasch et al., concluded that TMJ dysfunction after whiplash injury is rare and that whiplash is not a major risk factor for the development of TMJ dysfunction[18]. This study suggests. In a prospective study on sixty patients after near-end collision, Bergman et al., found that there was no significant increase in the incidence of disc displacement, joint effusion or any other injury to the TMJ after a whiplash trauma that could be revealed by MRI[19].

McLean at al., investigated 948 European Americans that arrived at the emergency room within hours after a motor vehicle collision (MVC)[20]. Six weeks later, the authors interviewed these patients and they were questioned about their pain symptoms, and whether they were involved in MVC-related litigation. The presence of neck pain and widespread pain six weeks after MVC was compared between those engaged in litigation (litigants) and non-litigation. The authors reported that the litigants were less educated and had more severe neck pain, overall pain and greater extent of pain at the time of evaluation. Furthermore, the predictor among individuals engaged in litigation differed from individuals not engaged in litigation. Pain outcomes among litigants are more strongly influenced by socioeconomic factors and less influenced by initial pain. Thus, monetary gain following litigation, may influence pain persistence or the worsening of pain.

The impact of ongoing litigation was investigated in 35 post-MVC patients compared to 19 non-litigation patients. Differences that were significant were observed in litigation vs non-litigation patients: earache (72% vs. 42%), TMJ noises (97% vs. 65%), headache (97% vs. 79%) and dizziness (82% vs. 58%). A greater number of complaints were noted by litigating patients (15 vs. 7).

Ferrari and Russell claimed that acute whiplash injury in whiplash-associated disorders Grade 1 and Grade 2 is in most cases a minor sprain[21, 22]. They present a model including several psychological and referral factors that lead some patients to report chronic symptoms, which they attribute to the accident[21]. If TMD symptoms arise from acute injury to the TMJ with acute whiplash injury (in the absence of indirect jaw impact), one would expect to find a relatively similar relationship between TMJ dysfunction symptoms and MVAs across the globe. For example, despite high whiplash claims in Norway, patients hardly ever report TMJ symptoms[23]. Similarly, in Australia, TMJ dysfunction claims are rare even though there is a high frequency of whiplash claims[24]. In North America, medical and social customs may be an important factor in the incidence of jaw pain in patients with whiplash. While reports of TMJ dysfunction are common in some regions[25], Heitz[26], found regions in the US where the phenomenon of reporting chronic TMJ dysfunction symptoms after MVA was virtually non-existent.

3. Discussion

Along decades, researchers claimed a relationship between whiplash and TMJ dysfunction, without verifying these claims[27–29]. Howard claimed that injury to the TMJ associated with extension-flexion motion may be induced by myospasm[30]. Based on principals of physics, extension-flexion movements do not produce forces in a direction or magnitude that would have a pathologic effect on the TMJ, or the masticatory muscles. No relationship between masticatory muscles myospasm and extension-flexion maneuvers occur that would have a greater potential to induce spasm than routinely experienced joint forces. Stress may cause myospasm that may be a major causative factor of TMJ dysfunction and myofascial pain syndromes. Howard and colleagues used high speed photography, dual plates attached to the upper and lower jaws, (with electronic signals) and monitored muscle activity to test for relaxation or contraction before, during and after the impact to mimic acute neck
injury[31, 32]. They found that none of the test subjects experienced TMJ dysfunction or related craniomandibular symptoms.

Christensen and McKay concluded that the theory of mandibular whiplash cannot conform to any known principles in anatomy, physiology or physics[33]. The behavior of patients with TMD is not consistent with joint inflammation that follows joint trauma. To associate injury to TMJ and whiplash, some researchers attempted to show a link between reported TMJ dysfunction symptoms and imaging abnormalities. These researchers presume that the patient’s memory is accurate regarding the absence of pre-accident symptoms. This assumption is inaccurate as seen by Marshall who found that MVA victims tend to forget their pre-accident pain[34].

Many clinicians tend to correlate TMJ dysfunction symptoms and abnormal imaging facts. However, TMJ abnormalities in asymptomatic populations are also prevalent[35, 36]. Muir and Gross recommend to be careful not to correlate the significance of radiologic abnormalities in patients with pain in TMJ[37]. Interestingly, Drace and Enzmann showed a significant prevalence of abnormalities on MRI of asymptomatic, healthy individuals[38]. Thus, there is a probability that an imaging abnormality existed, prior to the accident, in a patient that experienced whiplash. Castro and colleagues concluded that morphological and anatomic signs of injury to the cervical spine cannot be demonstrated up to a speed gauge of 10 and 15 km/h[39]. From preliminary results of the ongoing motion analysis it can already be concluded that hyperextension to the cervical spine does not occur in rear-end automobile collision involving velocity changes up to 15 km/h if headrests are installed[40]. In a study comparing post MVA patients with non-trauma TMJ dysfunction patients (control group), Grushka and colleagues reported that post MVA patients complained more of earache, neck, shoulder, back and extremities’ pain, poor sleep and stress than those in the control group[41]. Furthermore, MVA patients may be involved in dysfunction or dysregulation of central pain-modulating system and neuropsychological and cognitive changes. Evidence that central pain mechanisms play a role in chronic pain after MVA is shown in a study where poor recovery after injury was associated with reduced cold pressor pain tolerance and increased peak pain[42]. This theory is supported by a study of TMJ dysfunction following trauma compared with non-trauma TMJ dysfunction patients. Patients that experienced trauma were slower in terms of simple and complex reaction times and poorer on neuropsychological tests.43 The presence of a regional and widespread systemic disorders combined with psychological distress, is detrimental to the prognosis and should be considered in management, requiring a multidisciplinary approach.

4. Conclusion

The effects of whiplash on the development of TMJ dysfunction is unclear. TMJ dysfunction resulting from whiplash varies from low to moderate and the mechanism of injury remains poorly understood. The incidence of TMJ pain and clicking after whiplash injury is extremely low. Patients who did not experience clicking on resolution of their initial pain dysfunction do not develop this side-effect[26]. As stated by Obelienine and colleagues, when there is no preconceived notion of chronic pain or long term disability, and no involvement of the therapeutic community, insurance companies or litigation, the symptoms after an acute whiplash injury are brief, self-limiting, and do not evolve to the so-called late whiplash syndrome[17]. Therefore, to date, there is no direct correlation between whiplash injury and temporomandibular joint dysfunction. A systematic and careful approach is needed when treating TMJ symptoms after whiplash and a differential diagnosis should be considered when TMJ dysfunction manifestations occur long after the whiplash incidence. Future studies are needed to further validate the influence of psychosocial and economic factors on TMJ pain after whiplash.

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Conflict of Interest

The authors declare no competing interests.

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