Orofacial Injuries Due to Trauma Following Motor Vehicle Collisions: Part 2. Temporomandibular Disorders

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ABSTRACT

Temporomandibular disorders (TMDs) following motor vehicle collisions (MVCs) may result from direct orofacial trauma but also occur in patients with whiplash-associated disorder (WAD) without such trauma. TMDs may not be identified at the time of first assessment, but may develop weeks or more after the MVC. TMDs in WAD appear to occur predominantly in females and can be associated with regional or widespread pain. TMDs following MVC may respond poorly to independent therapy and may be best managed using multidisciplinary approaches.

Motor vehicle collisions (MVCs) may cause symptoms associated with temporomandibular disorders (TMDs) resulting in patients presenting to dental offices. Historically, there has been controversy over TMDs following MVCs. TMDs are “a collective term that embraces a number of clinical problems that involve the masticatory muscles, the TMJ [temporomandibular joint], and associated structures.” In 1992, a review showed a relationship between MVCs and TMDs, facial pain and headache. This article reviews literature published since then assessing TMDs in post-MVC patients, most of which strongly supports the association.

Whiplash-associated disorders (WADs) are a range of injuries caused by or related to a sudden distortion of the neck. WADs are commonly associated with MVCs, usually rear-end collisions, but they may also result from front or side impacts. WADs occur in approximately a third of all MVCs and are the most common motor vehicle injury treated in emergency rooms in the United States. In the United States, 677 cases per 100,000 population were reported annually, while in Canada, using the Saskatchewan Government Insurance database, WADs represented 83% of accident claims with an annual incidence of 67 visits per 100,000 people.

TMDs may be associated with WADs and include jaw pain or dysfunction in addition to headache, dizziness, hearing disturbances, neck pain and dysfunction following an MVC. Reduced or painful jaw movement may also occur in patients with WAD.

Direct or indirect trauma from MVCs has been associated with musculoskeletal pain in the head and neck (including TMDs), as well as other phenomena such as headache and neuropathic pains. Dentists have an important role in the recognition, diagnosis and management of injuries and pain following motor-vehicle-related trauma, ultimately to the benefit of their patients.
Assessing Patients with TMD Symptoms

To determine whether a patient who has been in a collision has a TMD, an appropriate history and examination of the head and neck should be undertaken, supplemented by diagnostic imaging, if necessary (e.g., pannomograph for screening bony abnormalities, cone beam computed tomography [CT] for more detailed bony assessment, magnetic resonance imaging for soft tissue abnormalities). The history should include questions related to pain in the TMJ and masticatory muscle areas (and other areas of the head, as well as the neck), TMJ sounds (e.g., clicking, crepitus) and catching or locking of the jaws with opening or closing.

Examination should include extraoral and intraoral palpation of masticatory muscles and the TMJs for symmetry, tenderness/pain, clicking or crepitus; observation of any deviation of the mandible on opening or closing; and range of mandibular movements. Cranial nerve function and the neck should be evaluated, including ranges of motion and sites of tenderness in the cervical musculature. Various TMD classification schemes are available to allow specific diagnoses based on the history and examination findings (e.g., Research Diagnostic Criteria for Temporomandibular Disorders).15

If the patient was seen before the MVC, it should be determined whether TMDs were present at that time and whether any changes in the condition occurred following the MVC (e.g., pain worsened or new symptoms, including joint sounds, limited jaw opening, jaw locking, etc.). If the patient has new symptoms of TMDs—additional symptoms or increased severity of prior TMDs—following an MVC, the potential relation to the MVC must be assessed comprehensively.

Delayed Diagnosis of WAD

There appears to be a risk of delayed onset of TMDs following an MVC. In a Swedish study, 60 consecutive WAD patients following MVC were compared with matched controls.16 The incidence of new symptoms of TMDs among these patients was 5 times that in the control group and was higher in females than males. TMDs were reported as the primary complaint by 5% of the patients at the first visit and by 19% at 1-year follow-up; no significant increase was seen among the control group. Pain began with the trauma in 7%, and more reported TMD at follow-up.

Delayed onset of new symptoms of TMDs was seen in a third of WAD patients with TMDs versus 7% of the control group, and TMDs were the primary complaint of 20% of WAD patients at 1 year. Painful jaw clicking had developed by follow-up in 19% of WAD patients, only 1 of whom had this symptom before the MVC, and painful locking developed in 14%, with only 1 patient having this symptom before the accident. The potential delay in onset of TMDs following an MVC raises concerns about diagnosis, prognosis, management and medico-legal issues.16

In contrast, some studies of limited sample size have reported that TMDs are not commonly identified following MVCs early following and up to 1-year post-MVC.17-19

Regional and Widespread Symptoms

Regional and widespread physical symptoms as well as psychological disturbances are common in MVC patients. Such somatic symptoms may be caused by air bag deployment, which is associated with a variety of injuries, including injury to the TMJ,20 maxillofacial fractures,21 burns,22 injuries to eyes,23 injuries to ears,24 paresis,25 neuropathic facial pain,26 basal skull fractures,27 transection of the internal carotid artery,28 atlanto-occipital dislocation29 and spinal cord injuries.30 Complaints of pain, stiffness and numbness of the jaw and face have been associated with WAD.31

In a study comparing 54 post-MVC patients with 82 nontrauma TMD patients (control group), post-MVC patients complained more of orofacial pain than the control group, who reported more jaw-joint sounds.32 Post-MVC patients had significantly more complaints of earache and stuffiness; neck, shoulder and back complaints; numbness or pain in extremities; headache; jaw pain on waking; facial pain; poor sleep; dizziness and stress than those in the control group.32

Another study documented greater pain reaction to palpation of masticatory muscles in post-MVC patients than nontrauma TMD patients.33 This agrees with other studies reporting a higher frequency of TMD pain and increased psychologic distress among WAD patients.11,17 Furthermore, hearing and vestibular complaints have been associated with WAD.34 Mild traumatic brain injury has also been documented in patients with WAD experiencing possible or documented loss of consciousness.7

Besides localized trauma to structural elements of the stomatognathic system (peripherally mediated and maintained pain), regional and widespread symptoms may be due to dysfunction or dysregulation of central pain-modulating systems and regional or widespread pain input involving neuropsychological and cognitive changes (centrally mediated and maintained pain). Evidence that central pain mechanisms play a role in chronic pain after MVC is shown in a study where poor recovery after injury was associated with reduced cold pressor pain tolerance and increased peak pain.35 Altered nociceptive input and central processing in WAD patients has also been reported in experimental pain studies.35,36 These findings
are supported by a study of TMDs following trauma compared with idiopathic nontrauma TMDs; patients in the former group were slower in terms of simple and complex reaction times and poorer on neuropsychological tests.39

The psychological consequences associated with MVCs contribute to the complexity of these patients as witnessed in a study comparing clinical and psychological characteristics of 34 TMD patients with trauma history (24 due to MVC) with 340 TMD patients without trauma history. TMD patients with trauma history displayed more severe subjective, objective and psychological dysfunction than those without trauma history.37

Clearly, the presence of a regional and widespread systemic disorder and psychological distress has a negative impact on prognosis and should be considered in management, which often requires a comprehensive multidisciplinary approach.

Prognosis of WAD and related TMDs

Approximately 15–40% of patients with acute WAD develop chronic symptoms.38,39 Chronic WAD represents a physical, medical, economic, and psychosocial problem. Severe neck pain, self-reporting of poor general health and stress response at initial evaluation post-MVC have been associated with increased risk of persistent pain, neck disability and ability to work.40 Furthermore, depressive symptoms have an impact on pain and passive coping has been associated with slower recovery.41

Specifically assessing post-collision TMDs, Kolbinson and others42 examined 50 such patients and compared them with 50 matched nontrauma-induced TMD controls. Post-trauma TMD patients reported more severe facial pain, neck pain, earache and headache as well as sleep disturbances. Examination confirmed greater tenderness in the masticatory muscles, neck muscles and TMJ in the trauma group. Greater impact on work and recreational activities was also reported for trauma patients. Post-trauma TMD patients received more types of treatment and more medications (including analgesics, muscle relaxants and antidepressants), had more health care visits, were treated over a longer period and had poorer outcomes.43

Another study confirmed that post-MVC TMD patients do not respond to management and require more treatment compared with nontrauma cases.44 Other studies have demonstrated similar findings, e.g., TMDs that develop post-MVC have a less favourable prognosis and affect quality of life. However, most patients who develop TMDs following an MVC often improve with time or with standard therapy. This was confirmed in a study that suggested that the response of post-trauma TMD patients to conservative therapy was similar to that of nontrauma TMD patients, although the post-trauma group continued to require analgesics, suggesting persistence of pain.45

In contrast, comparing 2 groups of TMD patients—one without a history of trauma to the head and neck (302 patients) and the other with a history of trauma that was linked to the onset of symptoms (98 patients)—the trauma group’s symptoms were more pronounced initially, but both groups responded equally well to conservative treatment when evaluated with the Helkimo dysfunction index after 1 year.46 Despite these findings, a minority of individuals develop a chronic condition that may reflect the more complex nature of regional and widespread pain, which may be the result of central hypersensitivity mechanisms47,48 or possible mild traumatic brain injury.

Approach to Management

Because of the regional and potentially widespread nature of their pain, patients with post-MVC TMDs should be managed following general principles with physical medicine, physical therapy and behavioural medicine and employing physical therapies and directed medications for musculoskeletal and chronic pain.2,9,32,33,43,44

In a population-based mail-out survey of 2000 adults, researchers noted that respondents had more negative beliefs about pain associated with WAD than other non-MVC caused pain; active coping strategies (activity and exercise) were reported as important for recovery by 55% of the WAD patients.49 WAD patients reported greater pessimism regarding return to usual activities, which may affect outcome.

A review of 36 randomized clinical trials of adults with WAD, with or without headache, may have implications for patients with TMDs and WAD.50 For acute WAD, in 1 trial, prednisone taken within hours of injury reduced pain at 1 week, but not at 6 months compared with placebo. For chronic symptoms, intramuscular lidocaine was superior to placebo and dry needling and similar to ultrasound. Myofascial trigger point injection was found to be effective, but no difference was documented between saline and botulinum toxin used as the active agent. Muscle relaxants and analgesics had limited evidence of effect.

In a study involving 55 patients with TMDs and WAD, there were no differences between those who were instructed in jaw exercises and those who received no treatment at 3- and 6-month follow-up.31 Clearly, prospective, randomized controlled trials with an adequate number of patients and using appropriate measurement
methods are greatly needed to enhance our knowledge of management approaches.

Conclusion

Although the etiology may be speculative, TMDs have been clearly documented following an MVC involving direct orofacial trauma and in a subset of WAD patients where no direct orofacial trauma is recognized. TMDs may not necessarily be diagnosed during a first assessment, but may manifest weeks or months after an MVC. TMDs in WAD are more common in females and can be associated with regional or widespread pain that may reflect central, systemic and psychological effects. Therefore, TMDs may represent a component of a WAD symptom cluster.

These findings suggest that multidisciplinary dental and medical management is necessary in many patients and that TMDs in these situations should not be interpreted as separate, independent conditions. Dentists should provide appropriate conservative, reversible forms of TMD management as part of a multidisciplinary team approach.

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References


