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Trauma and work-related pain syndromes: Risk factors, clinical picture, insurance and law interventions

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In the past decade, major progress has been made in our understanding of fibromyalgia syndrome (FMS). Various triggers have been implicated as contributing to symptom development in FMS when genetically susceptible individuals are challenged.

A substantial amount of data points towards the association between trauma and chronic widespread pain/fibromyalgia syndrome (CWP/FMS). There is abundant data suggesting that the pathogenesis of CWP/FMS might be related to cervical spine injury. Furthermore, several persistent local pain conditions may progress to CWP/FMS. These conditions may share a common pathogenic mechanism namely, central sensitisation. Physical trauma and emotional trauma co-exist in many traumatic events and may interact in the pathogenesis of CWP/FMS.

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Fibromyalgia syndrome (FMS) is the prototypical central nervous system sensitisation syndrome [1,2], and causes significant impairment in functioning and quality of life [3].

FMS is characterised by increased pain processing, clinically leading to manifestations such as allodynia and hyperalgesia [4,5].

Recent evidence suggests that genetic factors may play a role in the pathogenesis of FMS [6–8].

Various triggers have been implicated as contributing to symptom development in FMS, when genetically susceptible individuals are adequately challenged [9].

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It is within this context that both physical trauma and emotional stress or distresses have been previously investigated regarding their role in the investigation of chronic pain and FMS.

Wolfe [10] reported in 1986 that 24% of fibrositis patients cited trauma and 14% cited “stress” or “emotions” as the factor that “caused” the onset of the disease.

Wolfe [11] reported, as well, a case of FMS developing following a workplace injury, but in which the issues of compensation and work disability were not relevant. He concluded that this case report, narrated by the patient, suggests that there is such an entity as post-traumatic fibromyalgia, and that central nervous system, plasticity places a central role [11]. During the years, a substantial amount of data has been gathered, pointing towards the association between trauma and FMS [12–16].

Trauma, chronic pain and FMS

Twenty-nine of 127 patients (23%) with FMS reported having trauma, surgery or a medical illness before the onset of FMS, and were classified as having reactive fibromyalgia [17].

Waylonis and Perkins [18] provided a follow-up study of patients with post-traumatic fibromyalgia. A total of 60.7% noted the onset of symptoms after a motor vehicle accident (MVA) and 12.5% after a work injury.

Buskila et al. [12] assessed 102 patients with neck injury and 59 patients with leg fractures (control group) for non-articular tenderness and presence of FMS. FMS was 13 times more frequent following neck injury than following lower-extremity injury. FMS was noted at mean of 3.2 months (SD 1.1) after the trauma [12].

Approximately 70% of 238 patients diagnosed with diffuse/specific myofascial pain syndrome attributed the beginning of their chronic pain to some form of trauma [19].

Al-Alaf et al. [13] provided a case-control study examining the role of physical trauma in the onset of FMS.

Approximately one-third (39%) of FMS patients reported significant physical trauma in the 6 months before the onset of their disease, compared with only 24% of controls. It was concluded that physical trauma in the preceding 6 months is significantly associated with the onset of FMS [13].

A wide variety of operations have been shown to be associated with chronic pain syndromes [20].

A 2-year prospective study among cohorts of newly employed workers demonstrated that the prevalence of new-onset widespread pain was high but among this young, newly employed workforce, both physical and psychosocial factors played an important role [21].

McLean et al. [22] assessed currently available evidence regarding the ability of a motor vehicle collision (MVC) to trigger the development of FMS. They used consensus standards developed by the American College of Rheumatology Environmental Disease Study Group to assess the ability of an MVC to trigger FMS.

They concluded that the evidence that MVC trauma may trigger FMS meets established criteria for determining causality, and has a number of important implications, both for patient care, and for research into the pathophysiology and treatment of these disorders [22].

McLean et al. [23] proposed a model in which the acute physical and emotional effects of MVC involve an interaction between the direct effects of tissue injury and the emotional responses to the experimental threat.

Tishler et al. [24] reported that whiplash injury and road-accident trauma were not associated with an increased rate of FMS after more than 14.5 months of follow-up. However, in this study, the prevalence of diffuse pain in the subjects, who were diagnosed as experiencing whiplash injury, was 0.6%; this is 20 times less than the prevalence of chronic widespread pain (CWP) in the general population, approximately 10% in different studies including those from Israel, even without trauma [25,26]. No data were presented on ‘whiplash-associated symptoms’, such as neck pain, pain radiating to the chest or arm as well as the severity of these symptoms.

Wynne-Jones et al. [27] reported that the rate of onset of widespread pain after a motor vehicle crash is low and, at most, there is a modest increase in risk, particularly after adjusting for levels of psychological distress. The authors stress, however, that the number of new-onset pain episodes was small, a fact that limits the power of the study. Furthermore, just over 50% of the participants returned

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