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Whiplash associated disorder:

a review of current pain concepts

Following traumatic injury, the clinician is sometimes exposed to an apparent discrepancy between the magnitude or duration of pain described by patients and the magnitude of the objective physical injury. In the medico-legal sphere this discrepancy takes on greater importance, with the legal system requiring comment on the biological plausibility, treatment and prognosis for such conditions.

Whiplash-associated disorder (WAD) is a frequently encountered example of a medical condition where often there is an apparent disconnect between magnitude of injury and magnitude of disability. In this article we will review some of the current concepts that have been used to explain the pain and disability associated with WAD. We will also consider the biological mechanisms which may underlie these symptoms in order to propose rational treatment approaches to address this complex and multidimensional malady.

WHIPLASH-ASSOCIATED DISORDER

The Quebec Task Force on Whiplash-Associated Disorders defined whiplash as "an acceleration-deceleration mechanism of energy transferred to the neck that results in soft tissue injury that may lead to a variety of clinical manifestations including neck pain and its associated symptoms." That task force also coined the term "whiplash-associated disorders" (WAD) to describe the clinical entities related to the injury, and to distinguish them from the injury mechanism. The term 'whiplash' on its own should be used to refer to the acute, localised neck pain resulting from an acceleration/deceleration mechanism. Estimates vary widely, but authors report that about 30% to 50% of patients who sustain a symptomatic whiplash injury go on to report chronic, and potentially more widespread symptoms, which may be termed WAD.

A BRIEF REVIEW OF PAIN PERCEPTION: FROM NOCICEPTOR TO DISABILITY

Acute pain will typically be initiated peripherally through the activation of nerve fibres sensitive to noxious stimuli (nociceptors). From that point, to pain perception, interpretation and disability, there exists a complex neurochemical cascade involving both the peripheral and central nervous systems. Extensive reviews of these mechanisms have been written[1,2] but the main processes affecting pain perception are summarised in Figure 1.

From here, the process of disability and behaviour flowing from the perceived pain is influenced by a number of psychosocial constructs including beliefs, previous experiences or, potentially, the presence of an ongoing litigation process.

One key element of this process, relevant to the musculoskeletal medical expert, is the concept of central sensitisation (CS).

CS is a process of enhanced excitability of the neurons within the central nervous system in response to peripheral nociceptor stimulation.[3,4] These changes can remain long after nociceptive input has disappeared. In fact, in certain situations it is possible for either no, or minimal, tissue damage to induce pain perception in the centrally sensitised system. This may explain the discrepancy between the absence of evident tissue damage and persisting pain complaints in a number of painful musculoskeletal conditions including fibromyalgia and chronic WAD.

CS encompasses altered sensory processing in the brain, malfunctioning of descending pain inhibitory mechanisms, increased activity of pain facilitatory pathways and, ultimately lowered thresholds for peripheral inputs to be propagated to the point of consciousness.

There is good evidence for the presence of CS in musculoskeletal diseases other than WAD.[5-7] As such, it is perhaps surprising that there is seldom a documented appreciation of CS in WAD when assessing in the medico-legal arena.

The physical evidence for CS has been shown to be present both in the clinical and laboratory settings. Features identified include local and widespread hyperalgesia, referred pain and allodynia. These may be assessed using simple bedside testing. Others, such as decreased spinal reflex thresholds, inefficient diffuse noxious inhibitory controls activation and enhanced temporal summation of pain represent useful research tools.

HOW MIGHT YOU PROVE THE PRESENCE OF CS IN YOUR PATIENT?

The clinician should be alerted to the possibility of central sensitisation when pain persists beyond the typical period for physical healing, or where pain experience is outwith the normal response witnessed by the expert in clinical practice. Authors have proposed an algorithm to differentiate CS from neuropathic pain or nociceptive pain. The three elements for assessment are:

- Pain that is disproportionate to the injury
- A widespread distribution beyond the injury
- The use of the Central Sensitisation Inventory, a questionnaire designed for this population. Thresholds exist for differentiating the various pain drivers.

A number of other signs and symptoms can be present in a centrally sensitised state. Some of the common problems that have been described include sleep disturbance, altered body sense, changes in movement patterns, altered sensation and dyskinaesthesia. Some of these features lack the objectivity that is preferred by the musculoskeletal expert in the medico-legal arena and hence are rarely assessed or recorded.

Patients/claimants with persisting pain also tell of mood changes, emotional distress, difficulty with concentration, lethargy and feeling unwell. The multidimensional features of

persisting pain, and in particular WAD, have proven to be manifestations of the interrelated workings of many body systems including the immune system, endocrine system, nervous system and autonomic nervous system. The feelings of being unwell after a whiplash injury, as well as in other chronic pain states, have been attributed to these interrelated systems.[10]

CAN THE PRESENCE OF CS EXPLAIN THE COMMON CLINICAL FINDINGS IN WAD INCLUDING TRIGGER POINTS?

The common findings in WAD include neck pain and stiffness, dizziness, headaches and a general malaise. The symptoms can develop rapidly after the injury but are recognised to sometimes appear hours or days after the causative event.

Typically, the pain is the key reason why treatment is sought. Pain following a whiplash injury is often widespread, extending beyond the area of initial injury. One common feature in this context is the development of so-called 'trigger points'. Trigger points are manifested as discrete areas of tenderness, often associated with palpable differences in muscle compliance.

Some authors have proposed that trigger points are manifestations of central sensitisation.[8] Certainly, their presence should alert the examining expert to the presence of CNS sensitisation, although there is a recognised variability in identifying trigger points on examination.[9] Plausible biological mechanisms involving neurogenic inflammation and secondary allodynia have been proposed to explain the presence and cause of trigger points in relation to CS.[9]

REVIEW OF THE CURRENT MULTIMODAL WAYS TO REDUCE THE SYMPTOMS OF WAD

Multimodal treatment, a combination of education, exercise and manual therapy, is likely to be the most effective way to reduce the symptoms of WAD in both the early stages and in a persisting state.[11,12]

Considering that the risk factors for poor recovery include post injury pain, the number and severity of symptoms, a Neck Disability Index (NDI) score of more than 40%, catastrophising and post-traumatic stress symptoms (13), this would appear to be the logical approach.

Several authors agree that in the acute stages of WAD, education and advice are effective measures[14-16] and perhaps more so than longer courses of treatment. Expectations are known to influence the treatment efficacy for pain[5] and in the case of WAD, Ferrari (2014)[17] suggests that the expectations about recovery predict the outcome. Considering these findings, it is reasonable to suggest that the early messages given to the patient set the scene for subsequent treatment and hence impact upon recovery outcome.

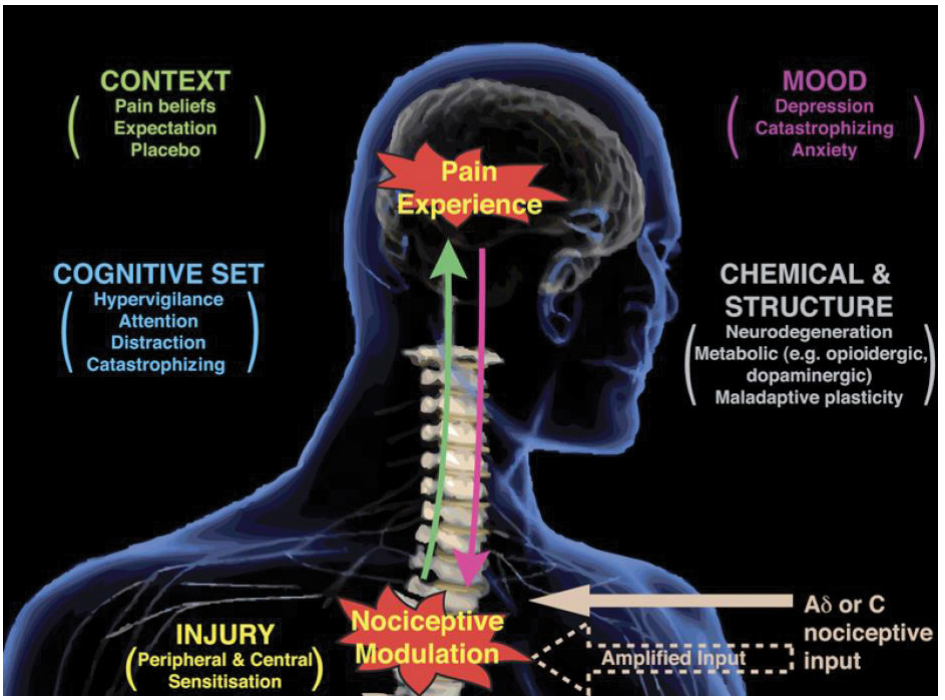

Fig. 1 Key factors that influence nociceptive inputs to affect pain perception.

Seeking the best outcomes for patients balanced with reasonable costs, the evidence points toward education, advice and exercise as the primary components of a treatment programme. WAD patients are currently in receipt of a range of adjunctive therapies, which depend upon the practitioner type, despite this evidence. Creating a template for treatment programmes would be a useful step towards ensuring that WAD patients receive the same evidence-based care. The programme duration and the number of sessions also need consideration, with the data suggesting better outcomes with fewer visits.[9] We can speculate that one of the reasons for this finding is the possibility that ongoing sessions lead to increasing dependence and passive treatments (e.g. mobilisation, massage, acupuncture) when the patient holds a belief that they will bring about recovery instead of moving towards active, self-directed treatment.

Considering the need for active engagement alongside the evidence that advice and education are important in acute and chronic cases,[14,16] we can confidently create an evidence-based programme that begins with pain and WAD education, self-management advice that includes exercises (movement), activity management and a short course of manual therapy, all of which is explained to the patient to ensure that expectations are aligned with the reality of what is known and understood by the condition. Naturally, the knowledge base will change as further studies are undertaken, and the guidelines will be updated accordingly.

SUMMARY

WAD is a clinical malady with a complex interplay between the acute injury, evolution into chronicity and maintenance of perceived pain and associated disability. While this article gives an insight into the mechanisms which may underlie pain perception in WAD it is clear that not all of these elements are in the expert sphere of the majority of orthopaedic professionals. For the musculoskeletal medico-legal expert, our opinion is often reduced to binary decisions – was a physical injury there? Is there a physical injury now? Was the initial physical injury caused by the accident?

It is clear that the majority of post-traumatic CS processes begin with a peripheral nociceptive stimulus. In this regard the confirmation of probable physical injury at the time of injury seems within the musculoskeletal remit. Similarly, the subsequent disability and limitations of physical activity through examination or investigation is within the expert sphere of the

orthopaedic professional.

Clinical experience and extensive research suggests it is futile to look for a 'thing' in WAD which is 'broken' to explain chronic symptoms. We propose that the identification of symptoms and signs suggestive of a process of central sensitisation may be a useful adjunct to physical examination. Findings such as cognitive deficits, sleep disturbance and poor tactile discrimination[19] are recognised associated features which patients may not volunteer for fear of sounding implausible, but may guide the multidimensional treatment and therapies needed to comprehensively tackle the problems.

Treatment that might be expected to be of use in WAD must address both the peripheral and central drivers of pain within a biopsychosocial framework. The treatment must certainly engage the patient with the modern concepts of pain that include CS.[19] Recent work suggests that advice and education are effective treatments and should be employed at least in parallel with traditional therapy approaches.

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