with EDS HM type, the burden of which was compared with that of FM or rheumatoid arthritis. The EDS group reported similar physical and overall function, and better psychological function than the FM group, whereas their levels of pain severity, life interference, and affective distress were significantly lower (98).

In conclusion, JHS should be taken into account as a possible trigger of widespread pain or FM in susceptible subjects. Excessive or inappropriate physical activity, with the consequent hyperextension of the joint capsule and repeated micro-traumas of ligaments and muscles, could lead to the over-activation of pain receptors in the joints and, by causing hypersensitivity and amplifying pain signals, could generate more widespread pain or trigger the typical symptoms of FM.

THERAPEUTIC APPROACHES

Treating the pain associated with FM and related conditions is a great challenge. Pain is the most frequent complaint of patients, especially those with the diseases described in this paper; clinicians are well aware of the fact that every treatment must take into account the associated symptoms, which need to be graded and prioritised. Various drug and non-drug therapies have proven positive effects on at least some CSS and, more generally, chronic pain.

Over the last few decades, substantial advances have been made in the treatment of these disorders, particularly for the pharmacological options, on the basis of our greater understanding of the pathophysiology of FM, our improved knowledge of CS mechanisms, and our ability to define symptom domains more precisely. Experts have pointed that a multidisciplinary approach is essential (99-103) as physicians need to bear in mind all of the potential comorbidities and overlapping conditions in order to avoid treatment failures and ensure that the most active problem is confronted in a timely and effective manner.

Well-designed and controlled clinical trials have shown that various classes of neuromodulatory agents can be helpful. Drugs that act on the noradrenergic and serotonergic pain pathways, or the ascending nociceptive pathways, and those that can improve other symptom domains such as fatigue, disturbed sleep and cognition, are available.

The treatment of FM is the most complete and extensively analysed, and the drugs that work well and have a high level of evidence in FM are also useful in some patients with other CSS. There is strong evidence in favour of dual re-uptake inhibitors such as tricyclic compounds (amitriptyline, cyclobenzaprine) and serotonin-norepinephrine reuptake inhibitors (milnacipram, duloxetine, venlafaxine) and for anticonvulsants (pregabalin and gabapentin); moderate evidence for tramadol, selective serotonin reuptake inhibitors, gamma hydroxybutyrate and dopamine agonists; and weak evidence for growth hormone, 5-hydroxytriptamine, tropisetron, S-adenosyl-L-methionin. There is no evidence in favour of opioids, corticosteroids, non-steroidal anti-inflammatory drugs (NSAIDs), or the benzodiazepines, and some data suggest that giving opioids to patients with central pain states might even worsen the pain (1, 104, 105).

The European League Against Rheumatism recommendations share this pharmacotherapeutic grading, but also strongly recommend tramadol and pramipexole and state the potential effectiveness of simple analgesics such as paracetamol and other weak opioids (99).

Ketamine, amitriptyline, and gabapentin are considered specifically effective in attenuating CS (8).

No pharmacological options have ever been beneficial in treating MPS, for which the deactivation of TP is the gold standard; muscle stretching and TP injections are by far the most effective means of relieving symptoms (50, 52). Analgesics and non-steroidal anti-inflammatory drugs can modulate the pain, but have no definite action on TP. Muscle relaxants are not useful (106).

Medication is the main method of managing the pain associated with TMJDs,

and the pharmacological options are not substantially different from those of other CSSs, particularly FM. Although many randomised and controlled clinical trials have been conducted to test these widely used drugs, a recent systematic review has shown that there is a lack of evidence in favour or against their use (75).

The presence of a clearer peripheral origin of pain and consequent potential peripheral sensitisation probably supports the use of non-narcotic analgesics, NSAIDs, and weak and strong opioids in TMJD and JHS, reserving strong opioids to the subset of patients who obtain no benefit from other therapies (107, 108).

Non-pharmacological therapies vary widely and it is often difficult to draw any conclusions concerning their value. However, physical therapy, the correction of abnormal postural or perpetuating factors, and different complementary and alternative medicines have often been suggested in order to improve the response to medication in a multidisciplinary manner and reduce the abnormal cycle of pain amplification and its related maladaptive and self-limiting behaviours (52, 85, 103, 109, 110).

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