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Chronic Pain and Sleep Disturbances: A Dangerous Coexistence

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Abstract

Chronic Pain (CP) new definition has brought benefits to address the complexity of this syndrome. Quality of Life (QoL) and Functional Recovery (FR) represent fundamental issues of clinical outcomes, which go beyond mere pain relief. Such a definition recognizes the bidirectional association between CP and SD. Patients who suffer from CP always have Sleep Disturbances (SD). These patients may feel agitated during the night and tired in the morning, leading to a significant decrease in personal productivity. The two-way relationship between poor sleep and pain operates to maintain and amplify sleep inadequacy and growth and intensification of pain. There is a great need to increase awareness on the contribution of sleep quality to the severity of functional impairment in CP patients. CP disorders are not a simple exacerbation of acute pain. The negative effect of pain and sleep problems on quality of life outlines the necessity for investigating how the two conditions are associated and how to structure treatments and prevention strategies.

Keywords: Chronic pain; Functional recovery; Cognitive function; Dementia; Analgesics

Description

The new definition of CP as a single disease entity has brought benefits in terms of definition of severity [1]. Now this issue is best described through a multidimensional framework, acknowledging both the multidimensional nature of pain and the evolving notion of pain as a biopsychosocial issue. Furthermore, it may address fundamental topics like QoL and FR, adding further emphasis to clinical outcomes which go beyond mere pain relief. During the past decades, QoL and FR have progressively emerged as two important goals of analgesic therapy. The biopsychosocial evolution of CP model induces us to consider functionality as an ensemble of factors, such as the ability to return to work, maintain cognitive function, and complete activities of daily living as well as the absence of mood and SD. Such a definition recognizes the bidirectional association between CP and SD [2], and redefines sleep quality as a new target of analgesic therapy. Poor sleep could lead to a significant decrease in personal productivity, playing a role in terms of financial and non-financial costs, becoming an important social and economic burden [3]. Sleep quality plays a key role in cognitive and emotional functioning, representing a risk factor for obesity, cardiovascular disease, diabetes, dementia, immune suppression, and mortality. Moreover, pain disorganizes sleep architecture, and disturbed and unrefreshing sleep increases spontaneous pain and lowers pain thresholds [4]. This two-way relationship between poor sleep and pain operates to maintain and amplify sleep inadequacy and growth and intensification of pain *via* a downward spiral, which perpetuates and amplifies itself over time. Epidemiology of CP demonstrates the role of sleep quality in the development of CP and vice versa. Notwithstanding this, at least theoretical, strong two-way relationship between CP an SD, little knowledge is available about the neurochemical determinants of this interplay and about the therapeutical strategies to break this vicious circle [5]. There is a great need to increase awareness on the contribution of sleep quality to the severity of functional impairment in CP patients. Furthermore, it is crucial to acquire knowledge of how pain intensity reduction may improve sleep quality as a result of analgesic drugs.

The close relationship between sleep and chronic pain

CP disorders are not a simple exacerbation of acute pain but require a switch of pain-processing mechanisms to a pathological state that may last indefinitely [6]. Opioid receptors are situated in several nuclei that actively control both sleep and pain. Many decades ago, it was hypothesized the potential involvement of the opioid system in sleep deprivation in inducing pain hypersensitivity [7]. Recently, the damage of inhibitory neuromodulation of pain has been demonstrated in many clinical pain disorders, with prominent sleep disturbance components [8-10]. Moreover, sleep deprivation imbalances endogenous opioid systems and decreases the analgesic efficacy of μ-opioid receptor agonists. Key neuroimaging findings in insomnia patients revealed overactivity in corticolimbic areas, controlled by monoaminergic (serotonergic and noradrenergic) pathways [11]. The corticolimbic brain areas primarily include the medial prefrontal cortex, amygdala, nucleus accumbens and hippocampus. These areas are also involved in the pathophysiology of certain CP conditions that appear to be aggravated in patients with sleeplessness. Clinical neuroimaging studies demonstrate a fundamental involvement of the corticolimbic system in the development, amplification and prognosis of chronic pain.

Effects of chronic pain and sleep disturbances coexistence

Patients with CP and SD are inclined to report a multiple overlapping of symptoms: insomnia, pain, depression, anxiety, low energy, and fatigue. Literature indicates that only about 10% of patients are mono-symptomatic, specifying that the vast majority of patients have multiple symptoms [12]. Patients with CP and SD are most likely to have multiple comorbidities, accompanied by negative affect and suicidal ideation. Pain catastrophizing has also been demonstrated to be a risk factor for SD and may secondarily influence pain through SD.

The role of opioid therapy to improve pain and sleep disturbances

A recent meta-analysis has shown that the pain-reliving and hypnotic properties of opioids do make them a good option for CP patients with concomitant SD [13]. Likewise, sleep improvement may be associated to adverse events and increased day-time sleepiness that could contrasts with concept of ameliorated sleep quality. A recent review examining the effects of opioids on sleep in CP demonstrated that improvement in sleep could be associated to daytime sleepiness and other adverse events like somnolence, sedation, drowsiness, and sleepiness [14]. In contrast, our recent analysis suggested that tapentadol combine significant pain relief and improved sleep quality, with good tolerability and absence of serious or severe adverse events. Additionally, there was evidence in favour of tapentadol when compared with oxycodone and oxymorphone [15].

Conclusion

Future trials need to be designed using validated outcome measures of sleep alongside pain. Moreover, there are scientific and clinical reasons to combine both self-reported and objective measures of sleep, including perceived sleep parameters such as sleep start latency, total sleep time, number and interval of awakening after sleep onset, sleep efficiency, and even subjective evaluations. Our findings offer the rationale for adopting sleep quality as a relevant outcome, complementary to pain relief in CP management.

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