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Active Pain in Diseases and Health Problems

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Abstract

Pain extent is a separate and distinct domain from intensity and refers to the overall number of body areas with pain. Research suggests that this pain domain may also be important to patient functioning. For example, Trait and colleagues found a significant association between pain extent and the tendency of patients to report greater complaints of weakness, fatigue, and depression. Similarly, Toomey and colleagues reported that patients with more pain sites were more likely to report pain as having a greater negative impact in their functioning.

Keywords: Patients tendency; Significant associations; Work ability; Pain population; Psychological functioning; Demyelinating form

Introduction

Tarp and colleagues found that pain extent, along with pain intensity, was a significant predictor of pain-related disability in a sample of female patients with chronic facial pain. Patients with pain at multiple sites have shown a reduced level of health-related functioning, are more likely to have difficulties with mobility regardless of physical impairments than those with no pain or localized pain, and have worse prognosis for future work ability [1]. In a series of studies, Kamaleri and colleagues reported significant associations between pain extent and functioning in patients with musculoskeletal pain. They found a strong and linear association between increasing number of pain sites and decreasing functional ability; a strong relationship with decreasing psychological health, sleep quality, and overall health; and future work disability after a 14-year period. As with research on the importance of specific pain sites to patient functioning, it is unclear if these findings regarding pain extent replicate in other populations of individuals with chronic pain, including persons with slowly progressive NMD [2].

Discussion

Most published studies on these issues have been conducted with low back patients receiving treatment at secondary and tertiary care facilities. Thus, these findings may not generalize to other populations of patients with pain. Further delineating the relative importance of pain site and extent in relation to patient functioning is particularly important in patients with NMD, because research indicates they typically experience pain in more than one location [3]. Given what previous studies have found in other pain populations, the authors hypothesize that pain extent would be negatively associated with psychological functioning and positively associated with pain interference, whereas pain intensity in specific pain sites would show stronger associations with measures of patient functioning than pain at other sites. More specifically, one would expect that pain in the low back and arms might evidence stronger associations with pain interference and psychological functioning than pain at other sites [4]. There are intriguing differences among degrees of pain in the slowly progressive forms of NMD. It is not unexpected that neuropathic diseases like Charcot Marie Tooth would rank high in pain intensity, given the pathogenesis of the disease, particularly the demyelinating forms. However, it is clear that 2 of the most common forms muscular dystrophy, myotonic type 1, and facio-scapula-humeral, are also high on the list of painful NMDs. NIH-PA Author Manuscript NIH-PA Author Manuscript NIH-PA Author Manuscript Worldwide, DM1 and FSHD are the first and third most common forms of dystrophic myopathies, respectively, with the dystro-phinopathies coming in second. Both DM1 and FSHD are autosomal dominant, slowly progressive neuromuscular disorders. DM1 is caused by a polynucleotide triplet expansion located on the un-translated region of chromosome location results in a toxic gain of function of abnormally stored RNA in the nuclei of affected cells, leading to deregulation of RNA binding protein levels and mRNA splicing processes of multiple genes [5]. This action is presumed responsible for the multisystem features typical of DM1, with involvement of skeletal, cardiac, and smooth muscles, and the central nervous, endocrine, ocular, respiratory, and gastrointestinal systems to varying degrees. In FSHD, most patients possess a large deletion in the polymorphic D4Z4 macro-satellite repeat array at 4q35, presenting with up to 10 repeats, as opposed to 11–150 repeats in unaffected individuals. This situation is complicated by a nearly identical repeat array present. The remarkably similar sequence identity between these 2 arrays can cause difficulties in molecular diagnosis. Each 3.3-kb D4Z4 unit contains a DUX4 gene that is activated on contraction of the 4q35 repeat array via induction of chromatin remodelling [6]. Myofiber synthesis of both DUX4 transcripts and protein causes significant cell toxicity. As a transcription factor, DUX4 may target several genes, resulting in cellular deregulation with inhibition of myogenesis, muscle degradation, and oxidative stress. Prior studies indicate that as many as nearly 90% and 70% of patients with FSHD and DM1 report pain, respectively. In addition to identifying pain as a major problem for patients with either of these NDMs, these studies also indicate that pain is more common in patients with FSHD versus DM1. The average severity of pain in patients with FSHD is less than that reported by patients with DM1.The reasons these disease in particular have more pain are not clear but they involve membrane-related pathology, and both disorders have underlying genetic expansion-type mutations and are multisystem disorders [7]. As already mentioned, almost all previously published studies on the effects of pain extent have been conducted on patients with musculoskeletal problems. Results from these reports seem to support

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that simply counting the number of pain sites might be important when assessing a patient's pain problem. This approach does not seem adequate, however, for people with an NMD. At least, for this specific population, our data suggest that overlooking the pain intensity of the specific sites may result in a failure to capture the true meaning and implications of the pain experience of these patients. Reasons for this failure include the fact that NMDs involve pathophysiology in the peripheral nerves or muscles as part of the underlying disease process, which is distinctly different from a musculoskeletal disorder. Thus, a variety of abnormal processes may generate and maintain the symptom of pain in NMDs, and conceptually, it is likely that no one mechanism may be disease specific, although this topic remains to be studied. It is more likely that any given NMD would have several mechanisms associated with it. Thus, accounting for the pain in any single patient may require hypothesizing one or more mechanisms at work simultaneously. Once neuropathic pain is present, all levels of the nervous system, peripheral, central, and autonomic, may play a role in the generation and maintenance of pain. Therefore, independent of actual clinical diagnosis, several different pathophysiologic processes may be present simultaneously [8]. Further, some patients with neuropathic pain may also develop secondary myofascial pain. Myofascial pain may mimic neuropathic pain and result in referred pain distant from the actual soft tissue source and is a logical explanation for the chain of events occurring in a hereditary neuropathy like Charcot Marie Tooth disease. However, these myofascial pain generators are likely further accentuated in diseased, dystrophic muscle such as seen in DM1 or FSHD. It is probable that, to some extent, skeletal muscle pathophysiology plays a significant role in pain generation in this setting. Because of active, on-going muscle degeneration, there is significant risk for overwork weakness and exercise-induced muscle injury, even with simply doing activities of daily living. Dystrophic muscle is susceptible to exercise induced muscle injury, particularly eccentric muscle contractions [9]. Patients with NMD are susceptible to overwork weakness and muscle injury, resulting in excessive delayed-onset muscle soreness. This soreness usually occurs 24–48 hours after exercise. Other symptoms might include muscle cramping, heaviness in the extremities, prolonged dyspnea, and fatigue. Fatigue in this setting is likely multi-factorial because of deconditioning and impaired muscular activation, but likely contributes to pain. The importance of considering pain site when assessing pain and its impact in persons with chronic pain is reinforced by data indicating that pain extent is significantly associated with pain interference and psychological functioning. Pain extent likely plays a significant role in many chronic pain populations. The nature of pain in individuals with DM1 and FSHD suggests that this would be important in patients with these diagnoses as well. However, it is not clear whether pain in the low back and arms is more strongly associated with pain interference and psychological functioning than pain at other locations; research is needed to address this specific question. Nonetheless, the study findings reviewed support the idea that pain site matters, although further study is clearly warranted. It seems, however, that the pain sites that matter most to persons suffering from chronic pain in the setting of an NMD like DM1 or FSHD may differ significantly from those of persons with other chronic pain conditions [10]. The findings have important implications for understanding and treating, pain in persons with NMD. Clearly, a comprehensive assessment of these persons will require going beyond the mere assessment of overall or general pain intensity and will require gathering information about the intensity of each pain problem. Thus, in this population, both a quantitative and qualitative assessment of the pain experience should be promoted. Especially relevant would be to attend to the pain in legs, feet, hips, and knees beyond overall pain intensity, because they are all significantly related to pain interference. Pain experienced in the head should also be addressed given its potential impact on psychological functioning above and beyond what is expected from overall pain intensity.

Conclusion

The authors also hypothesize that pain interference and psychological functioning are associated with pain intensity at different sites, although this hypothesis needs empiric confirmation with data. Intuitively, the sites most likely to exert the strongest associations are the ones related to ambulation, which makes biomechanical sense, given that these muscles are particularly taxed physically and consequently susceptible to contraction-induced injury, as discussed earlier

Acknowledgement

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Conflict of Interest

None

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