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Review Article

Is chronic pain in degenerative joint disease maintained by central pain memory rather than ongoing peripheral pathology?

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ABSTRACT

Chronic pain is a key feature of degenerative joint disease however pain severity often shows poor correlation with the degree of structural damage seen on imaging. This is commonly referred to as pain–structure discordance. It challenges the traditional view that joint pathology alone determines symptom severity. A number of pieces of evidence from pain science suggests that central mechanisms, including central sensitisation and maladaptive neuroplasticity, may contribute to the persistence of pain beyond the initial peripheral condition. These processes are sometimes described as "pain memory". This involves the amplification of nociceptive signalling within the spinal cord and brain, which leads to heightened pain perception that may potentially become partially independent of ongoing joint pathology. This narrative review explores the concept that chronic pain in degenerative joint disease may be maintained, at least in part, by central pain mechanisms rather than solely by peripheral structural changes. Evidence from clinical observations and outcomes following joint replacement surgery is discussed to illustrate how pain can persist despite stable disease or removal of damaged joint tissue. Understanding the role of central pain mechanisms is essential for patient assessment and supports a more integrated model of degenerative joint disease pain that incorporates both peripheral and central factors.

Keywords: Degenerative joint disease, Maladaptive neuroplasticity, Central sensitisation, System-structure Discordance, Pain memory, Osteoarthritis, Pain–structure discordance

INTRODUCTION

Degenerative joint disease, which are also known as osteoarthritis (OA) is a long term chronic condition caused by the breakdown of joint cartilage, leading to pain swelling and reduced mobility.[1-4] As of December 2023, an estimated 350,000 people are diagnosed with OA each year with a median age of 55.[5] It is most common in the knees, hip and also the spine. The pain involved with OA is due to the triggering of nociceptive signalling in the nociceptors surrounding the affected joint.[6] This signal which is associated with pain can go through one of two options. The first is that it may get redirected immediately into the spinal reflex loop. This causes a rapid response. The second option is that the nociceptive signal is transported to areas of the brain that integrate information with higher-ordered sensations such as the feeling of pain. The frequent signalling of this input can lead to something known as maladaptive neuroplasticity, where the nervous system changes in a way that amplifies pain, creating central sensitisation. This hyperactivity of the central nervous system can cause worsening pain signals and widespread pain even without ongoing tissue damage.[7,8] This central sensitisation can in turn lead to the formation of something we now call "pain memory". This means that despite a moderate X-ray appearance in conditions such as OA, the pain can feel excessive and persistent regardless of the structural damage. This is also known as system-structure discordance that is a common issue faced by patients suffering from osteoarthritis. This leads to patients

feeling helpless with regards to their chronic pain which can trigger symptoms of depression and anxiety in some.

AIM OF THE REVIEW

This review aims to explore whether symptom-structure discordance can be explained by maladaptive neuroplasticity rather than ongoing peripheral tissue pathology. It examines the neurobiological mechanisms through which maladaptive changes in central and peripheral pain processing can amplify or maintain pain in the absence of proportionate structural damage. In addition, the review analyses existing experimental studies, clinical trials, and observational evidence that challenge a purely structural and mechanical model of pain, highlighting findings that support central sensitisation, altered nociceptive signalling, and cognitive affective modulation as key contributors to persistent pain states.

LITERATURE REVIEW

A narrative review of peer-reviewed literature was conducted to examine maladaptive neuroplasticity, symptom-structure discordance, and their relationship to persistent pain following joint replacement surgery for degenerative disease. Electronic databases including PubMed, NIH, Scopus, and Google Scholar were searched for articles published between 2000 and 2026. Search terms included combinations of "maladaptive neuroplasticity", "central sensitisation", "symptom-structure discordance", "degenerative joint disease", "osteoarthritis", "joint replacement", and "post surgical pain". Eligible studies included clinical trials, cohort studies, imaging studies, and systematic or narrative reviews involving adult human participants undergoing hip or knee arthroplasty for degenerative conditions. Studies were included if they explored pain outcomes disproportionate to structural findings or examined neuroplastic or central mechanisms of pain. Animal studies, acute traumatic injuries, non surgical pain models and non peer reviewed sources were all excluded. Given the heterogeneity of study designs and mechanistic frameworks, a narrative review was done to explore the mechanisms involved and their implications in future research and clinical studies. This way a thorough analysis can be done of existing literature, which will allow us to begin to understand the possible effect on future patients.

DISCUSSION

Symptom-structure discordance in degenerative joint disease

Across numerous studies on degenerative joint disease, we can see that the severity of pain in relation to radiographic severity is inconsistent. This means that despite a patient showing a modest knee OA radiographic, they could be suffering from pain which could be described as disabling. In a systematic search done, it revealed that the proportion of patients with pain in their knees found to have radiographic osteoarthritis ranged from 15-76%, and in patients with radiographic knee OA the proportion with pain ranged from 15-81%.[9]
On the other hand, evidence from large population based cohorts suggested a strong association between radiographic severity of knee osteoarthritis and pain outcomes. Data from the Multicentre Osteoarthritis Study (MOST) and the Framingham Osteoarthritis Study, involving 696 and 336 participants respectively, showed that higher Kellgren-Lawrence grades were proportionately associated with increased likelihood and severity of knee pain. Notably, individuals with Kellgren-Lawrence grade 4 showed markedly higher odds of frequent knee pain compared with grade 0 in both cohorts.[10] These findings were consistent across cohorts however they do not disprove the presence of symptom structure discordance at the individual patient level or exclude centrally mediated pain mechanisms in patients whose pain persists despite surgical intervention.

Evidence of altered pain processing in patients with disproportionate pain

Quantitative sensory testing (QST) studies can provide objective evidence of altered pain processing in individuals who report pain disproportionate to radiographic osteoarthritis severity. In cohort analysis done by dividing patients by concordance between pain and structural findings, it showed that individuals with high clinical pain, but low radiographic severity demonstrated significantly lower pressure pain thresholds and enhanced temporal summation compared with those with concordant low pain. These differences were observed both locally at the knee and at remote sites, which indicates widespread hyperalgesia as opposed to focal nociceptive sensitisation. Statistical analyses showed that group differences in pain sensitivity remained significant after adjustment for age, sex, body mass index, depressive symptoms, and radiographic severity, supporting an association independent of structural disease burden. Collectively, these findings demonstrate that patients with disproportionate pain exhibit quantifiable abnormalities in central pain processing, measurable through psychophysical testing, that are not explained by joint pathology alone.[3]

These findings affirm that symptom-structure discordance in degenerative joint disease can be partly mediated by maladaptive neuroplastic changes in central pain processing rather than persistent peripheral nociceptive signalling. The presence of widespread hyperalgesia and enhanced temporal summation implies central sensitisation, characterised by increased excitability and reduced inhibition within nociceptive pathways.[11,12] In the context of joint replacement surgery, this has important implications as structural correction may remove the primary peripheral driver of pain, yet centrally maintained sensitisation may persist, limiting symptom resolution. While the association between altered pain processing and

disproportionate pain is strong, causality cannot be definitively established, and the extent to which central sensitisation is reversible following surgical or rehabilitative interventions remains uncertain. Further longitudinal studies are required to clarify whether early identification of altered pain processing can inform patient selection and preoperative counselling.

Pain outcomes following joint replacement surgery

In a systematic review done in 2018 reviewing patients who underwent total knee arthroplasty (TKA), it showed that 10-34% of patients reported unfavourable pain outcomes between 3 months and 5 years after the surgery. It reported that in the UK 100,000 primary TKA operations are done annually meaning that there are potentially 20,000 patients who would experience chronic pain after TKA per year. This is important to highlight as chronic pain can lead to a lower quality of life leading to functional limitations, depression, poor general health, anxiety, sleep issues and excessive opioid use.[13]

The statistics found in this systematic review highlight the issue with a strictly mechanical and structural model of pain in patients with OA. While surgery effectively addresses biomechanical dysfunction and nociceptive input arising from degenerative joint conditions, it does not proportionately reverse pain perception. The persistence of pain supports the contribution of centrally mediated mechanisms, including maladaptive neuroplasticity and impaired endogenous pain inhibition, which may remain active after removal of the peripheral driver. However, heterogeneity in outcome measures, follow up duration and importantly the definitions of persistent pain across studies limits direct comparison, underscoring the need for standardised assessment frameworks and longitudinal evaluation of central pain mechanisms in post arthroplasty populations to understand the extent of effects.

Psychological and affective correlates of post-operative pain

A paper written on neural mechanisms associated with OA pain outlines how knee OA arises from both central and peripheral mechanisms, contributing to the observed dissociation between structural severity and symptom intensity. The author highlight's role of malfunctioning cortical excitability networks and impaired endogenous pain modulation systems in maintaining chronic knee OA pain, although the exact mechanism remains incompletely understood, it supports the biopsychosocial model of OA pain in which central sensitisation and maladaptive neuroplasticity are a key driving factor in chronic pain. A total of 105 patients were assessed using the Western Ontario and McMaster Universities Osteoarthritis (WOMAC) pain scale and visual analog scale (VAS) pain assessments. It showed that pain experienced during physical activity, as measured by the WOMAC pain score, was strongly associated with central neural mechanisms. Higher WOMAC scores were predicted by reduced intracortical inhibition ($p=0.021$), increased beta-band oscillatory activity ($p=0.027$), and dysfunctional conditioned pain modulation ($p<0.001$), alongside psychological and peripheral sensitisation factors. Using this information we can see that the association between higher WOMAC pain scores and reduced intracortical inhibition suggests that this inhibitory gating is compromised, resulting in excessive cortical responsiveness to afferent sensory input during movement. As a consequence, normal mechanical signals generated by joint motion may be amplified and interpreted as painful. We can also see the elevated beta-band oscillatory activity which further supports this interpretation, as beta rhythms are associated with maintenance of the current motor state and suppression of motor adaptability. Persistently increased beta activity has been linked to rigid motor strategies and reduced capacity to update motor output in response to sensory feedback.[14] In the context of pain, this rigidity may reinforce maladaptive movement patterns and perpetuate nociceptive signalling. Taken together, these converging abnormalities suggest that movement evoked pain reflects a state in which both cortical inhibitory control and spinal brainstem pain suppression are insufficient, allowing pain to persist independently of peripheral tissue damage. This supports the concept of maladaptive neuroplasticity as a key contributor to ongoing pain despite structural correction.[15]

CONCLUSION

This review highlights that pain severity in degenerative joint disease and following joint replacement frequently demonstrates discordance with structural pathology, challenging a purely mechanical model of pain. Evidence from psychophysical, neurophysiological, and clinical outcome studies indicates that altered central pain processing, including central sensitisation, impaired descending inhibition and maladaptive neuroplasticity contributes substantially to persistent pain particularly during movement. Moreover, pain measured during physical activity and at rest reflects distinct underlying mechanisms, with movement evoked pain more closely associated with neural dysfunction and pain at rest more strongly influenced by psychological factors. These distinctions help explain why structural correction through joint replacement does not uniformly result in symptom resolution. Recognising the contribution of central mechanisms has important implications for patient selection, preoperative counselling, and post operative management and also supports the need for integrative treatment approaches that address both peripheral and central drivers of pain. Future research should focus on identifying patients at risk of centrally mediated pain and developing targeted interventions to improve outcomes following joint replacement surgery.

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