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Short Communication

Body Composition, Obesity, Inflammatory Markers, and Non-Specific Low Back Pain

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Abstract

An opinion piece questioning the diagnosis of 'non-specific low back pain', with a focus on lower back pain being of systemic origin in those with both unfavorable body composition and absence of a pathoanatomical cause.

Keywords: Non-Specific Low Back Pain; Obesity; Inflammatory Markers; Body Composition; Waist Hip Ratio

Abbreviations: Low Back pain (LBP), C-reactive protein (CRP)

Introduction

The introduction begins with the statement of the problem, prevalence of low back pain and introduces the concept of 'non-specific low back pain'. Low back pain (LBP) is the most common musculoskeletal (MSK) problem globally [1]. LBP is a complex condition that can be diagnosed more specifically according to the mechanism of injury, location, and additional involvement of other areas of pain. However, one of the most common diagnoses, accounting for 90% of LBP (Mayer et al, 2017) is 'non-specific LBP' which means that pain cannot be explained by a pathoanatomical cause [2].

Certain clinical tests such as lumbar spine flexion, slump and straight leg raise might be sensitive and specific [3] for diagnosing conditions such as lumbar spine radiculopathies, but often, common musculoskeletal clinical tests to detect the aetiology of pain are limited, which results in a diagnosis of 'nonspecific low back pain'. This frequently results in a frustrating outcome for the patient due to the lack of clarity in the diagnosis. Clinicians are often left in a position where they validate the patient's pain, while being unable to specifically explain the pathoanatomical cause, which can lead to sub-standard rehab approaches. Despite the acceptance of this diagnosis in clinical circles, it is important to challenge and reframe this to improve the accuracy of the therapeutic approach.

While it is common for BMI to be addressed in clinical circles to meet surgical requirements, it is rarely mentioned as a direct cause of LBP. Furthermore, while BMI is often used to measure body compositional health, the work of Goosens and colleagues (2017) have highlighted that BMI alone is not a sufficient marker to measure one's body compositional profile. Instead, factors such as the distribution and volume of adipocytes are more influential than BMI alone [4]. This is important as this position helps to expand on the simplistic explanation of excessive mass acting on joint structures, since adipocytes exert autocrine and exocrine functions, which can result in low grade systemic inflammation [5]. An excess number of adipocytes is proposed to lead to unfavourable endocrinological changes which can lead to an increase in inflammatory markers. [6] described elevated IL-6, and reduced IL-10 levels in peripheral blood of chronic LBP patients, thereby suggesting an imbalance between proinflammatory and anti-inflammatory mediators which can contribute to the pathophysiology of LBP. Another study found

that several chemokines are elevated in non-specific acute and chronic LBP (Injeyan et al, 2018) [7]. showed that C-reactive protein (CRP) levels are raised in a diverse group of LBP patients. This is interesting as CRP is a commonly used marker of inflammation that is used in medical environments but does not receive recognition as an indicator of LBP.

Such research supports the notion that excess adipocytes can elevate inflammatory markers which have been shown to be associated with LBP [2, 8]. Given this information, clinicians must be aware that the 'non-specific LBP' might only be nonspecific due to the limitations of the diagnostic techniques within the musculoskeletal clinical setting. While Organisations are unlikely to sanction blood tests to ascertain whether systemic factors are causing a patient's LBP, there are some feasible and indirect, cost-effective measures that can be used clinically. One example is waist to hip ratio, as a high waist to hip ratio has been shown to be strongly associated with both an unfavorable inflammatory profile [9] and LBP [10].

It is important that a clinician is aware of such research as this might be able to change the narrative in the absence of pathoanatomical/pathomechanical causes and presence of high BMI, high adiposity, or a high waist to hip ratio. As a result, a clinician could start the process by communicating the following message to the patient. 'The assessment demonstrated that there is no anatomical structure that is causing your back pain. However, your waist to hip ratio is high, and research has demonstrated that this can increase the number of fat cells which can increase inflammation in the body which has been shown to increase your risk of low back pain. As a result, implementing methods to improve your waist to hip ratio might address this and improve your outcomes. It might be useful to consider the role of exercise and nutrition to achieve this'.

Should clinicians be receptive and ready to move beyond conventional musculoskeletal assessments, it is plausible that the information presented in this short piece might just end up reframing the narrative surrounding nonspecific lower back pain. This in turn, may serve to elucidate the diagnosis while improving education and therapeutic interventions with the goal of improving patient outcomes.

Discussion

The paper draws on research that demonstrates both an indirect and direct link between unfavourable body composition/obesity, adiposity, inflammatory markers, and low back pain. The discussion refers to potential barriers and suggests a cost-effective measure to use in a clinical environment which could reframe and challenge conventional approaches to treating this condition in this population.

Conclusion

The paper concludes by encouraging clinicians to think and investigate beyond the conventional musculoskeletal clinical assessment for LBP to improve diagnostic accuracy, education, therapeutic interventions and outcomes for this population group.

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Conflict of Interest

No Conflict-of-Interest.

No financial interest and no conflict of interest exists in writing this paper.

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