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Low back pain: shifting the paradigm from non-specific to specific

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ABSTRACT

Low back pain is a leading cause of disability worldwide and it imposes huge economic burden on affected individuals and the government. It can be broadly classified into specific and non-specific low back pain. Non-specific low back pain constitutes about 90% of all low back pain, and it is now a leading cause of years lived with disability. The present review aims to describe the evolution of the term non-specific low back pain, to show that the term may no longer be appropriate and to propose hypothesis that can explain low back pain in general. A review of the literature shows that several terms have been used to describe non-specific low back pain. Examples of such terms are mechanical low back pain, and idiopathic low back pain with few inconsistencies in the usage of the term. The term, non-specific low back pain, became widely accepted after introduction of the concept "diagnostic triage" by Waddell in 1994. The concept classified low back pain into specific, radicular syndrome and non-specific. As explained by Waddell, only radicular syndrome and specific types of low back pain require further diagnostic evaluation. This has been the practice ever since. Recent evidence from MRI findings of symptomatic and asymptomatic individuals however, suggest that diagnostic evaluation, coupled with specific provocation tests can lead to specific diagnosis of low back pain in majority of the cases. Based on these recent evidences and others, it can be hypothesized that low back pain is a spectrum of disease with a specific cause.

INTRODUCTION

Low back pain (LBP) is a leading cause of disability worldwide and it imposes huge economic burden on affected individuals and the government.[1] Among those with low back pain, the economic burden is more pronounced in people with chronic low back pain (CLBP).[2] For convenience and simplicity, practitioners have suitably coined the term 'specific' for a type of LBP that responds to focused treatment. Example of such 'specific LBP' are LBP due to infection, cancer, or fracture. There is another type of CLBP that the pathoanatomical cause has eluded clinicians for a while and is assumed to have no known targeted treatment. Practitioners have conveniently termed this type of LBP 'non-specific'(NSLBP).[3] NSLBP has attracted and continue to attract a lot of attention from researchers due to several reasons. First, it represents about 95% of LBP cases and it is now a leading cause of years lived with disability. It has become a serious public health problem with a lifetime prevalence of 84%, and total annual health cost estimated to range between $9 billion in Australia and $100 billion in the USA.[3-5] Second, because of the problem of associating it with a particular pathoanatomical mechanism, there is a growing trend to regard it as a "complex" disease entity that is heterogenous in nature rather than a homogenous disease entity.

For example, Wand and colleague succinctly brought this debate to the fore, and critically examined the strength and weaknesses of each side of the debate, but strongly concluded that there was not enough evidence to support a heterogeneous NSLBP. They rather inferred that there may be other alternative explanation.[6] Third, a number of observational studies have shown that there is no correlate between the clinical presentation of NSLBP and radiological findings. There are a lot of asymptomatic individuals who have evidence of degenerative disease on

plain radiograph and MRI while there are other individuals who have pain but with no specific radiological findings.[7,8] Fourth, because of its presumed complex nature and lack of a specific pathoanatomical mechanism, researchers have proposed several modes of treatment, and this is coupled with release of several clinical guidelines.[9-11]

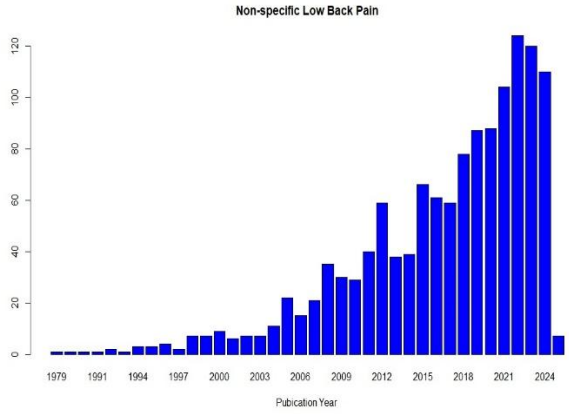

Figure 1: The increase in the number of publications on LBP over the years.

Figure 1 shows the rapid increase in the number of publications on NSLBP, especially in the last four years, but despite this large turnout of literature, it seems little progress has been made in unravelling this elusive pathology, both in diagnosis and treatment. Hence, the need for an updated review. The aims of this present review are to describe the evolution of the term NSLBP.[1] It is imperative to do so at this time because many researchers and clinicians have ascribed different meaning to the term, and this may explain the different approach to its diagnosis and treatment as seen in the literature. Revisit the possibility that the term "non-specific" LBP may no longer be appropriate in describing low back pain in light of recent evidences introduce "new insight" that may explain the poor clinical correlate between symptomatology and radiological findings.[2,3]

EVOLUTION OF NON-SPECIFIC LOW BACK PAIN

The term NSLBP first appeared in the literature in 1956 by E.G Shaw and J.G Taylor when they presented the result of their retrospective study on lumbar fusion for low back pain.[12] They applied the term to cases where they could not obtain a diagnosis from the review of the case notes and radiographs that was used in the study. They however stated that the diagnosis considered in the study were spondylolisthesis, degenerative disc disease, and congenital anomalies.

Nothing was said of other possible diagnosis like facet joint arthropathy, sacroiliac joint disease and spinal stenosis, and advanced imaging was not available at that time. In 1982, White and Gordon mentioned that there are other terms such as low back strain, lumbago and mechanical low-back pain that have been used to describe NSLBPP though no references was cited. They went further to state that those low back pain could be described using the term 'idiopathic', and were probably the first people to do so.[13] The import of their description become relevant when four years late, Deyo expressly classified low back pain into mechanical, non-mechanical and visceral type citing White and Godorn, and he described the mechanical type, in a way that suggest NSLBP i.e., it could arise from any of the structures in the back.

This sentence should be continuous with the preceding paragraph.[1,2]

This classification by Deyo is important because it represents the earliest attempt at harmonizing the different inconsistencies used in describing LBP. Subsequent articles written by Deyo and colleagues in 1992, 1995, 1996 and 2001 further revealed other inconsistencies in the usage of the term NSLBP.[14-17] Only strain, sprain and degenerative disease were considered non-specific or idiopathic, and they were classified also as examples of mechanical low back pain similar to herniated disc, spinal stenosis and spondylolisthesis. This usage is however different from other authors like White and Gordon et al who consider lumbar sprain, lumbar strain, and mechanical low back pain as idiopathic or non-specific, while Shaw et al and Taylor et al used it to describe a lack of diagnosis due to inconclusive reviews from case notes and radiographs.[12,13]

It therefore shows that the term NSLBP meant different things to different authors, and that its previous usage is not the same as it is presently being used in modern times. Highlighting this fact is very important as will be shown shortly. In 2004, Waddell et al introduced the concept 'Diagnostic triage' for low back pain, and this concept categorized low back pain into (a) NSLBP (b) radicular pain or spinal stenosis and (c) specific low back pain. The term was widely accepted in the academic community and it became the reference point for subsequent discussion and research on low back pain.[18-22]

There are however few important points that should be mentioned as regards to the concept of diagnostic triage. First, NSLBP is now described to mean a LBP that arises from structures in the back but without a specific known cause, and no longer refers specifically to lumbar sprain or strain. Second, the inherent definition of NSLBP in the new concept encompasses what previous authors described as mechanical low back pain. Third, herniated disc and spinal stenosis are now in a separate category, and are no longer classified as either NSLBP or mechanical low back pain. Fourth, the usage of the NSLBP term as defined by Waddell is premised on two facts (i) it has no specific treatment (ii) the radiological findings do not correlate with the symptomatology. Fifth, it is based on this diagnostic triage that many international guidelines gave their recommendations. Sixth, the diagnostic triage concept proposed that only radicular syndrome and

specific LBP should have further diagnostic evaluation because NSLBP is now considered "insignificant", since it is assumed that it poses no threat to the spine and hence requires no further diagnostic work-up. Unfortunately, NSLBP has drawn more attention in the research community compared to the other two. The reason why it is important to highlight how NSLBP evolved from mechanical LBP to diagnostic triage is because it shows how other diseases of the spine like degenerative disc disease, spondylolysis, spondylosis and spondylolisthesis, which ordinarily would have required further investigations are now lumped together as NSLBP.

Further diagnostic evaluation was deemphasized for NSLBP as long as clinical evaluation is not suggestive of other ominous diagnosis. Hence, we are at an era where majority of the symptomatic LBP are not further investigated as long as the LBP does not bear the ominous sign of 'specific' or 'radicular' symptoms. But based on current evidence in the literature, should the spine community and clinicians generally continue to follow this approach?

NON-SPECIFIC LOW BACK PAIN MAY NOT BE NON-SPECIFIC

There is growing evidence that the term NSLBP may be a misnomer because it can be shown that what was previously described as NSLBP may actually have specific causes. For example, Nicodemus and colleagues were able to prove that there are pain generators at the back which have strong association with sacroiliac joint (SIJ) dysfunction and hence can be regarded as 'specific' causes of low back pain. Also, previous mantra in the management of NSLBP requires no further diagnostic investigation because of the non-specific nature of the diagnosis.[23] There are however growing number of studies, from 41 studies in 2007 to 62 studies in 2023 that have shown that specific diagnosis of LBP can be made using specific diagnostic tests. These findings are as a result recent systematic reviews.[24-26]

The findings of the review show that Pfirrmann scale, annular fissures, modic changes and centralization phenomenon are specific to herniated disc. Similarly, the review also shows that radionuclide imaging, distraction test, absence of midline low back pain and pain provocation tests can be used to make a diagnosis of SIJ dysfunction in a patient classified as having NSLBP.[25] It shows that with accurate clinical evaluation and appropriate diagnostic tests, many conditions labelled as NSLBP can now have specific diagnosis.

This observation was accurately depicted in a recent article written by Suzuki et al where they combined specific clinical evaluation and appropriate diagnostic tests to make specific diagnosis of LBP in conditions that previously would have been classified as NSLBP and they concluded that this approach reduced the rate of NSLBP to 22% while allowing specific diagnosis of LBP to be made in 78%.[43] Making specific diagnosis of LBP give clinicians opportunity to offer targeted therapy to patients which has reduced the rate of ambiguous treatment and improved clinical outcomes of such patients.[27-31]

THE PARADIGM SHIFT AND EVOLVING CONCEPT

As discussed in the previous section, NSLBP may not be non-specific after all, and it is no longer surprising that a lot of authors share similar opinion with other authors suggesting a move away from usage of the term. For example, Abraham et al, considered the term NSLBP as a myth and further stated that usage of the term prevents clinicians from searching for the specific causes of LBP. He advised that the term should be abandoned.[32] Similarly, Wiechert et al considered usage of the term as flawed because according to them, clinicians are presently not taking advantage of the modern diagnostic tools available at their disposal.[33] They suggested that all efforts should be channelled to establishing a definite diagnosis of LBP. One reason many researchers and practitioners continue to support usage of the term is because of evidence from previous studies that symptomatic and asymptomatic individuals with LBP have similar findings on MRI.[8,34]

There are however recent evidences which suggest that those previous inconclusive imaging findings may require interpretation in a new light. For instance, Jensen et al in 1994 pointed out that though asymptomatic individuals may have disc bulges and disc protrusion on MRI, the prevalence is higher in symptomatic patients and that unlike asymptomatic individuals, symptomatic individuals are more likely to have disc extrusion.[35] They were also able to show that number of disc abnormality increases with age.

In 2015, twenty-one years after the report of Jensen et al, Brinjikji et al conducted a meta-analysis of previous studies comparing MRI findings in asymptomatic and symptomatic adults, and concluded from their findings that symptomatic patients had significant association with MRI evidence of disc bulge, disc degeneration, disc protrusion, modic changes and spondylolysis compared to asymptomatic individuals.[36] Similarly, Kasch et al showed in their study that the MRI findings increases with age, and that the MRI findings is associated with severity of LBP, though they concluded that the findings cannot be used to predict future LBP.[37]

If the findings of these studies are combined with the result of Han et al, one can formulate two hypotheses: (1) if abnormal MRI findings are present in asymptomatic and symptomatic individuals but symptomatic patients have more abnormal findings, one can hypothesize that LBP is a progressive disease that can be graded from not severe (asymptomatic) to severe disease (symptomatic). This hypothesis can be explained using two analogies of two diseases, one non-orthopaedic disease and one orthopaedic

disease. First is haemorrhoid, which can be graded from I to IV.[25]

There are many people with grade 1 haemorrhoid who are asymptomatic and who would never present for care in their lifetime. If those with grade 1 haemorrhoid improve their diet and maintain a good lifestyle, the haemorrhoid may either regress or it may not progress beyond grade I. If however, such patients do nothing about it, the haemorrhoid may progress to higher grades which may require intervention.[38] The second analogy is osteoarthritis (OA) of either the knee or hip joints. There are patients who have hip or knee pain but without radiological features of OA. There are also patients who have no problems with either their knee or hip but have radiological features of OA.Conservative care is usually advised in early OA while surgical intervention is offered in severe form of OA.[39,40] The second hypothesis is that since specific causes of LBP can be identified based on specific MRI findings coupled with some provocation test, it may be convenient to propose that all low back pain have a cause and the reality that the causes of some LBP are still unknown is not an excuse to categorize them as non-specific. It been shown that there are several structures that make up the spine and each of these structures could be a pain generator.[41] Hence, as proposed by Malik et al, evaluation and management of LBP needs a paradigm shift that move from treating LBP as non-specific to using all diagnostic arsenal at our disposal to identify the specific causes of LBP thereby improving the target care of patients previously categorized as having NSLBP.[42]

CONCLUSION

LBP still remains a major global health problem but with the current available evidence, it will be a misnomer to consider majority of its cause as non-specific. Evidence has shown that with detailed evaluation and diagnostic work-up, a cause of the LBP can be found. It is proposed that LBP should be considered a spectrum of disease and hence, researchers, clinicians and general practitioners should consider shifting away from the existing method of evaluating LBP to a more pragmatic and definitive approach.

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