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Is Low Grade Infection a Contributing Factor of Disc Degeneration?

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Abstract

Intervertebral Disc Degeneration (IVD) has a multi factorial origin. Genetic and environmental factors are major determinants. However the pathomechanisms of IVD is inadequately understood. Low grade infection has also been implicated, however, its scientific rigor has been questioned. This article reviews the current literature in this field. Evidence suggests that low grade infection can also be imputed as a potential causative factor of IVD and perhaps serves as a paradigm shift of our current thinking in this entity.

Keywords: Infection; Low Back Pain; Degeneration

Mini Review

Our current consensus is that the underlying causes of disc degeneration are multi factorial but in many aspects obscured. Different factors including ageing, genetic predisposition, mechanical loading, metabolic disorders, toxins and infection acting independently or collectively activate or contribute to the degeneration cascade [1]. However, a generally accepted disease model has not been established. In a recently published paper the authors suggested a vicious circle as a model of the disease which involves abnormal mechanical forces, catabolic cell response, and alterations in extracellular matrix [2]. They also suggest that low grade infection could possibly trigger cells to decay the extracellular matrix of the intervertebral disc [2].

The concept that spinal infection can cause intervertebral disc degeneration is not new [3,4]. Several studies have drawn the attention to the relationship between Modic I signals in MRI scan (high intensity bone marrow signal in T2-weighted and low signal intensity in T1-weighted series) and infection [5]. In fact when the patients do not have a persistent severe back pain, abnormal blood count and elevated inflammatory markers, it is challenging to differentiate between Modic I changes and low-grade infection [5]. The strong relationship between Modic I changes and back pain has been also reported [6].

Furthermore, there is an increasing interest and debate on the theory that low grade spondilodiscitis from Propionibacterium play an important role in the pathogenesis of disc degeneration and the clinical manifestation of the disease. Stirling cultured Propioni bacterium acne in 84% of patients with disc herniation and sciatica [3]. More recently anaerobic bacteria and mainly Propionibacterium acne has been associated with Modic I changes and back pain [7]. Other authors isolated anaerobic pathogens from disc tissue however, the possibility of tissue contamination during harvesting cannot be excluded [8,9].

Additionally, two studies published in 2013 by a Danish – British research group supported the theory that the occurrence of Modic I changes in the vertebrae adjacent to a previously herniated disc may be due to oedema surrounding an infected disc with anaerobic bacteria of low virulence. They also supported that a treatment protocol based on certain antibiotics (amoxicillin and clavulanate) were more effective in this group of patients (Modic I) than placebo [7,10]. Subsequently, an extensive discussion was initiated in the medical community raising serious doubts on the scientific rigor of the studies, questioning the patient selection, the treatment methodology and duration, the risk of tissue contamination, the authors' conflict of interest [11,12], and the risks of the inappropriate and extensive use of antibiotics [13].

In a previous study, we were able to identified HSV-1 and CMV DNA in humans disc samples harvested during discectomy. Using a polymerase chain reaction based assay, we searched for the DNA of eight different herpes viruses in 16 patients and two controls. Herpes Simplex Virus type-1 (HSV-1) was the most frequently

detected virus (56.25%), followed by Cytomegalovirus (CMV) (37.5%). Evidence of inflammation (TNF-α and IL-6) was also detected in the disc specimens empowering the involvement of virus infection to the inflammation process. We proposed that the presence of the viruses into the disc might create the circumstances where the disc is susceptible to mechanical stress and trauma [14].

Low-grade infection, might affect the nucleus pulposus cells metabolic status, and enlarge the susceptibility of the intervertebraldisc to environmental and genetic factors, which trigger or promote the biological cascade resulting in disc degeneration [2,14]. Despite the drawbacks and critique, subclinical infection is an attractive new theory of disc degeneration and low back pain [15]. In 2005, Barry Marshal along with his colleague Robin Waren won the Nobel Prize in Medicine because they proved that peptic ulcers are caused by a bacterium called Helicobacter pillory and not by stress or acidity as medical community believed. Their erratic paper published in Lancet in 1983 was a major shift in medical thinking and consequently in clinical practice [16]. Though it is hard to believe at first glance, bacteria have been implicated in the pathogenesis of other conditions that do not primarily impose as infectious diseases and a paradigm shift may be on the horizon [13].

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