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Saudi Journal of Biological Sciences

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REVIEW

Medical ozone therapy as a potential treatment modality for regeneration of damaged articular cartilage in osteoarthritis

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Received 5 October 2015; revised 19 January 2016; accepted 1 February 2016

The material in this review paper submitted to Saudi Journal of Biological Sciences has neither been published, nor is being considered elsewhere for publication.

KEYWORDS

Osteoarthritis (OA);
Articular cartilage;
Ozone (O₃) therapy;
Reactive oxygen species
(ROS)

Abstract Osteoarthritis (OA) is the most common degenerative joint disease and a growing health problem affecting more than half of the population over the age of 65. It is characterized by inflammation in the cartilage and synovium, resulting in the loss of joint structure and progressive damage to the cartilage. Many pro-inflammatory mediators are elevated in OA, including reactive oxygen species (ROS) such as nitric oxide (NO) and hydrogen peroxide (H₂O₂). Damaged articular cartilage remains a challenge to treat due to the limited self-healing capacity of the tissue and unsuccessful biological interventions. This highlights the need for better therapeutic strategies to heal damaged articular cartilage. Ozone (O₃) therapy has been shown to have positive results in the treatment of OA; however the use of O₃ therapy as a therapeutic agent is controversial. There is a perception that O₃ is always toxic, whereas evidence indicates that when it is applied following a specified method, O₃ can be effective in the treatment of degenerative diseases. The mechanism of action of O₃ therapy in OA is not fully understood and this review summarizes the use of O₃ therapy in the treatment of damaged articular cartilage in OA.

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Peer review under responsibility of King Saud University.



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<http://dx.doi.org/10.1016/j.sjbs.2016.02.002>

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Please cite this article in press as: Manoto, S.L. et al., Medical ozone therapy as a potential treatment modality for regeneration of damaged articular cartilage in osteoarthritis. Saudi Journal of Biological Sciences (2016), <http://dx.doi.org/10.1016/j.sjbs.2016.02.002>

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1. Introduction

Osteoarthritis (OA) is the most common type of arthritis affecting more than half of people over the age of 65 and is more prevalent in women (18%) than in men (9.6%) after menopause (Musumeci et al., 2015). It is a chronic, degenerative joint disorder affecting millions of people worldwide (Ashkavand et al., 2013). OA is considered as one of the most common causes of disability affecting the joints of the knee, hips and hands. It can be classified into two different forms namely: primary and secondary OA. Primary or idiopathic OA is gene dependent while secondary or post traumatic OA occurs mainly after a traumatic event (Musumeci et al., 2015). Although primary and secondary OA are caused by different factors, they both result in the same abnormalities, a degenerative phenomenon complicated by inflammatory reactions (Reynard and Loughlin, 2012). OA is a complex disease causing a change in the tissue homeostasis of articular cartilage

and the subchondral bone. The common features of OA include loss of cartilage, narrowing of joint spaces, hypertrophic bone changes and the formation of osteophyte (Ashkavand et al., 2013). The latter refers to the overgrowth of the bone and cartilage occurring at the joint margins. The stiffening of the subchondral bone causes the bone to be less able to absorb impact loads and thereby leading to increased stress in the cartilage (Li and Aspden, 1997). The primary cause of OA remains largely unknown and various factors can play a role in the development of OA (Fig. 1). The development of knee arthritis has been strongly associated with excess weight, obesity, gender and previous knee injury. Diabetes is also associated with the progression of knee OA (Musumeci et al., 2015). The signs and symptoms of OA usually include: pain, stiffness of the joints, muscle weakness and swelling of the knee.

Articular cartilage is a specialized connective tissue covering the joint surfaces that has no nerve supply and is therefore

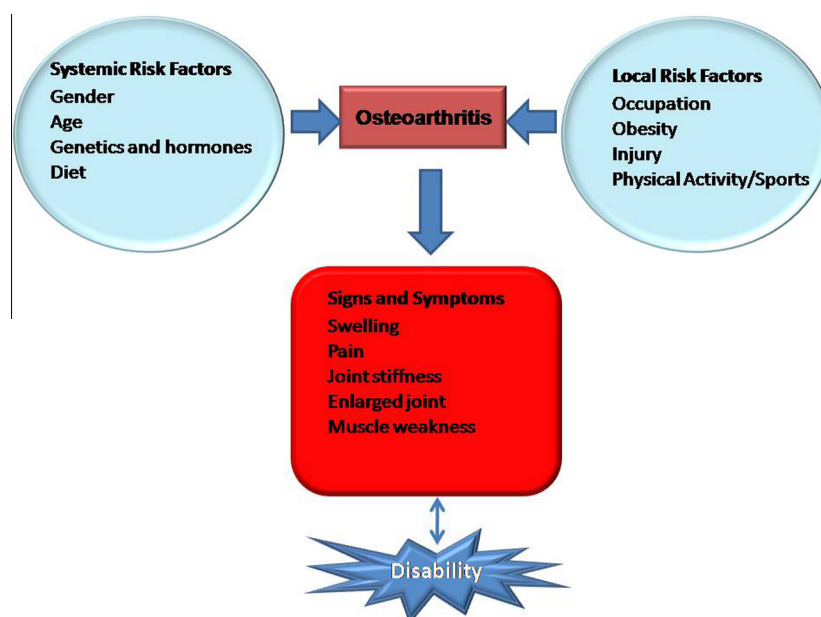


Figure 1 An illustration of the risk factors that are involved in osteoarthritis (adapted from Ashkavand et al., 2013).

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