Correspondence

Temporomandibular joint osteoarthritis - Histopathological study of the effects of intra-articular injection of Triamcinolone Acetonide

Dear Sir,

I read with interest an article "Temporomandibular joint osteoarthritis - Histopathological study of the effects of intra-articular injection of triamcinolone acetonide" by Haddad.1 This is one of the few studies carried out on the histopathological changes of the arthrodial joints following intra-articular injections of corticosteroids. Hereby, I would like to discuss the role of intra-articular injections of corticosteroids in the treatment of osteoarthritis. Corticosteroids are beneficial in the treatment of osteoarthritis in humans. But the mechanism of action of the corticosteroids in the relief of symptoms in osteoarthritis is complex. Osteoarthritis is a slow progressive disease characterized by destruction of the cartilage induced by a complex interplay of genetic, metabolic, biochemical, and biomechanical factors with secondary components of inflammation. Chondrocytes are probably the most important cells responsible for the development of the osteoarthritic process. They secrete a family of zinc containing enzymes called matrix metalloproteinases (MMPs) which include stromelysin, collagenase, and gelatinase. In osteoarthritis, the activities of MMPs are increased in the articular cartilage.2 These enzymes degrade all components of articular extracellular matrix and can cause destruction of articular cartilage.3 Recent studies have demonstrated that intra-articular corticosteroids offer a chondroprotective effect in osteoarthritic lesions, which is mediated through the suppression of stromelysin synthesis.4 Corticosteroids may also act by down-regulation of urokinase plasminogen activator (u-PA) activity. It has been shown that u-PA may be the first enzyme in the cascade of activation of pro-matrix metalloproteinases by the fibrinolytic system.5 Intra-articular corticosteroids slow cartilage catabolism and osteophyte formation in animals.6,7 The protective effect of triamcinolone has been found to be dose dependent in a model of chemically induced articular cartilage damage.8 Haddad9 has concluded that intra-articular injection of steroids into human osteoarthritic temporomandibular joints (TMJ) acts as a lytic agent. There is no conclusive evidence in his study that lytic effects of the cartilage were produced by intra-articular injections of steroids, as the histopathological changes in the 2 groups of patients of TMJ osteoarthritis, ie one receiving steroids and the other not receiving steroids, are similar except with a quantitative difference. The slightly higher destruction of all layers of articular cartilage receiving intra-articular injections of triamcinolone may be related to the advanced stage of the primary disease process itself or overuse of an already damaged joint through pain relief afforded by the intra-articular corticosteroids. The higher number (59%) of patients with osteoarthritis who obtained a satisfactory response with intra-articular steroids, may very well be due to the chondroprotective action rather than lytic action of the steroid. The studies of the effects of intra-articular steroids in the human osteoarthritic joints are sparse. Further controlled studies, including immunohistochemical studies, are required to have a better insight of such an engimatic subject.

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Reply from the Author

Author declined to reply.

References