

Osteoarthritis of the  
Temporomandibular Joint

Talia Becker

*Oral Medicine specialist at IDF clinic, Sheba hospital, Israel*

## Article Information

Received date: Mar 03, 2017

Accepted date: Mar 06, 2017

Published date: Mar 07, 2017

## Corresponding author

Talia Becker, Oral Medicine specialist  
at IDF clinic, Sheba hospital, Israel,  
Tel: +972-505808886;  
Email: becker@zahav.net.il

Distributed under Creative Commons  
CC-BY 4.0

Article DOI 10.36876/smmd.1011

## Editorial

Osteoarthritis (OA), a degenerative disease of the articular cartilage, is one of the most frequent pathologies of the Temporomandibular Joint (TMJ), characterized by spontaneous pain at rest or in function, decreased range of motion, and articular noise. Fibrillation, erosion of the articular surfaces, chondrocyte proliferation, articular cartilage eburnation, synovitis, and the inhibition of articular component synthesis [1] have been demonstrated. Magnetic Resonance Imaging (MRI) and Computed Tomography (CT) are most commonly used for the examination of osseous TMJ changes. MRI has been the gold standard for examination of soft tissue changes [2] but CT has been found to be superior to MRI in the assessment of bony TMJ components [3,4].

Disc displacement is considered as an initial change in TMJ pathology, frequently leading to secondary tissue damage, and it precedes the onset of TMJ OA [5]. The collapse of lubrication function is assumed to be an important cause in the pathogenesis. Prostaglandin E<sub>2</sub> (PGE<sub>2</sub>) and leukotriene B<sub>4</sub> (LTB<sub>4</sub>) were found to be present in the synovial fluid of inflamed, dysfunctional temporomandibular joints. A positive correlation was found [6] between the levels of these lipid mediators of pain and inflammation and an index of clinical joint pathology. Tumor Necrosis Factor-Alpha [TNF- $\alpha$ ] may promote the release of proteinases and stimulate the expression of degrading enzymes and inflammatory mediators, resulting in TMJ inflammation and bone and cartilage degradation [7].

Intra-articular TMJ injections of sodium hyaluronate or a corticosteroid in patients with osteoarthritis of the TMJ were found to reduce pain and improve function [8]. The reported positive clinical effects of steroids or sodium hyaluronate in various TMJ diseases may indicate reparative bony changes [4]. The application of Hyaluronic Acid (HA) has been gaining ground as a conservative and effective treatment for OA of the TMJ. It is regarded as a useful long-term therapy with similar or better therapeutic effects than corticosteroid use. The evidence available suggests that the application of HA regulates various inflammatory mediators in osteoarthritic processes in the TMJ [1].

Plasma Rich In Platelet-Derived Growth Factors (PRGFs) is a biological therapy based on the use of the patient's own plasma, from which a cocktail of cytokines and proteins is obtained. Recent clinical studies have demonstrated the beneficial role of the injection of PRGF, showing improved clinical outcomes compared with HA [9]. Further evidence in regard of the current options of therapy is required.

## References

1. Iturriaga V, Bornhardt T, Manterola C, Brebi P. Effect of hyaluronic acid on the regulation of inflammatory mediators in osteoarthritis of the temporomandibular joint: a systematic review. *Int J Oral Maxillofac Surg*. 2017.
2. Emshoff R, Gerhard S, Ennemoser T, Rudisch A. Magnetic resonance imaging findings of internal derangement, osteoarthrosis, effusion, and bone marrow edema before and after performance of arthrocentesis and hydraulic distension of the temporomandibular joint. *Oral Surg Oral Med Oral Pathol Oral RadiolEndod*. 2006; 101: 784-790.
3. Larheim TA. Current trends in temporomandibular joint imaging. *Oral Surg Oral Med Oral Pathol Oral RadiolEndod*. 1995 Nov; 80: 555-576.
4. Møystad A, Mork-Knutse BB, Bjørnland T. Injection of sodium hyaluronate compared to a corticosteroid in the treatment of patients with temporomandibular joint osteoarthritis: a CT evaluation. *Oral Surg Oral Med Oral Pathol Oral RadiolEndod*. 2008; 105.
5. Wei L, Xiong H, Li B, Cheng Y, Long X. Boundary-lubricating ability and lubricin in synovial fluid of patients with temporomandibular joint disorder. *J Oral Maxillofac Surg*. 2010 Oct; 68: 2478-2483.
6. Quinn JH, Bazan NG. Identification of prostaglandin E<sub>2</sub> and leukotriene B<sub>4</sub> in the synovial fluid of painful, dysfunctional temporomandibular joints. *J Oral Maxillofac Surg*. 1990; 48: 968-971.

OPEN ACCESS

ISSN: 2576-5442

7. Kellesarian SV, Al-Kheraif AA, Vohra F, Ghanem A, Malmstrom H, Romanos GE, et al. Cytokine profile in the synovial fluid of patients with temporomandibular joint disorders: A systematic review. *Cytokine*. 2016; 77: 98-106.
8. Bjørnland T1, Gjaerum AA, Møystad A. Osteoarthritis of the temporomandibular joint: an evaluation of the effects and complications of corticosteroid injection compared with injection with sodium hyaluronate. *J Oral Rehabil*. 2007; 34: 583-589.
9. Fernández-Ferro M, Fernández-Sanromán J, Blanco-Carrión A, Costas-López A, López-Betancourt A, Arenaz-Bua J, et al. Comparison of intra-articular injection of plasma rich in growth factors versus hyaluronic acid following arthroscopy in the treatment of temporomandibular dysfunction: A randomised prospective study. *J Craniomaxillofac Surg*. 2017.