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Histological findings in TMI treated with high condilectomy for internal derangement

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ABSTRACT

Intra-Articular Temporo-Mandibular Disorders (TMD) are characterized by displacement of the disc that causes the condyles to slip back over the disc thus resulting in TMJ discal damage and erosion of the condyle's bone. The etiology of temporomandibular disorder (TMD) is multidimensional: biomechanical, neuromuscular, bio-psychosocial and biological factors may contribute to the disorder.

The study involved 46 joints in 27 patients with a diagnosis of Intra-Articular Temporo-Mandibular Disorders (TMD) according to Axis I of Diagnostic Criteria for Temporomandibular Disorders (DC/TMD) for Clinical and Research Applications and underwent surgery between 2011 and 2014.

Patients were divided into three groups.

Group 1 were included patients with Disc Displacement (DD) without reduction without limited opening, Group 2 patients with DD without reduction with limited opening. Finally, Group 3 included patients with Degenerative Joint Disease (DJD) TMD.

In all cases, diagnosis of Intra-Articular Temporo-Mandibular Disorders (TMD) was confirmed by preoperative examination (clinical, MRI and/or CT scan). Tissue specimens were obtained from all 50 joints for histopathology.

The aim of this study was to analyse histological features of the surgical specimens obtained from patients with Intra-Articular Temporo-Mandibular Disorders who underwent surgery and assess the association with clinical findings and imaging.

Preliminary results show in Group 1, fibrocartilage is preserved and regular, there are isolated outbreaks of bone resorption and focal sclerosis. In Groups 2 and 3 fibrocartilages are irregular and thickness varies widely and sclerosis is more pronounced.

In early stages of TMD, the disc antero-medial displacement might play a fundamental role in the etiopathogenesis that can became an irreversible joint damage thus leading to a wide spectrum of articular symptoms and signs in TMD (Cohen et al., 2014; Hagandora and Almarza, 2012; Nah, 2012).

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1. Introduction

Temporo-Mandibular Disorders (TMD) are a multifactorial disease process with various causes, including parafunctional habits (eg, bruxing, tooth clenching, lip or cheek biting), emotional distress, acute trauma to the jaw, trauma from hyperextension (eg. dental procedures, oral intubations for general anaesthesia, yawning, hyperextension associated with cervical trauma), instability of maxillomandibular relationships, laxity of the joint, and

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comorbidity of other rheumatic or musculoskeletal disorders. Most common symptoms include local pain, limited mouth opening and TMJ noises, chronic muscle pain and headache (De Rossi et al., 2014). Pain-related TMD can impact the individual's daily activities, psychosocial functioning, and quality of life ("National Institute of Dental and Craniofacial Research. FacialPain."). Furthermore, TMD symptoms occur disproportionately between the sexes with a much higher incidence reported in females; female to male ratios range between 2:1-8:1 (van Loon et al., 2002; Warren and Fried, 2001). Most patients presenting symptoms are between 20 and 50 years of age, an unusual distribution for a disease that is considered a degenerative disorder (van Loon et al.,

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2002). Temporomandibular disorders (TMD) are a significant public health problem affecting approximately 5%–12% of the population. While up to 25% of the population may experience symptoms of TMD, only a small percentage of affected individuals search for treatment. For instance, studies in the 1980s detected TMD symptoms in 16% up to 59% of the population, although only 3%-7% of the adult population actually sought care for pain and dysfunction associated with TMD (Solberg et al., 1979; Carlsson, 1999). TMD is the second most common musculoskeletal condition (after chronic low back pain) resulting in pain and disability ("National Institute of Dental and Craniofacial Research. Facial Pain."). Up to 70% of cases of TMD are accompanied by malpositioning of the temporomandibular joint (TMJ) disc, termed "internal derangement" (ID) (Farrar and McCarty, 1979). While disease progression is poorly understood, the primary pathology appears to be a degenerative condition known as osteoarthritis (OA). In TMD patients, it is readily apparent that once joint breakdown begins, OA can be crippling, leading to morphological deformity and functional obstruction (Zarb and Carlsson, 1999).

Although onset is not well characterized, correlations between disc displacement and osteoarthritic change have been identified. Temporomandibular joint disorders of TMJ are characterized by displacement of the disc that causes the condyles to slip back over the disc thus resulting in TMJ discal damage and erosion of the condyle's bone. The TMJ is a unique load-bearing joint located in the craniofacial region and is formed of fibrocartilage. The anatomical structure of the TMJ is essential for joint function, including unhindered jaw movement while speaking and masticating, but it is commonly affected by degenerative diseases and finally osteoarthritis. The temporomandibular joint (TMJ) has the capacity to adapt to external stimuli, and loading changes can affect the position of condyles, as well as the structural and cellular components of the mandibular condylar cartilage (Dutra et al., 2018).

The mandibular condylar cartilage has four distinct cellular zones that express different types of collagen and non-collagen proteins: 1) the superficial or articular zone; 2) the proliferative zone, composed of undifferentiated mesenchymal cells and that responds to loading demands; 3) the prehypertrophic zone, composed of mature chondrocytes expressing collagen type 2; and 4) the hypertrophic zone, the region where the hypertrophic chondrocytes expressing collagen type 10 die and undergo calcification. The non-mineralized region is rich in proteoglycans which provide resistance to compressive forces (Benjamin and Ralphs, 2004).

Maintenance of the homeostasis of all cellular regions of the anatomical structure and the mineralization of the subchondral portion are essential to the health, load-bearing capacity, and integrity of the TMJ.

The multiple collagen transgenic mouse model as described by Utreja et al. (2016) is a great tool to use to understand changes in bone. In Literature some animal models for an in-depth histological evaluation of histological stains are used to study mineralization, cell proliferation, and apoptosis; sheep seems to be an excellent experimental model for TMJ studies (Dutra et al., 2018; Angelo et al., 2016; Pirttiniemi et al., 2004).

Chisnoiu et al. describe the changes in the mandible that were observed in Wistar rats. All samples developed changes in the thickness of the condylar cartilage compared to the control group. The most important modifications with severe cartilage thickness reduction have been obtained in case of the BSES group where all subjects were exposed to a combination of biomechanical and emotional stress. The histopathological analysis was performed in order to observe the TMJ modifications in rats; there were no histological modifications in the control group on serial transversal sections in TMJ (Chisnoiu et al., 2016).

As with all animal models, there are difficulties in the transition to humans and clinical care.

Disc displacement represents the first pathological step responsible for clinical manifestations of what has been known as the temporomandibular joint pain-dysfunction syndrome or similarly described conditions. Effective clinical management takes new importance due to progression in advanced degenerative states that may occur (Wilkes, 1989).

Management options are different with respect to severity of degeneration and related to Intra-Articular TMD stages.

Tissue characterization and histological damage are essential to identify the etiopathological mechanisms related to the disc displacement. Remodelling of the load-bearing joints is an essential adaptation process needed for appropriate stress distribution and function. The importance of the integrity of condyle-disc unit is highlighted by the high correlation of structural alterations with higher severity degrees of intra-articular TMD.

Embree et al. present a surgical rabbit TMJ disc perforation model demonstrating cellular and histological evidence that signify cartilage degeneration. Severe cartilage damage was evident, whereby cartilage eroded to the subchondral bone with a diffuse loss of cells shown for the first time in an animal model in which TMJ disc injury induced heterotopic ossification (HO). There are several plausible mechanisms underlying condylar cartilage degeneration in this model, including joint atrophy due to TMJ disuse or condyle excessive loading that is associated with OA (Laverty et al., 2010).

The aim of this study was to analyse histological features of the surgical specimens obtained from patients with TMD that have been surgically treated; all patients underwent high condylectomy with TMJ arthroplasty, and to assess the association with clinical findings and imaging (Zhang et al., 2018).

2. Materials and Methods

This retrospective study included 27 patients for a total number of 50 joints, 5 males (16%) and 22 females (84%) who underwent surgery between 2011 and 2014.

All patients received a comprehensive history and physical examination with imaging studies.

Inclusion criteria were a TMJ evaluation, consisting of a standardized clinical examination and a subjective evaluation, with a visual analogue scale to assess TMJ pain and jaw function.

Moreover, patients were investigated for headache, local pain, cervical pain, brachial pain, tinnitus and vertigo.

All patients underwent a panoramic radiograph and a real-time MRI at 15 frames per second during the natural opening and closing of the mouth when a TMJ disc displacement was suspected, while CT scan was performed in patients suspected of TMJ degenerative joint disease according to Diagnostic Criteria for Temporomandibular Disorders (DC/TMD) for Clinical and Research Applications Axis I.

Only patients affected by TMJ Disc Displacement (DD) without reduction with and without limited mouth opening and Degenerative Joint Disease (DJD) evaluated according to Axis I were included in the study and surgically treated.

Patients affected by Disc Displacement (DD) with reduction (DDWR) and DD with reduction with intermittent locking were excluded because no surgical treatment was recommended.

Patients were divided into three Groups: Group 1 were included patients with Disc Displacement (DD) without reduction (DDwoR) without limited opening, Group 2 patients with DD without reduction with limited opening.

Finally, Group 3 included patients with Degenerative Joint Disease (DJD) TMD.

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