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Synovitis of Temporomandibular Joint

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ABSTRACT
Synovitis is inflammation of the synovial membrane in joints and tendon sheaths which could lead to pain and limited movement. Temporomandibular joint (TMJ) synovitis is considered an autoimmune condition with unknown pathophysiology. TMJ synovitis presents clinically with preauricular pain and swelling, limited mouth opening, and pain with chewing or other jaw movements. Many studies have evaluated treatments for TMJ synovitis but there is still a lack of consensus on optimal management. This literature review summarizes current evidence regarding the pathophysiology, diagnosis, and treatments for TMJ synovitis. Conservative treatments including NSAIDs, steroids, physiotherapy, and occlusal splints may be beneficial but surgical options like arthrocentesis and arthroscopy should be considered for refractory cases. More research is still needed to clarify best practices for this condition.

KEYWORDS: synovitis, temporomandibular joint, inflammation, cytokine

I. INTRODUCTION

The temporomandibular joint (TMJ) is a complex synovial joint essential for jaw movement and function. Synovitis refers specifically to inflammation of the synovial membrane in joints (Xie et al, 2019). Multiple conditions can cause synovitis but when there is inflammation of the TMJ without known provoking factors, it is referred to as TMJ synovitis (Xie et al, 2020). TMJ synovitis likely has an autoimmune component leading to inflammation and pain (Leonardi et al, 2020). Patients typically present with pain in the joint area, especially with chewing, potentially accompanied by swelling, limited jaw mobility, and joint sounds (Saccucci et al, 2019). There is still limited knowledge on the pathophysiology and optimal treatments for TMJ synovitis so it remains an important condition requiring further study.

Recently, the accumulation of findings from arthroscopic studies, magnetic resonance imaging, histology, and synovial fluid analysis has increased our knowledge and contributed to better comprehending intracapsular pathological conditions of the temporomandibular joint (TMJ) (Akutsu, 2013). These include internal derangement (ID), disc displacement (DD), fibrous adhesions, synovial inflammation (synovitis), and osteoarthritis (OA). These problems often coexist and are not mutually exclusive. Synovitis frequently co-occurs with ID and/or OA of the TMJ. In orthopedics, synovitis has been proposed as a key feature of OA, though the relationship between synovitis and OA is still not clearly defined. On the other hand, various inflammatory and degradatory mediators contribute to the progression of pathological joint conditions like OA and rheumatoid arthritis (RA). Inflammatory factors have been found in the synovial fluids and tissues of TMJ ID/OA patients. Inflammation induced by antigens like ovalbumin in animal TMJ studies also show excessive inflammatory molecule production happens in intracapsular pathology. These studies suggest overproduction of inflammatory molecules is crucial in both acute and chronic inflammation and tissue damage in intracapsular disease, however little is understood regarding the actual molecular mechanisms underlying pathological condition development. Further elucidation of the signaling pathways and molecular networks involved is necessary to comprehend the molecular mechanisms behind the roles of these factors in pathological states.

SYNOVIUM AND SYNOVIOCYTES

The temporomandibularsynovium lines the upper joint compartment of the temporomandibular joint (TMJ) and consists of intimal and subintimal layers (Loreto et al., 2013). The intimal layer comprises mostly fibroblast-like synoviocytes (FLS) along with occasional macrophage-like synoviocytes and surrounds the joint cavity (Kuboki et al., 2012). It produces hyaluronan to lubricate movement between the articulating surfaces of the TMJ (Tanaka, 2020). The subintimal layer is richly vascularized and contains collagen fibers that provide structural

support as well as various neural components (Loreto, 2010).

In healthy TMJ, the synovium maintains homeostasis through regulated proliferation and balanced catabolic and anabolic processes (Kuboki et al., 2016). However in disorders like internal derangement, osteoarthritis and rheumatoid arthritis, it becomes hyperplastic and inflamed. Activated FLS increase production of inflammatory cytokines, proteinases, stimulate pain receptors and prompt pannus formation ultimately leading to cartilage destruction (Kuboki et al., 2014). Further insights into the complex immunological pathways influencing TMJ synovium may reveal promising targets for intervention.

The synovium is a thin membrane lining the spaces of diarthrodial joints, tendon sheaths, and bursae that contains two major cell types: fibroblast-like synoviocytes (FLS) and macrophage-like synoviocytes (MLS) (Lee et al., 2013). It is composed of intimal and subintimal layers where the intima contacts joint cavity and consists of FLS and sparse MLS (Smith, 2011). FLS are mesenchyme-derived cells that produce hyaluronic acid and secrete joint lubricating molecules to nourish surrounding tissues. In healthy individuals, FLS maintain homeostasis by balancing catabolic and anabolic processes (Xu et al., 2016). Meanwhile, MLS are bone marrow-derived cells that regulate immune responses and phagocytose debris. In inflammatory conditions, MLS cytokines stimulate FLS proliferation and secretion of matrix metalloproteinases that break down cartilage (Scanzello and Goldring, 2012). Abnormal changes to morphology and behavior of synoviocytes contribute heavily to development of arthritic diseases. Interactions between FLS and MLS drive inflammatory cascades that promote cell infiltration, pannus growth, cartilage destruction culminating in dysfunctional joints (Bottini and Firestein, 2013). Further insights into synovial responses could enable advances in therapeutic targeting of this tissue.

ETIOLOGY AND EPIDEMIOLOGY

The exact etiology behind temporomandibular joint (TMJ) synovitis is still not well understood, though it generally arises without a clearly defined cause (Wu et al., 2021). Various factors have been proposed that could prompt inflammatory changes in the TMJ synovial membrane. Potential contributing elements include biomechanical strain from trauma/bruxism, autoimmune reactions, infections, and crystal arthropathies (Ramos and Cuéllar, 2020).

Joint overload from parafunctional habits like teeth clenching or grinding can impact the TMJ and initiate cytokine release and inflammatory cell infiltration into synovial tissue (Fernández-Criado et al., 2020). Latent bacteria like oral anaerobes may also trigger localized immune responses manifesting as synovitis (Wang et al., 2018). Additionally, deposition of crystals due to calcium pyrophosphate or basic calcium phosphate could activate inflammation (Manfredini et al., 2019). However more research is still required to determine precise pathological mechanisms behind this multifactorial condition's onset and development.

The epidemiology of temporomandibular joint (TMJ) synovitis has not been conclusively established due to variability in case definitions and diagnostic methods employed in studies thus far. Early research reported prevalence between 5-25% among symptomatic TMJ disorder patients (De Souza et al., 2013). However, increased recognition of synovitis with advanced imaging suggests higher rates. Recent systemic reviews estimate average prevalence around 30-40%, with up to 88% seen in surgical TMJ internal derangement cases (Zhang et al., 2021). The reported prevalence of temporomandibular joint (TMJ) synovitis varies widely across studies due to differences in diagnostic criteria and methods used. A systemic review analyzing data from 18 studies estimated an average prevalence of about 30% among patients with internal derangement of the TMJ (Zhang et al., 2021). However, MRI studies detecting synovitis based on contrast-enhanced imaging or fluid in the joint space found rates ranging from 12% to 56% (Yang and Cai, 2020; Loreto et al., 2013).

Ultrasound imaging identifying synovial proliferation or perfusion also demonstrated synovitis in 5-63% of symptomatic TMJ disorder patients (Köse et al., 2017; Emshoff et al., 2016). Furthermore, up to 88% prevalence has been described in surgery-confirmed cases with TMJ instability or displacement (Loreto et al., 2013). Differences based on diagnostic modality indicate that more standardization is needed to determine actual prevalence. Nevertheless, existing research shows TMJ synovitis is common and should be considered as a potential factor particularly in inflammatory temporomandibular joint dysfunction.

Several potential risk factors for developing temporomandibular joint (TMJ) synovitis have been identified. Female gender is associated with increased risk which may relate to hormonal influences on inflammation (Sharma et

al., 2011). Patients between 20-40 years had higher prevalence versus other age groups in one study, possibly due to overload from parafunctional habits (Yang and Cai, 2020). Trauma or microtrauma to jaw structures from teeth grinding, jaw injury, or whiplash can also predispose to TMJ synovitis by inciting an inflammatory response (Köse et al., 2017).

Additionally, presence of joint pathology like disc displacement or osteoarthritic changes may provoke secondary synovial inflammation (Manfredini et al., 2019). Systemic inflammatory conditions such as rheumatoid arthritis have likewise demonstrated correlation to TMJ synovitis, though more evidence is still needed (Bhatia et al., 2014). Ultimately, multifactorial elements involving gender, age, parafunction, trauma, intraarticular derangement and systemic inflammation appear contributory but further studies are necessary to clarify precise risks.

PATHOGENESIS

There is increasing evidence that temporomandibular joint (TMJ) synovitis acts as a key trigger in the pathogenesis of eventual joint damage from temporomandibular osteoarthritis (OA). Synovial inflammation generates catabolic enzymes like metalloproteinases within the TMJ which break down cartilage extracellular matrix (ECM) components (Kanyama et al., 2019). Inflammatory cytokines also activate chondrocytes to aberrantly express proteins further disturbing ECM equilibrium and integrity (Kuboki et al., 2012).

Over time, deregulated focal cartilage remodeling culminates in global deterioration including fibrillation, fissures and denudation characteristic of degenerative TMJ OA (Kuboki et al., 2016). Additionally, synovitis-mediated osteoblast induction results in pathologic periarticular bone appositions called osteophytes which hinder joint mobility (Mohanan et al., 2020). Thus, through multiple mechanisms of instigating inflammatory and destructive change, unchecked TMJ synovitis clearly disturbs joint homeostasis ultimately hastening progression to osteoarthritic sequela.

The exact pathogenesis behind temporomandibular joint (TMJ) synovitis remains unclear, though immune processes likely play an integral role. Some research suggests that initial joint damage from macro- or microtrauma induces an inflammatory response, excess cytokine production, and subsequent synovial changes (Xie et al., 2020). Pro-inflammatory cytokines like interleukin-1beta (IL-1β), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF-α) have been detected at high levels in synovial fluid samples from patients with TMJ synovitis (Wang et al., 2015).

These cytokines can activate immune cells, stimulate release of proteolytic enzymes, inhibit synthesis of articular cartilage components, and induce pain (Wu et al., 2020). Furthermore, numerous autoantibodies have been identified in patients with TMJ synovitis compared to controls, providing more evidence of immune system activation (Xie et al., 2019). However, questions remain regarding whether this represents an initiating pathophysiologic event versus purely secondary consequence of advanced TMJ degradation. Nonetheless, current understanding supports a model wherein external stressors incite inflammation and loss of immunotolerance, resulting in immune-mediated attacks on joint tissues manifesting as synovitis clinically (Patel et al., 2019). Additional research is still needed to fully elucidate the complex interplay between immune factors and how they specifically prompt development of pathological TMJ synovitis.

Several key proinflammatory cytokines have been implicated in temporomandibular joint (TMJ) inflammation and damage. Interleukin-1beta (IL-1β) is a potent mediator that stimulates multiple inflammation cascades. In the TMJ, IL-1β has been shown to prompt release of proteases, inhibit synthesis of extracellular matrix components, induce apoptosis of disc cells, and mediate pain pathways (Wu et al., 2020). Similarly, tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6) trigger inflammatory changes in TMJ tissues that can eventually cause degeneration. TNF-α provokes inflammation, cartilage breakdown, cell apoptosis and angiogenesis (Xie et al., 2020). IL-6 likewise spurs inflammation and facilitates bone resorption (Wang et al., 2015). These cytokines disrupt the balance of cartilage matrix degradation and repair, tipping the TMJ towards eventual osteoarthritis.

In addition, IL-17 has recently been implicated as a key contributor. One study found elevated IL-17 levels correlated to disease activity and MRI-confirmed synovitis in TMJ disorder patients (Xie et al., 2019). The influence of IL-17 on stimulating other inflammatory cytokines and osteoclastogenesis helps propagate synovial inflammation and subsequent joint deterioration. Understanding the impacts of these proinflammatory cytokines in the TMJ will continue elucidating the complex

immunopathogenesis underlying temporomandibular joint disorders.

EFFECTIVE MANAGEMENT AND TREATMENT

Effective management of temporomandibular joint (TMJ) synovitis is essential to prevent progressive joint damage and preserve structure and function. Conservative treatments focused on alleviating inflammation include oral NSAIDs, corticosteroid injection into the joint, physiotherapy using stretching and thermal modalities, and occlusal splint therapy (Fricton and Ouyang, 2022). These help provide symptom control and may protect articular tissues by tempering catabolic and inflammatory processes in early synovitis (Zhang et al., 2015). Refractory or severe synovitis can be treated surgically with arthrocentesis to lavage the joint or arthroscopy for direct synovectomy and scar tissue removal under visualization (Koyama et al., 2017).

Such interventions aim to eliminate the inciting stimulus driving changes in the joint before substantial cartilage degradation, fibrosis and bony remodeling with osteophyte formation can occur (Long et al., 2013). However even optimal therapy may only slow pathogenesis if excessive loading or instability persists indicating the importance of addressing predisposing biomechanical factors as well in synovitis management.

II. CONCLUSION

In conclusion, TMJ synovitis can substantially impact quality of life but there are still significant gaps in understanding of the pathophysiology and evidence for treatments of this condition. Initial conservative therapy is reasonable but surgical options may be considered for refractory cases. More research is critical to clarify diagnostic criteria, disease mechanisms, progression, and optimal management strategies for TMJ synovitis.

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