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Different Intra-Articular Injection Medications Used for Treatment of Temporomandibular Joint (TMJ) Internal Derangement: A Review of Literature

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Abstract

Temporomandibular joint disorders (TMD) is a summative term that includes all the problems relating to the TMJ and related musculoskeletal structures. TMJ disc displacement was a progressive problem that ultimately led to degenerative joint disease. This condition is devastating and affects patients' quality of life. Therefore, treatment included attempts at repositioning or replacing the disc, either by non-surgical or surgical means; however, in the 1990s, it was noted that open joint disc repositioning surgery or discectomy often led to progressive degenerative joint disease and fibrosis Intra-articular injections are a standard treatment for temporomandibular joint (TMJ) disorders, utilizing various medications to alleviate pain and improve joint function. Recent studies have highlighted several effective agents with distinct mechanisms and outcomes. This article aims to review various medications used and recommended in the literature and present the mechanism of action and results of each one and recent novel compilations indicated for treating TMG ID, which failed to improve with conservative treatment.

Key Words: *Temporomandibular Joint (TMJ).*

Introduction

TEMPOROMANDIBULAR joint disorders (TMD) is a collective term embracing all the problems relating to the TMJ and related musculoskeletal structures. It has plagued humanity throughout history, and treatment has been reported even during the time of the ancient Egyptians. It is characterized by joint sounds, pain, and restricted jaw movement **[1,2,3].**

The temporomandibular joint (TMJ) is a diarthrodial joint dictated by associated muscles and limited by ligaments **[1].** The TMJ joint is a synovial joint with articular surfaces of both the temporal bone and the condyle, comprised of fibrocartilage. It has an upper and lower compartment separated by the articular disc. The non-articular surfaces are lined by a synovial membrane that produces synovial fluid that fills the joint, which is surrounded by the TMJ capsule **[4].** Two types of movement are possible: Rotation and translation.The joint is thus referred to as "ginglymoarthrodial": A combination of ginglymoid and arthrodial, which correspond to rotation and translation, respectively **[1,5].**

• *Role of synovial fluid in lubrication:*

A small amount, less than 2ml of synovial fluid, is usually present within the healthy articular cavities of TMJ. The passive volume of the upper compartment is estimated to be 1.2ml, and that of the lower compartment is estimated to be 0.9ml. It serves two purposes. It acts as a medium for providing metabolic requirements to the articular non-vascular surfaces of the joint and phagocytosis of particular debris. Moreover, it acts as a lubricant between articular surfaces during function, decreasing friction between the disc, condyle, and fossa during joint movement **[4].**

Local hypoxia of joint components might be due to abnormal mechanical insult of the joint. Reperfusion of hypoxic cells can lead to an explosive increase in free radicals. These free radicals may lead to hyaluronic acid degradation, an impor-

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tant component of synovial fluid. Degradation of hyaluronic acid, in turn, may impair the lubrication of the TMJ, which may increase friction between the surfaces of the different joint components *[6,7]*. Exact flow mechanisms between articular cartilage and synovial fluid are unclear. However, the net result is that the coefficient of friction for the normally functioning joint is approximately 14 times less than that of a dry joint *[1]*.

• *Temporomandibular joint disorders:*

Previously, authors thought it may have been intracapsular or extracapsular or had a combined origin. The extracapsular form generally results from muscles surrounding the TMJ, while the intracapsular form results from a pathology of the articular surfaces or abnormality in the mechanical relationship of the articular structures *[8,9]*.

Several classification systems have evolved for TMD, but most are based on vague clinical criteria or assumed but not proven etiologic factors. As de Bont and colleagues described, TMD classification is separated into non-articular and articular categories *[1]*. The International Headache Society classified TMD as a subtype of secondary headache disorder in the International Classification of Headache Disorders II *[10]*. The American Academy of Orofacial Pain has expanded on this classification to articular and masticatory muscle disorders as follows *[11,12]*.

A- Articular disorders:

1. Congenital or developmental, 2. Disc derangement (internal derangement) disorders. (a. Displacement with reduction. b. Displacement without reduction (closed lock). c. Perforation), 3. Degenerative joint disorders. (a. Inflammatory: capsulitis, synovitis, polyarthritides (rheumatoid arthritis, psoriatic arthritis, ankylosing spondylitis, Reiter's syndrome, gout)) b. Non-inflammatory: osteoarthritis, 4. Trauma, 5. TMJ hypermobility. 6. TMJ hypomobility. 7. Infection. 8. Neoplasia *[12]*.

B- Masticatory muscle disorders:

1. Myofascial pain disorder, 2. Local myalgia, 3. Myositis, 4. Myospasm, 5. Myofibrotic contracture, 6. Neoplasia.

The three most common temporomandibular disorders are myofascial pain dysfunction, internal derangement, and osteoarthritis *[12]*.

Internal Derangement: The AAOMS defines internal joint derangement as a disruption of the internal aspects of the TMJ. This could be attributed to either disc displacements or adhesions/impingements with or without a normal disc position, causing alterations in the regular biomechanical motions of the intra-articular components *[13]*. Mc Carty and Farrar *[14]* 1979 concluded that disc displacement was a progressive problem that ultimately led to degenerative joint disease. Therefore, treatment included attempts at repositioning or replacing the disc, either by non-surgical or surgical means; however, in the 1990s, it was noted that open joint disc repositioning surgery or discectomy often led to progressive degenerative joint disease and fibrosis. Furthermore, arthroscopy emerged as a reliable alternative to reduce pain and increase mouth opening without changing the disc position *[15]*. However, there has been a conceptual shift from internal derangement and disc displacement being considered a primary diagnosis that required correction towards the current understanding that disc displacement/internal derangement represents an endpoint of a process in which there has been biomechanical failure from a specific cause. This underlying cause must be identified and managed if treatment is to be successful in the long term. We now know that disc malposition is present in approximately 33% of the population and that the joints are asymptomatic despite disc displacement through adaptation *[16,17]*.

Internal derangement presents with continuous pain that is localized to the TMJ and is exacerbated by jaw movement. Mechanical interferences in the joint, such as clicking and locking, often result in restricted mandibular opening or deviation of mandibular movements during opening and closing (clicks are brief sounds produced by mandibular movements associated with disc displacement with reduction) *[18-20]*.

Classification of internal derangement:

Wilkes, in 1989, classified internal derangement into five stages according to clinical, radiographic, and anatomical changes occurring *[21,22]*.This classification helps diagnose the internal derangement of the TMJ. The most interesting topic concerning TMJ internal derangement has been disc displacement, with the most common direction being the anterior displacement, but lateral and medial displacements occur (Table 1, Fig. 1) *[21,23]*.

Table (1)

	A) Clinical	B) Radiologic	C) Anatomic/pathologic
Early Stage	- No significant mechanical symptoms other than reciprocal clicking in early opening, no pain or limitation of motion.	- There is slight forward displacement, pleasing anatomic contour of the disc, and negative tomograms.	- Excellent anatomic form, slight anterior displacement, and passive in-coordination are demonstrable.
Early/Intermediate Stage	- One or more pain episodes, beginning major mechanical problems consisting of mid- to late-opening loud clicking, transient catching, and locking.	- Slight forward displacement, beginning disc deformity, slight thickening of posterior edge, and negative tomograms.	- Anterior disc displacement, early anatomic disc deformity, good central articulating area.
Intermediate Stage	- Multiple episodes of pain, significant mechanical symptoms consisting of locking (intermittent or fully closed), restriction of motion, and difficulty with function.	- Anterior disc displacement with significant disc deformity (increased thickening of posterior edge), negative tomograms.	- Marked anatomic disc deformity with anterior displacement, no complex tissue changes.
Intermediate/Late Stage	- There is a slight increase in severity over the intermediate stage.	- The severity increased over the intermediate stage, and positive tomograms showed early to moderate degenerative changes, such as eminence and deformed condylar head flattening.	- Increase in severity over the intermediate stage, hard tissue degenerative remodeling of both bearing surfaces (osteophytosis), multiple adhesions in anterior and posterior recesses, no perforation of disc or attachments.
Late Stage	- Crepitus, scraping, grating, grinding symptoms, episodic or continuous pain, chronic restriction of motion, difficulty with function.	- Disc or attachment perforation, filling defects, gross anatomic deformity of the disc and hard tissues, positive tomograms with essentially degenerative arthritic changes.	- Gross degenerative changes of disc and hard tissues, perforation of posterior attachment, multiple adhesions, osteophytosis, flattening of condyle and eminence, and subcortical cystic formation.

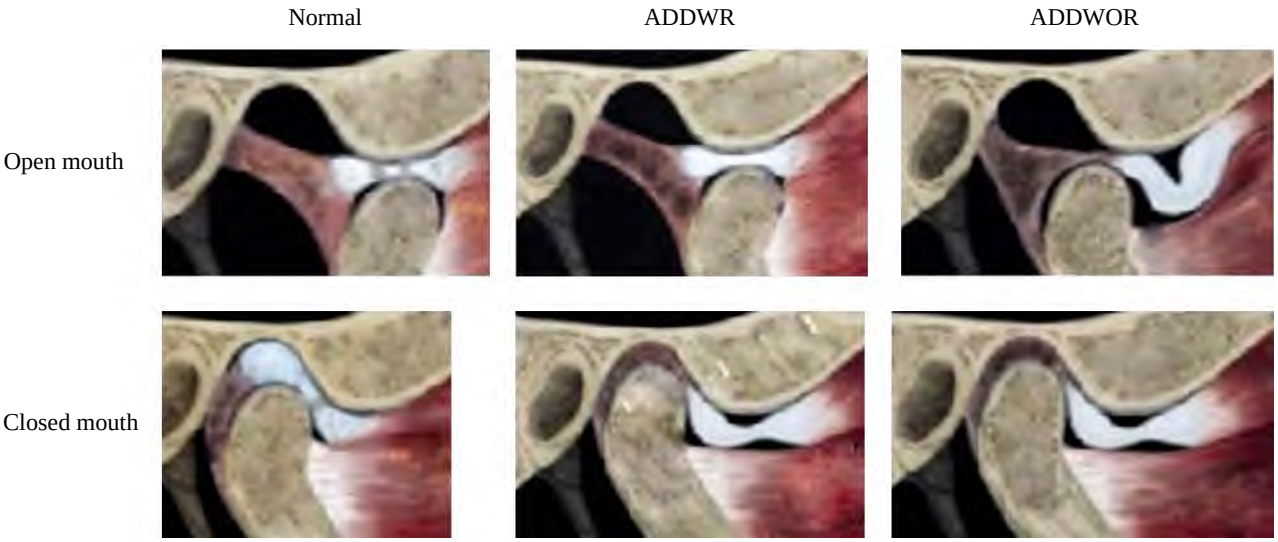


Fig. (1): Diagram representing the anterior disc displacement with and without reduction.

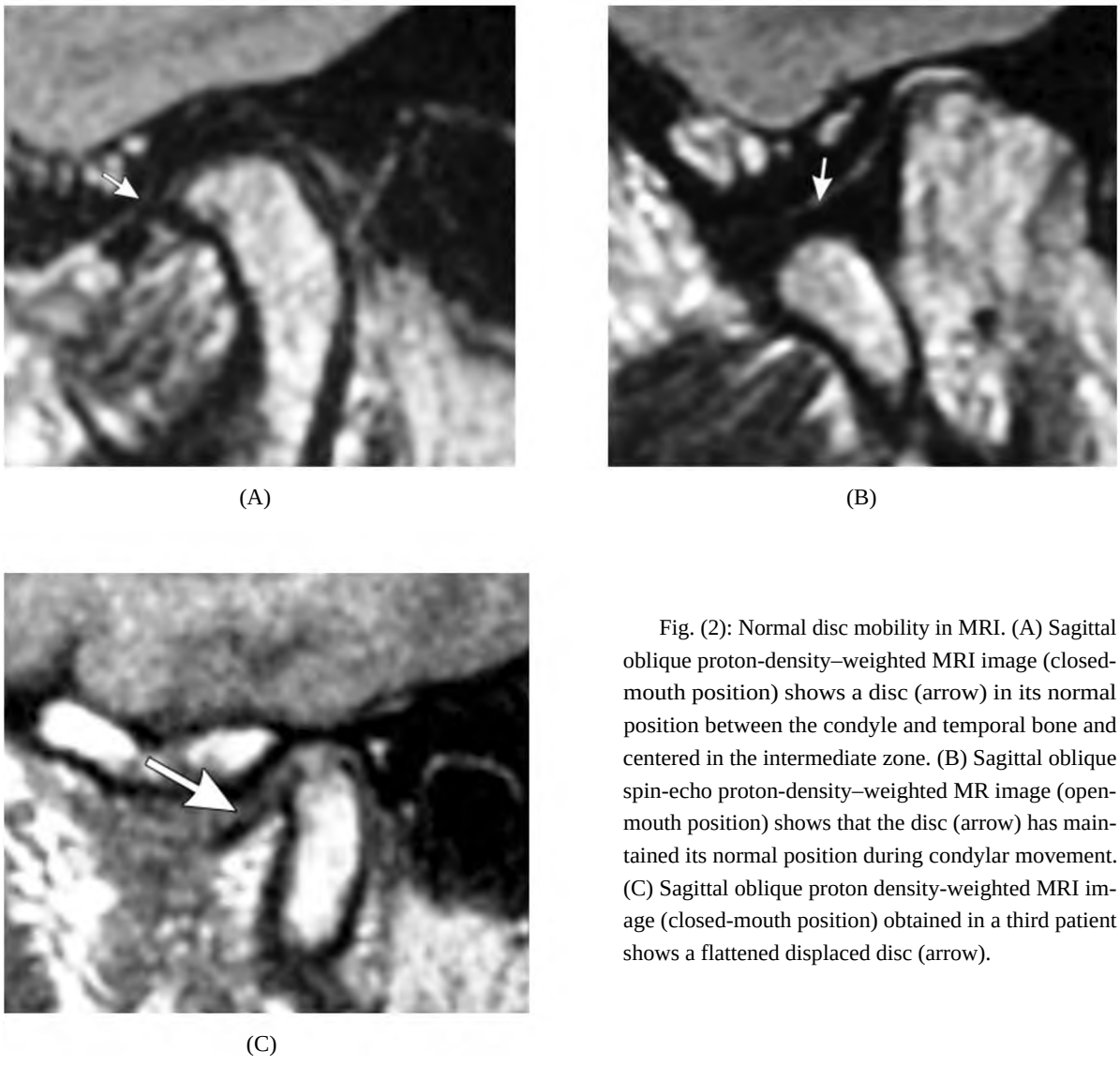
Tasaki et al., *[24]* proposed a classification of disc position comprising eight categories besides superior disc position and a tenth indeterminate category. The classification is based on sagittal and coronal MRI images. Superior and anterior disc displacement were described as follows (Fig. 2):

1- *Superior disc position:* The posterior band of the disc is superior to the disc's condyle or central thin zone (intermediate zone), located between the articular eminence's posterior surface and the condyle's anterior aspect.

2- Anterior disc displacement: Posterior band of disc anterior to anterior prominence of the condyle *[25]*.

• *Etiology of internal derangement:*

An increase in the articular friction caused by a series of events has been implicated as a cause of disc displacement, as shown in Table (2): *[7]*



(A) (B)

(C)

Fig. (2): Normal disc mobility in MRI. (A) Sagittal oblique proton-density–weighted MRI image (closed-mouth position) shows a disc (arrow) in its normal position between the condyle and temporal bone and centered in the intermediate zone. (B) Sagittal oblique spin-echo proton-density–weighted MR image (open-mouth position) shows that the disc (arrow) has maintained its normal position during condylar movement. (C) Sagittal oblique proton density-weighted MRI image (closed-mouth position) obtained in a third patient shows a flattened displaced disc (arrow).

Table (2)

Trauma	- De Leeuw, in 2008 **[6]**, stated that the most common etiologic factor in the development of ID is trauma, which includes microtrauma and microtrauma **[26]**.
Occlusal factors	- Loss of the posterior occlusal support causes discal and condylar degeneration. Any degenerative changes in the disc alter its normal biconcave shape, representing a risk factor for disc displacement **[26]**.
Inclination of the Articular Eminence	- Authors have suggested that a steep articular eminence may be an etiologic factor for disc displacement, but this assumption has not been verified **[27,28]**. There is a belief that there is a relation between the steepness of articular eminence and the progression of ID **[29]**.
The role of the Lateral Pterygoid muscle	- A hypoactivity or hyperactivity of this muscle or poor coordination of its two bellies are thought to be possible causes of functional imbalance of the TMJ **[26,30]**.
Hypermobility (Joint laxity)	- If repeated subluxation or hypermobility of the mandible occurs, elongation of the ligaments could occur, leading to the development of ID **[4]**.
Increased Friction	- Nitzan, 2001, **[7]** believed that the collapse of the lubrication system plays an important role in the chain of events leading up to disc displacement.
Emotional disturbances	- There have been suggestions that stress and anxiety have a close relation to the occurrence of TMD, especially in those with chronic pain **[31]**.

• *Treatment of internal derangement:*

Treatment of internal derangement (ID) generally includes two strategies; the first is a conservative (non-surgical approach). Meanwhile, others use invasive approaches (surgical techniques) **[32-34].**

• *Conservative management:*

The primary treatment for ID of the TMJ is generally conservative. Various non-surgical modalities have been proposed to treat ID, including patient education, soft diet, pharmacotherapy, physiotherapy, splint therapy, and heat therapy **[4,35,36].** A combination can adequately treat more than 80% of patients presenting to secondary care with conservative techniques, including rest, reassurance, non-steroidal anti-inflammatory (NSAID) medication, and bite splints. The most common conservative therapy is splint use, as physical therapy seems to be insufficient in patients with ADDWR **[37,38].**

1- Medication (pharmacotherapy): (Analgesics, Muscle relaxants, anti-inflammatory drugs (NSAIDs), Tricyclic amines (antidepressants) (TCAs), Anticonvulsants (gabapentin) **[39-43].**

2- Physical therapy: Jaw exercises (e.g., relaxation, rotation, stretching, isometrics, and postural), Application of superficial heat and/or cold,Massage, Manual mobilization, Ultrasound, Low-intensity laser, Pulsed diathermy, Other non-invasive techniques such as iontophoresis, phonophoresis **[44,45].**

3- Psychological or psychiatric treatment by appropriately qualified practitioners (including behavioral modification therapy) **[46,47].**

4- Occlusal appliances (i.e., intraoral appliances that provide even and balanced occlusal contact without forcefully altering the mandibular rest position or permanently altering the dental occlusion).These devices are worn on the teeth like a retainer or a removable denture and are usually made of processed hard acrylic. The most commonly used is the anterior repositioning splint (ARS) **[48-50].**

5- Intra-articular injections: Arthrocentesis is a technique for breaking down joint adhesions and flushing inflammatory mediators from the articular space. Different studies showed an increased range of motion tre, tremendously improved function, and reduced pain. Moreover, arthrocentesis proved minimally invasive, relatively safe, and can be performed in outpatient clinics under local anesthesia. To emphasize prognosis, intra-articular injection of various medications has been studied. Several medications, including normal saline, dextrose, local anesthesia, botox, corticosteroids, sodium hyaluronate, non-steroidal anti-inflammatory drugs, and platelets derivatives, were proposed to benefit from their analgesic, anti-inflammatory, or lubricator properties **[38,51-53].**

Corticosteroids:

Corticosteroids are powerful anti-inflammatory pharmacological drugs in oral forms or can be injected into the articular space. They have multiple actions that contribute to their anti-inflammatory effects, including the inhibition of phospholipase A2 enzyme, which decreases the production of proinflammatory prostaglandins and leukotrienes, interleukin-1 and decreases the number and activity of proinflammatory cells, including lymphocytes, eosinophils, basophils, and macrophages [54]. It was found that patients treated with intra-articular steroid injection had better pain control than those using a systemic steroid regimen. Thereby, systemic side effects such as muscle weakness, skin thinning, and peptic ulceration could be avoided [55].

Contraindications of intra-articular steroid injection are local infection and joint fractures. Side effects include joint destruction with repetitive injections, so 3 months are advised between injections. Many types of corticosteroid drugs can be used in intra-articular injection. In a survey of members of the American College of Rheumatology, the most commonly used corticosteroids were methylprednisolone acetate, triamcinolone acetonide, hydrocortisone acetate, betamethasone sodium phosphate, and prednisolone [56,57].

Hyaluronic Acid:

Hyaluronic acid (HA) (called hyaluronan or sodium hyaluronate) is a polysaccharide of the family of glycosaminoglycans, which can be found in many extracellular tissues, including synovial fluid and cartilage. chondrocytes and synoviocytes of the joints produce [58]. Hyaluronic acid. Two major complementary components are involved in synovial joint lubrication: 1) Boundary lubrication and 2) Whole fluid film [59]. It is suggested that sliding the disc in the TMJ is enabled due to phospholipids protected by hyaluronic acid constituting an efficient lubrication system. In 2001, Nitzan [7] suggested that displacement occurs due to degradation of the joint lubrication mechanisms. Accordingly, harmonic disc transference takes place thanks to the presence of phospholipids and hyaluronic acid. The phospholipids bind intimately to the joint surfaces and are highly effective lubricants. Recently, hyaluronic acid proved to adhere to the phospholipids of the joint surfaces, protecting them from lysis induced by phospholipase A2 (PLA2), an enzyme acting specifically on phospholipids. Synoviocytes, chondrocytes, and osteocytes produce PLA2 [20]. Joint overloading may be accompanied by uncontrolled generation of reactive oxygen species (ROS) that causes degradation of the HA, followed by the exposure of the phospholipids to lysis by phospholipase A2 [60].

Hyaluronic acid (HA) intra-articular injections are gaining attention as a treatment option in managing temporomandibular disorders. This highly viscous, high-molecular-weight substance is important in joint lubrication and cartilage protection [61-63]. HA serves as a lubricant, anti-inflammatory, and pain reliever. In addition to its role in lubrication, it protects the cartilage from damage by increasing the production of proteoglycan aggregate that reduces pain by increasing prostaglandin E2 and bradykinin levels in the synovial fluid [64]. It was started to treat osteoarthritis of the knee. Such treatments, called visco-supplementation, are administered as a course of injections. Findings of reduced molecular weight and concentration of hyaluronate in osteoarthritic joints highlight injectable forms' use to restore synovial fluid properties, so-called viscosupplementation, which can help treat TMJ osteoarthritis and anterior disc displacement [65-69]. Bertolami et al., in 1993 [70] examined in a multicenter study with a duration of 6 months follow up, the effect of the intraarticular injection of hyaluronate in 80 patients (experimental group): 35 with anterior disc displacement with reduction (ADDWR), 8 with anterior disc displacement without reduction (ADDW/OR), and 37 with degenerative joint disease (DJD). Forty-one patients were included in The placebo group: 15 with ADDWR, 6 with ADDW/OR, and 20 with DJD. A single dose of hyaluronate or saline was injected into the TMJ upper compartment. The results showed significant improvement in the ADDWR patients treated with the hyaluronate group regarding joint noise, mandibular deviation, and visual analog pain scores. The maximum difference in mouth opening between the hyaluronate and placebo group was 8 mm at week five. However, no statistically significant differences within or between groups were shown. In 2008, Oliveras et al. [65] conducted a study on forty-one patients with Wilkes stage II disease and divided them into two groups. The study group received one intra-articular injection of sodium hyaluronate; the control group was given two tablets of a combination of methocarbamol and paracetamol; the evaluation of efficacy by both the patients and investigators was better for the test group [65].

A comparison has also been made of the effect of hyaluronate versus corticosteroids. Kopp et al., 1985 were the first to publish a report on the effect of hyaluronate on treating temporomandibular disorders (TMD). The authors used the intra-articular injection of hyaluronate or corticosteroids in patients with TMD. A randomly double-blind

controlled trial was performed in 33 patients with TMD for whom conservative treatments had failed. Eighteen patients received 0.5ml of hyaluronate injections in the upper articular compartments of the joint twice, 2 weeks apart, and 15 patients received corticosteroids using the same approach. Follow-up was made for up to 4 weeks. The overall effect showed no statistically significant difference between the two groups ***[63]***.

Non-steroidal anti-inflammatory drugs (NSAIDs):

Non-steroidal anti-inflammatory drugs (NSAIDs) are medicines with anti-inflammatory, analgesic, and antipyretic properties ***[71]***. Their mechanism of action results from the inhibition of cyclooxygenase 1 (COX-1) and cyclooxygenase 2 (COX-2) ***[72]***. COX-1 is constantly found in the human body, and its action involves the production of compounds such as prostaglandins and thromboxane; those components play roles in the body ***[73]***. Prostaglandins are responsible for creating the protective lining of the stomach through inhibition of the production of hydrochloric acid and stimulating gastric mucus secretion ***[74]***. Therefore, long-term usage of NSAIDs, inhibiting the production of prostaglandins, promotes the development of ulceration in the digestive system lining ***[75]***. The increased production of COX-2 occurs due to inflammation, and its activation causes the symptoms that characterize it, such as redness, swelling, pain, increased temperature, and tissue function disorders. Therefore, NSAIDs may reduce the symptoms mentioned above. (76) NSAIDs are a large group of drugs classified primarily due to their effect on cyclooxygenases: (1) Inhibiting both COX-1 and COX-2 or (2) Suppressing primarily COX-2 ***[77]***. Preparations that inhibit COX-1 and COX-2 include, among others, ketoprofen, acetylsalicylic acid, indomethacin, nabumetone, ibuprofen, Piroxicam, tenoxicam, meloxicam, diclofenac, naproxen, and nimesulide ***[78,79]***. In turn, drugs that selectively suppress COX-2 are primarily coxibs. ***[80]***. These drugs can be administered orally (e.g., in the form of tablets, sachets, and syrups), rectally (suppositories), intravenously, intramuscularly, and in the form of creams, gels, and patches ***[81,82]***. Depending on the classification of NSAIDs, their doses, or the dosing method, they exert both therapeutic effects and potential side effects with varying intensity ***[83]***.

A systematic review was designed to summarize randomized controlled trials of intra-articular administration of non-steroidal anti-inflammatory drugs (NSAIDs) for temporomandibular disorders. Comparing arthrocentesis alone to arthrocentesis with NSAIDs, slight differences in joint pain were noted. For tenoxicam, differences were under 1 point on a 0–10 scale after 4 weeks, with inconsistency. Results revealed that Piroxicam showed no significant difference, and both groups had minimal pain. For maximum mouth opening (MMO), tenoxicam showed no significant difference. Based on one small trial with bias concerns, Piroxicam increased MMO by nearly 5mm. Conclusions: Currently, there is no strong scientific evidence supporting the injection of NSAIDs into the temporomandibular joint to relieve pain or increase jaw movement. Preliminary reports on Piroxicam with arthrocentesis and tenoxicam or diclofenac without rinsing justify further research ***[84]***.

Another study concluded that arthrocentesis with Piroxicam, dexamethasone, or Hyalgan is similarly effective and promising in relieving TMJ symptoms with non-reducing disc displacement. Additional high-level prospective studies are recommended to confirm the adequate dosage of each treatment protocol, frequency of the required injections, and combination between those protocols and other modalities to achieve long-term results ***[38]***.

PRP, PRF, PRGF, PDGF, and stem cell therapy An expanding corpus of research indicates the prospective advantages of intra-articular PRP, PRF, PRGF, and PDGF injections in managing TMDs. According to the current evidence, PRP injections may provide a greater pain reduction compared to placebo injections in temporomandibular joint osteoarthritis (TMJ-OA) at both 6 months (moderate level of evidence) and 12 months (moderate level of evidence) following the injection ***[85]***. PRP and PRF exhibited similar short-term efficacies in treating TMDs, while PRF was more advantageous in long-term efficacy. Therefore, PRF was recommended for treating TMDs ***[86]***. Moreover, compared to saline, PRP exhibits a prolonged duration of pain reduction and augmentation of MMO. Nevertheless, further standardized RCTs are imperative to address the discrepancies in preparation protocols and study heterogeneity across different groups ***[87]***. PRP injections provided adjuvant efficacy to arthrocentesis or arthroscopy in pain reduction for temporomandibular joint osteoarthritis in the long term. Furthermore, PRP injections significantly reduced pain better than HA, saline, or no injections ***[88]***. Eight randomized controlled clinical trials were analyzed in a systematic review assessing the benefits of applying PRP or PRGF injections simultaneously or after arthrocentesis or arthroscopy. The utilization of intra-articular injections of PRP and plasma rich in growth factors (PRGF) showcased noteworthy distinctions in

pain alleviation across three investigations, along with enhanced mandibular function, evidenced in two studies [89]. Based on limited evidence, the intra-articular introduction of mesenchymal stem cells into the TMJ could potentially yield significant effectiveness in diminishing joint pain and enhancing MMO in individuals with TMDs [90,91].

Innovative solutions in temporomandibular disorder treatment were presented in the literature, applying combinations of more than one medication for TMJ injection. Hegab and coworkers et al., compared the use of arthrocentesis (AC) + PRP + HA with AC + PRP and AC + HA as a control group [92]. They concluded that the administration of PRP in combination with HA after AC is novel and deserves further attention. The results suggested that using PRP + HA in combination with AC improved the maximum mouth opening, reducing pain, and improving joint sounds, compared to AC + HA. Attia et al., compared a group treated with a combination of HA and PRP with HA and CS controls. However, they concluded that HA and PRP relieved TMJ pain better than HA and CS over a 6-month perspective. It revealed that, from a short-term perspective, the combination of HA and CS gave a better prognosis, which might be attributed to the potent anti-inflammatory effect of CS [93,94].

Conclusion:

Corticosteroids: Commonly used for their anti–inflammatory properties, corticosteroids like dexamethasone have effectively reduced pain and improved function. Hyaluronic Acid (HA): This agent enhances joint lubrication and has been associated with improved outcomes when combined with arthrocentesis. Non-Steroidal Anti-Inflammatory Drugs (NSAIDs): Medications such as Piroxicam and tenoxicam are frequently administered post-lavage and have shown efficacy in pain management. However, they carry the risk of systemic complications and are less significant than corticosteroids. Platelet-rich plasma (PRP): Demonstrated superior outcomes in pain reduction and improved mouth opening compared to corticosteroids. Saline-PRP injections have shown significant short-term benefits in pain perception and maximum mouth opening. Combination Therapies: Studies indicate that combining lavage with HA or PRP injections yields more promising results than the individual use of each one. Combining saline with PRP or steroids has also shown promising outcomes in improving joint symptoms and function. However, more clinical trials are recommended to prove clinical evidence. Although short-term benefits are evident, the long-term outcome of these treatments varies. Despite the effectiveness of these intra-articular injections, some studies suggest that conservative treatments may still benefit certain patients, indicating a need for personalized treatment approaches based on individual patient responses to therapy.

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أدوية الحقن داخل المفصل المختلفة المستخدمة لعلاج الاضطراب الداخلى فى المفصل الصدغى الفكى : مراجعة للمقالات

اضطراب المفصل الصدغى الفكى (TMD) هو مصطلح شامل يشمل مشاكل مختلفة تتعلق بالمفصل الصدغى الفكى والهياكل العضلية الهيكلية المقابلة. كان انزياح قرص المفصل الصدغى الفكى مشكلةً متفاقمة أدت في النهاية إلى مرض تنكسى فى المفصل. هذه الحالة مدمرة وتؤثر على جودة حياة المرضى. لذلك، شمل العلاج محاولات إعادة وضع القرص أو استبداله، سواءً بوسائل غير جراحية أو جراحية؛ ومع ذلك، فى تسعينيات القرن الماضى، لوحظ أن جراحة إعادة وضع قرص المفصل المفتوح أو استئصال القرص غالبًا ما تؤدى إلى مرض تنكسى وتليف تدريجى فى المفصل. تُعد الحقن داخل المفصل علاجًا قياسيًا لاضطرابات المفصل الصدغى الفكى، باستخدام أدوية متنوعة لتخفيف الألم وتحسين وظيفة المفصل. وقد أبرزت الدراسات الحديثة العديد من العوامل الفعالة، ولكل منها آليات ونتائج مميزة. يهدف هذا المقال إلى مراجعة الأدوية المختلفة المستخدمة والموصى بها فى الأدبيات وتقديم آلية عمل كل منها ونتائجها والمجموعات الجديدة الحديثة المشار إليها لعلاج الاضطراب الداخلى لمكونات المفصل الصدغى واختلال الغضروف الداخلى للمفصل الصدغى الفكى سواء كان متراجعلا لوضعه او غير متراجع والتى فشلت فى التحسن بالعلاج التحفظى.

وقد خلصت نتائج الدراسة الى ان العوامل الدوائية المختلفة تؤدى إلى تحسن ملحوظ فى الأعراض المرتبطة بالاضطراب الداخلى فى المفصل الصدغى الفكى. علاوة على ذلك، يُقدّر التحسن الكبير في وظيفة المفصل الصدغى الفكى بهذه التقنيات البسيطة قليلة التدخل، مما يؤدى إلى تحسين جودة حياة المرضى.