BG Pulse Journal USA
ISSN:1947-2536 http://www.bgpulseusa.com/index.php/bg
BG PULSE JOURNAL

TEMPOROMANDIBULAR DISORDERS: DEVELOPMENTAL MECHANISMS, CLINICAL COURSE, DIAGNOSIS AND CONTEMPORARY MANAGEMENT — A NARRATIVE REVIEW OF CURRENT EVIDENCE

Davron Kh. Sunnatov[1], Yasmina A. Bekmirzayeva[1], Nilufar Kh. Abdukhalimova[1], Azizabonu A. Toyirova[1], Samandar S. Alikhodjaev[1], Zukhriddin Z. Rakhmiddinov[1], Ulugbek A. Khatamov[2]

[1] Faculty of Dentistry, Kimyo International University in Tashkent, Tashkent, Republic of Uzbekistan

[2] Scientific Supervisor, PhD, Associate Professor, Department of Therapeutic Dentistry, Kimyo International University in Tashkent, Tashkent, Uzbekistan.

E-mail: hatamovulugbek@yahoo.com

Doi: https://doi.org/10.5281/zenodo.22917303

Abstract

Background. Temporomandibular disorders (TMDs) comprise a heterogeneous group of painful and functional conditions of the temporomandibular joint (TMJ), the masticatory muscles and functionally associated orofacial structures, classified under ICD-10 code M26.6. Over the past two decades the conceptual model of TMD has shifted from an occlusion-centred mechanical paradigm to a multifactorial biopsychosocial framework.

Aim. To synthesise current evidence on the aetiology and molecular pathogenesis of TMDs, to characterise the clinical course of their principal phenotypes, to appraise the performance of contemporary diagnostic instruments, and to systematise evidence-based treatment principles.

Methods. A narrative review with a structured literature search was performed in PubMed/MEDLINE, Scopus and the Cochrane Library for records published between January 2005 and May 2026. Priority was given to systematic reviews, meta-analyses, network meta-analyses and clinical practice guidelines. Quantitative estimates were extracted and tabulated without re-analysis.

Results. Pooled global prevalence estimates ranged from 29.5% to 34.0% across recent meta-analyses, with a consistent female predominance (36.7% vs 26.7%). Intra-articular disorders affected 31.1% of adults, disc displacement with reduction being the most frequent subtype (25.9%). Cohort evidence identified female sex, depressive and anxiety symptoms, perceived stress, poor sleep quality, obstructive sleep apnoea, comorbid pain conditions, heightened pain sensitivity and trauma as risk factors for TMD onset. In TMJ osteoarthritis, mechanical overload activates IL-1β-, TNF-α- and IL-6-driven signalling through NF-κB and MAPK cascades, upregulating MMP-3, MMP-9, MMP-13, ADAMTS-4 and ADAMTS-5, degrading the extracellular matrix and driving subchondral bone remodelling, peripheral nociceptor sensitisation and central sensitisation. DC/TMD Axis I criteria showed high accuracy for pain-related diagnoses (myalgia, sensitivity 0.90, specificity 0.99; arthralgia, 0.89/0.98) but limited sensitivity for intra-articular diagnoses (disc displacement with reduction, 0.34; degenerative joint disease, 0.55). A network meta-analysis of 153 trials and 8,713 participants found the largest pain reductions with cognitive behavioural

therapy combined with biofeedback or relaxation (mean difference −2.62 cm on a 10 cm visual analogue scale) and therapist-assisted jaw mobilisation (−2.57 cm), both with moderate certainty.

Conclusion. TMDs are best understood as multifactorial disorders in which mechanical, inflammatory, neurophysiological and psychosocial mechanisms interact. Management should begin with reversible, education-based and exercise-based interventions; irreversible occlusal modification, discectomy and the combination of NSAIDs with opioids are subject to strong recommendations against. The governing clinical principle is to treat the patient rather than the joint.

Keywords: temporomandibular disorders; temporomandibular joint; osteoarthritis; myalgia; arthralgia; disc displacement; central sensitisation; DC/TMD; magnetic resonance imaging; cone-beam computed tomography; biopsychosocial model; orofacial pain

1. Introduction

The temporomandibular joint (TMJ) is among the most complex articulations of the human body, and its anatomical and functional characteristics differ substantially from those of other synovial joints. It simultaneously performs rotational and translational movements, and the right and left joints operate as a single functional unit constrained by the rigid mandibular arch. Because of the continuous activity of the mandible during mastication, deglutition and speech, the joint is exposed to high and repetitive mechanical loading.

Temporomandibular disorders (TMDs) occupy the border zone between dentistry, maxillofacial surgery, neurology, physiotherapy and pain medicine. Their clinical spectrum extends from isolated joint sounds to severe chronic pain, restricted mouth opening, disturbed mandibular kinematics and degenerative osseous change. In the International Classification of Diseases, 10th revision, these conditions are coded as M26.6, and the International Classification of Orofacial Pain (ICOP) places painful TMD within a broader taxonomy of orofacial pain [3].

Epidemiological estimates vary with the diagnostic criteria applied, the population studied and the methodology of the primary studies. Recent meta-analytic work places the global prevalence of TMD between approximately 29% and 34% [4,5]. It is nonetheless essential to distinguish clinically significant, treatment-requiring TMD from isolated joint sounds, which are common in asymptomatic populations and do not in themselves constitute disease.

Scientific understanding of TMD has changed markedly over the past decades. Where earlier models emphasised occlusion or mechanical dysfunction as the principal cause, the contemporary concept regards TMD as a multifactorial, biopsychosocial condition: biological injury or mechanical loading may generate the peripheral source of pain, while psychological, sleep-related and social factors influence its persistence and severity [1,6,7].

The aim of this review is to analyse the aetiology and pathogenesis of TMDs in the light of current molecular and biopsychosocial evidence, to describe the clinical course of their principal forms, to appraise the diagnostic performance of contemporary assessment instruments, and to systematise evidence-based treatment principles.

2. Materials and Methods

This work is a narrative review with a structured search strategy. PubMed/MEDLINE, Scopus and the Cochrane Library were searched for records published between January 2005 and May 2026, using combinations of the terms “temporomandibular disorders”, “temporomandibular joint

osteoarthritis", "DC/TMD", "biopsychosocial", "chronic orofacial pain", "disc displacement", "molecular signalling" and "management". Reference lists of retrieved articles were screened manually.

Priority was given to systematic reviews, meta-analyses, network meta-analyses, diagnostic accuracy studies and clinical practice guidelines. Particular weight was placed on the DC/TMD diagnostic protocol, systematic reviews of TMD risk factors, molecular reviews of TMJ osteoarthritis, and the 2023 BMJ Rapid Recommendations guideline with its accompanying network meta-analysis [8–11,17]. Quantitative estimates reported in the tables and figures of this review were extracted from the cited sources as published; no re-analysis or pooling of primary data was undertaken. Because the review synthesises published aggregate data only, ethical approval was not required.

3. Functional Anatomy and Biomechanics

The TMJ is formed by the condylar process of the mandible, the mandibular fossa and the articular eminence of the temporal bone. The articular disc, interposed between the condyle and the temporal component, adapts the incongruent articular surfaces to one another and distributes load across the joint (Figure 1).

Unlike the hyaline cartilage of most synovial joints, the TMJ disc consists predominantly of dense fibrous connective tissue. Its central portion is relatively avascular and aneural, whereas the posterior attachment — the retrodiscal or bilaminar tissue — is rich in blood vessels and nerve endings. Consequently, the source of joint pain is usually not the disc itself but the inflamed or mechanically loaded tissues surrounding it, principally the retrodiscal tissue, the capsule and the synovial membrane.

The joint capsule, the lateral ligament, the retrodiscal tissues, the synovial membrane and the periarticular musculature together maintain normal TMJ biomechanics. Mandibular movement is not a simple hinge action: during the initial phase of mouth opening condylar rotation predominates, whereas in the later phase anterior translation of the condyle–disc complex onto the articular eminence becomes dominant. Disruption of any component of this mechanism may result in disc displacement, altered movement trajectory or redistribution of mechanical load.

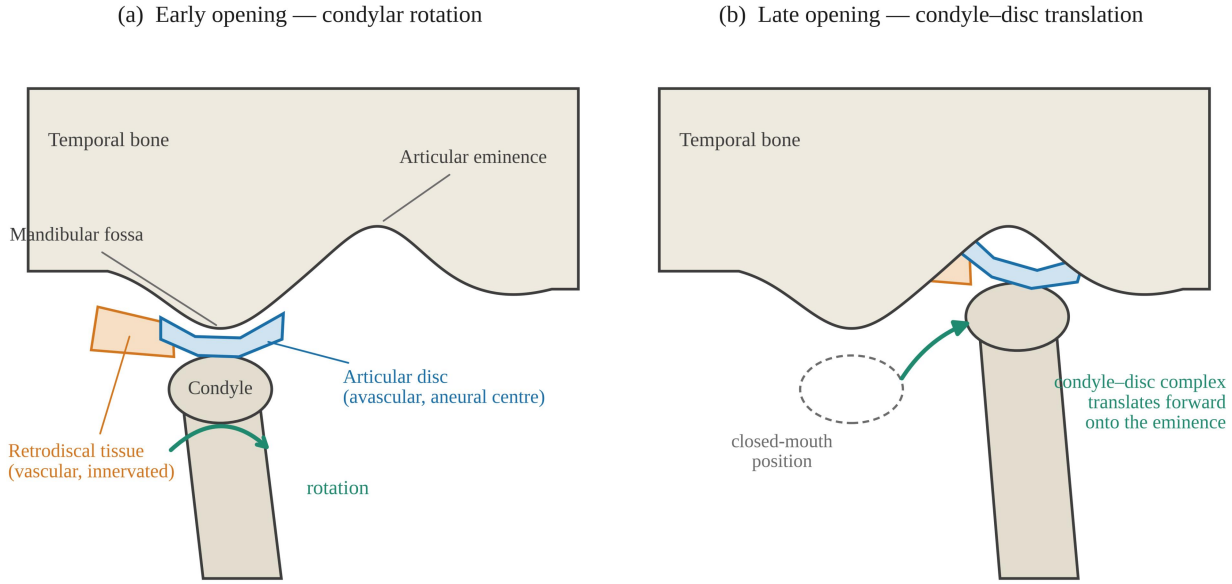


Figure 1. Functional biomechanics of the temporomandibular joint. (a) During early mouth opening, rotation of the condyle within the mandibular fossa predominates. (b) During late opening, the condyle–disc complex translates anteriorly onto the articular eminence. The biconcave articular disc is avascular and aneural centrally, whereas the retrodiscal tissue is vascular and richly innervated and is a principal source of joint pain. Schematic representation; not to anatomical scale.

4. Epidemiology

Recent meta-analyses provide convergent but not identical estimates of TMD frequency (Table 1, Figure 2). A meta-analysis of 74 studies and 172,239 participants reported a global prevalence of 34%, with marked geographical variation (South America 47%, Asia 33%, Europe 29%, North America 26%) and the highest burden in the 18–60-year age stratum (41%) [4]. A subsequent meta-analysis of 27 studies and 20,971 participants estimated the pooled prevalence at 29.5%, with 36.7% in women versus 26.7% in men, corresponding to an approximately 1.75-fold greater likelihood in women [5].

When intra-articular conditions are analysed separately, the pooled prevalence of TMJ disorders reaches 31.1% in adults and the elderly but only 11.3% in children and adolescents; disc displacement with reduction is the single most frequent subtype in both strata (25.9% and 7.4%, respectively), whereas degenerative joint disease shows a steep age gradient (9.8% versus 0.4%) [12] (Table 2).

Symptom-level estimates follow the same hierarchy: myalgia (37.2%) and joint sounds (29.8%) are considerably more common than arthralgia (16.8%) or limited opening and locking (8.1%) [5]. This distribution has a direct clinical implication — the majority of positive findings in a screened population are muscular or acoustic rather than structural, and clicking alone is not an indication for treatment.

Table 1. Pooled prevalence estimates of temporomandibular disorders in recent meta-analyses.

Source	Evidence base	Population	Pooled prevalence	Principal subgroup findings
Valesan et al., 2021 [12]	21 studies (systematic review and meta-analysis)	Adults and elderly; children and adolescents	31.1% (adults); 11.3% (children)	Disc displacement 19.1%; degenerative joint disease 9.8% in adults versus 0.4% in children
Zieliński et al., 2024 [4]	74 studies; 172,239 participants (35,259 cases)	General population, all ages	34.0%	South America 47%, Asia 33%, Europe 29%, North America 26%; 18–60 years 41%, >60 years 36%, ≤18 years 27%
Alqutaibi et al., 2025 [5]	27 studies; 20,971 participants (6,075 cases)	General population, all ages	29.5%	Women 36.7% versus men 26.7% (≈1.75-fold); <18 years 38.5%, ≥18 years 34.1%; Europe 33.8%, Asia 27.9%, North America 19.4%

Note. TMD — temporomandibular disorders; TMJ — temporomandibular joint. Estimates are reported as published; differences reflect variation in case definition, diagnostic instrument and population sampled.

Table 2. Prevalence of intra-articular temporomandibular joint disorders by age stratum.

Diagnostic category	Adults and elderly	Children and adolescents	Clinical comment
Any TMJ disorder	31.1%	11.3%	Approximately threefold higher burden in adults
Disc displacements (all types)	19.1%	8.3%	Most frequent group of intra-articular findings
Disc displacement with reduction	25.9%	7.4%	Most prevalent single subtype; clicking alone is not an indication for treatment
Degenerative joint disease	9.8%	0.4%	Steep age gradient consistent with a cumulative-load and remodelling

Diagnostic category	Adults and elderly	Children and adolescents	Clinical comment
			model

Note. DDwR — disc displacement with reduction; DJD — degenerative joint disease. Data from Valesan et al. [12]. The most prevalent single subtype in both strata is DDwR. Subtype estimates were pooled from differing subsets of the included studies and are therefore not strictly additive: the DDwR estimate is not a subset proportion of the pooled disc-displacement estimate.

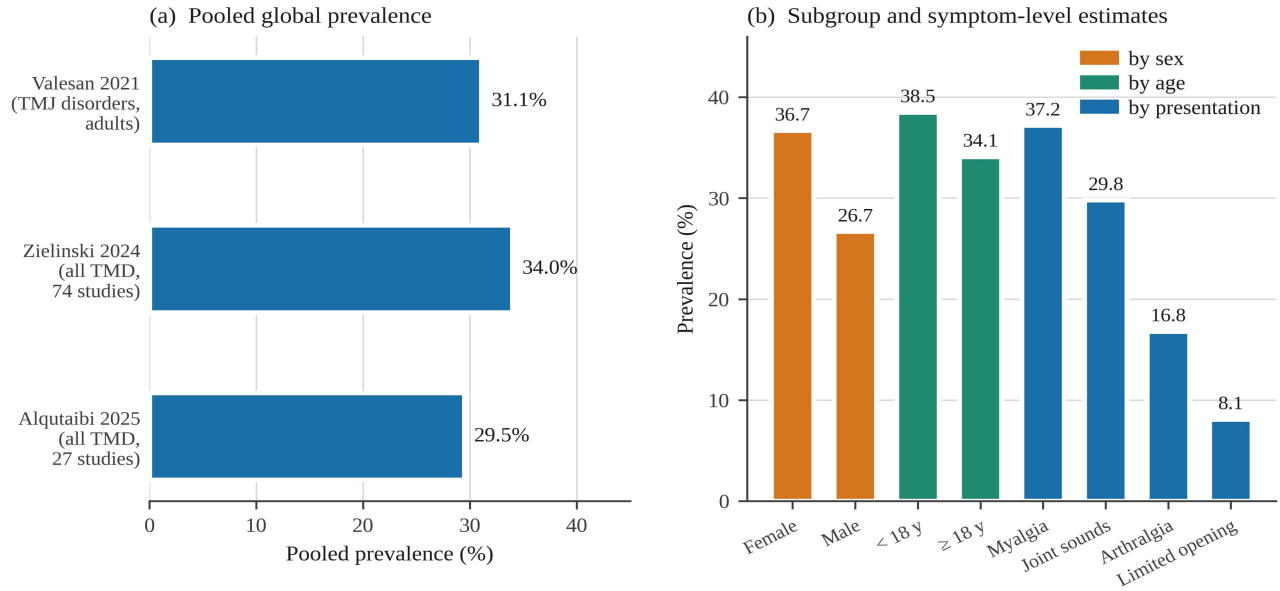


Figure 2. Prevalence of temporomandibular disorders. (a) Pooled global estimates from three recent meta-analyses. (b) Subgroup and symptom-level estimates. Data from Valesan et al. [12], Zieliński et al. [4] and Alqutaibi et al. [5]; subgroup values in panel (b) are from [5].

5. Aetiology and Risk Factors

The most important principle in explaining the aetiology of TMD is the abandonment of single-cause models. A systematic review of 21 cohort studies published between 2005 and 2021 identified female sex, depressive and anxiety symptoms, perceived stress, poor sleep quality, symptoms of obstructive sleep apnoea, comorbid conditions such as irritable bowel syndrome, low back pain and headache disorders, heightened pain perception, orofacial anomalies and both extrinsic and intrinsic injury as factors associated with the subsequent onset of TMD [8] (Table 3). The authors emphasised that heterogeneous methodology across primary studies limits the certainty of these associations.

A distinction must be drawn between a risk factor and a direct cause. Bruxism, for example, may increase the mechanical load transmitted to the TMJ, yet TMD does not develop in every individual with bruxism; contemporary consensus therefore treats bruxism not as a disorder in itself in otherwise healthy individuals but as a behaviour that may act as a modifiable contributing factor within a multifactorial system [13]. Similarly, stress and anxiety are not the sole cause of TMD but may amplify or perpetuate pain.

5.1. Mechanical loading and microtrauma

Because the TMJ is in near-continuous use, its tissues are highly sensitive to mechanical stimuli. Under physiological conditions, mechanical loading supports tissue adaptation. When the intensity or duration of loading exceeds adaptive capacity, pathological mechanotransduction may ensue, establishing a closed pathological cycle: excessive mechanical load → activation of cellular mechanosensors → expression of inflammatory mediators → matrix degradation → deterioration of the mechanical properties of the tissue → further relative overload.

Parafunctional behaviours — prolonged gum chewing, forceful clenching, bruxism, habitually unilateral mastication, nail biting and object biting — may increase this load in susceptible individuals. Mechanical loading should nevertheless not be regarded as a universal cause: disease development is determined jointly by the biological state of the joint tissues, genetic predisposition, inflammatory status and the individual characteristics of pain processing.

5.2. Occlusion

The role of occlusion in TMD has historically been overstated. Current evidence does not support malocclusion as a direct and sole cause of TMD. This has an important clinical corollary: selective grinding of teeth, permanent alteration of the occlusal scheme or irreversible orthodontic and prosthodontic interventions undertaken solely to relieve TMJ pain are not scientifically justified and may cause harm. In the contemporary approach, occlusion is assessed as one component of the patient's stomatognathic system but is not accepted as an automatic cause of TMD [2,14].

5.3. The biopsychosocial model

The biopsychosocial model is central to the modern understanding of TMD. Its biological component encompasses inflammation, trauma, degeneration, mechanical loading, genetic predisposition and muscular dysfunction. The psychological component includes anxiety, depressive symptoms, catastrophising, stress and fear of pain. The social component comprises sleep, occupational activity, family environment, social support and daily functioning [1,6,14].

A critical clinical point follows: it is incorrect to tell a patient that their pain is “psychological”. Psychological state does not invent pain; rather, it exerts a real biological influence on the central mechanisms of pain processing. Sleep disturbance is a further important contributor to the development and chronification of TMD, since sleep deficiency lowers pain thresholds, activates stress systems and alters central processing of nociceptive signals. It is therefore insufficient to examine only the jaw joint in a patient presenting with chronic TMJ pain; sleep quality, nocturnal jaw activity, daytime clenching habits and general fatigue must also be assessed.

Table 3. Factors associated with the onset of temporomandibular disorders in cohort studies.

Domain	Factors identified	Clinical implication
Biological and hormonal	Female sex; hormonal factors (high oestrogen, low prenatal testosterone exposure)	Consistent female predominance across prevalence studies; not modifiable, but relevant to index

Domain	Factors identified	Clinical implication
		of suspicion
Psychological	Depressive symptoms; anxiety symptoms; perceived stress; somatisation; catastrophising	Axis II screening is indicated in every patient with persistent pain; modifiable through cognitive behavioural therapy
Sleep-related	Poor sleep quality; symptoms of obstructive sleep apnoea	Sleep assessment should form part of the routine history in chronic TMJ pain
Pain-related and comorbid	Heightened pain perception; pain on jaw mobility and on palpation; irritable bowel syndrome; low back pain; headache disorders	Suggests shared central pain-processing mechanisms; predicts poorer response to local measures alone
Mechanical and behavioural	Oral parafunction; bruxism; extrinsic and intrinsic injury; orofacial anomalies	Potentially modifiable; bruxism is a contributing behaviour rather than a disease in otherwise healthy individuals [13]

Note. Synthesised from Da-Cas et al. [8] (21 cohort studies, 2005–2021) and Warzocha et al. [14]. The authors of the source review emphasise that methodological heterogeneity across primary studies limits the certainty of these associations; the factors listed are associations with onset and should not be interpreted as proven direct causes.

6. Molecular Pathogenesis of Temporomandibular Joint Osteoarthritis

TMJ osteoarthritis (OA) is among the most complex forms of TMD, and interpreting it merely as “joint wear” is scientifically inadequate. Osteoarthritis is a whole-joint-organ process in which interdependent pathological changes occur in the articular cartilage, the subchondral bone, the synovial membrane, the capsule, the ligaments and the periarticular structures [10,15,16].

Mechanical stress and tissue injury activate synovial cells and lead to the release of mediators, among which IL-1β, TNF-α and IL-6 are the best characterised. These cytokines amplify synovial inflammation, alter chondrocyte metabolism, enhance matrix degradation and sensitise peripheral nociceptors. TMJ osteoarthritis is therefore most appropriately regarded as a degenerative–inflammatory disease rather than a purely mechanical one.

IL-1β is one of the principal catabolic mediators in TMJ OA: it increases the expression of matrix-degrading enzymes in chondrocytes and disrupts normal cartilage turnover. TNF-α, acting together with IL-1β, amplifies the inflammatory cascade and promotes activation of the NF-κB and MAPK signalling pathways, generating the molecular chain IL-1β/TNF-α ↑ → NF-κB/MAPK ↑ → MMP/ADAMTS ↑ → cartilage matrix ↓ [10].

NF-κB governs the transcription of inflammatory genes, while the MAPK family — comprising the ERK, JNK and p38 cascades — controls the cellular response to mechanical and inflammatory signals. Molecular reviews of TMJ OA additionally implicate the Wnt/β-catenin, TGF-β, BMP, FGF, Indian Hedgehog and Notch systems. The Wnt/β-catenin pathway participates in chondrocyte differentiation and bone metabolism, and its pathological activation may contribute to cartilage degradation and subchondral bone change; imbalance of TGF-β and BMP signalling

influences different stages of the degenerative process. The molecular pathogenesis of TMJ OA thus rests not on a single pathway but on several interconnected signalling systems [10,15] (Table 4).

Normal cartilage maintains its mechanical properties largely through type II collagen and proteoglycans. In osteoarthritis catabolic processes come to predominate over anabolic ones: MMP-3, MMP-9 and MMP-13, together with ADAMTS-4 and ADAMTS-5, degrade extracellular matrix components. As the matrix is lost, cartilage progressively forfeits its capacity to retain water, to distribute compressive load and to preserve elasticity, so that mechanical stress increases further and stimulates additional mediator production.

Chondrocytes, the principal cells of articular cartilage, synthesise matrix components and maintain metabolic homeostasis. Under mechanical stress, oxidative stress and inflammatory mediators their metabolic activity is disturbed and apoptosis increases, reducing the intrinsic repair capacity of cartilage. Reactive oxygen species impair mitochondrial function, damage cell membranes and potentiate apoptosis, and a bidirectional relationship exists between oxidative stress and inflammation: oxidative stress → inflammation → mitochondrial dysfunction → further oxidative stress. Osteoarthritis is therefore not simply "matrix breakdown" but a process accompanied by profound alteration of chondrocyte biology [15,16].

Osseous change in TMJ OA is not independent of cartilage pathology, since a continuous biological and mechanical interaction exists between cartilage and subchondral bone. Resorption, sclerosis, alteration of the cortical layer, osteophyte formation and condylar deformation may all be observed; some studies indicate that bone resorption predominates in early disease, whereas reparative remodelling and sclerosis prevail at later stages [16]. The integrated cascade is summarised in Figure 3.

Table 4. Principal molecular mediators and signalling systems implicated in temporomandibular joint osteoarthritis.

Level	Mediator or system	Principal action in TMJ osteoarthritis	Downstream consequence
Pro-inflammatory cytokines	IL-1β	Key catabolic mediator; induces matrix-degrading enzymes in chondrocytes	Disruption of normal cartilage turnover
	TNF-α	Amplifies the inflammatory cascade in synergy with IL-1β	Activation of NF-κB and MAPK signalling
	IL-6, PGE_2, nitric oxide	Sustain synovial inflammation and alter chondrocyte metabolism	Peripheral nociceptor sensitisation
Signalling pathways	NF-κB	Master transcriptional regulator of inflammatory genes	Increased cytokine and protease expression

Level	Mediator or system	Principal action in TMJ osteoarthritis	Downstream consequence
	MAPK (ERK, JNK, p38)	Transduces mechanical and inflammatory signals to the nucleus	Coupling of mechanical overload to catabolic gene expression
	Wnt/β-catenin	Regulates chondrocyte differentiation and bone metabolism	Cartilage degradation and subchondral bone change when pathologically activated
	TGF-β / BMP, FGF, IHH, Notch	Govern tissue development, differentiation and matrix turnover	Imbalance influences distinct stages of degeneration
Proteolytic enzymes	MMP-3, MMP-9, MMP-13	Degrade type II collagen and other ECM components	Loss of tensile and compressive competence of cartilage
	ADAMTS-4, ADAMTS-5	Aggrecanases degrading proteoglycan	Reduced water retention and load distribution
Cellular mechanisms	Chondrocyte apoptosis; ROS and mitochondrial dysfunction	Impair synthesis and self-repair of the matrix	Self-perpetuating degenerative cycle
Osseous compartment	Subchondral bone remodelling	Early resorption followed by reparative sclerosis	Osteophytes, erosion, condylar deformity

Note. ADAMTS — a disintegrin and metalloproteinase with thrombospondin motifs; BMP — bone morphogenetic protein; ECM — extracellular matrix; IHH — Indian Hedgehog; MAPK — mitogen-activated protein kinase; MMP — matrix metalloproteinase; NF-κB — nuclear factor kappa B; ROS — reactive oxygen species; TGF-β — transforming growth factor beta. Compiled from Lu et al. [10], Zhang et al. [15] and Cardoneanu et al. [16].

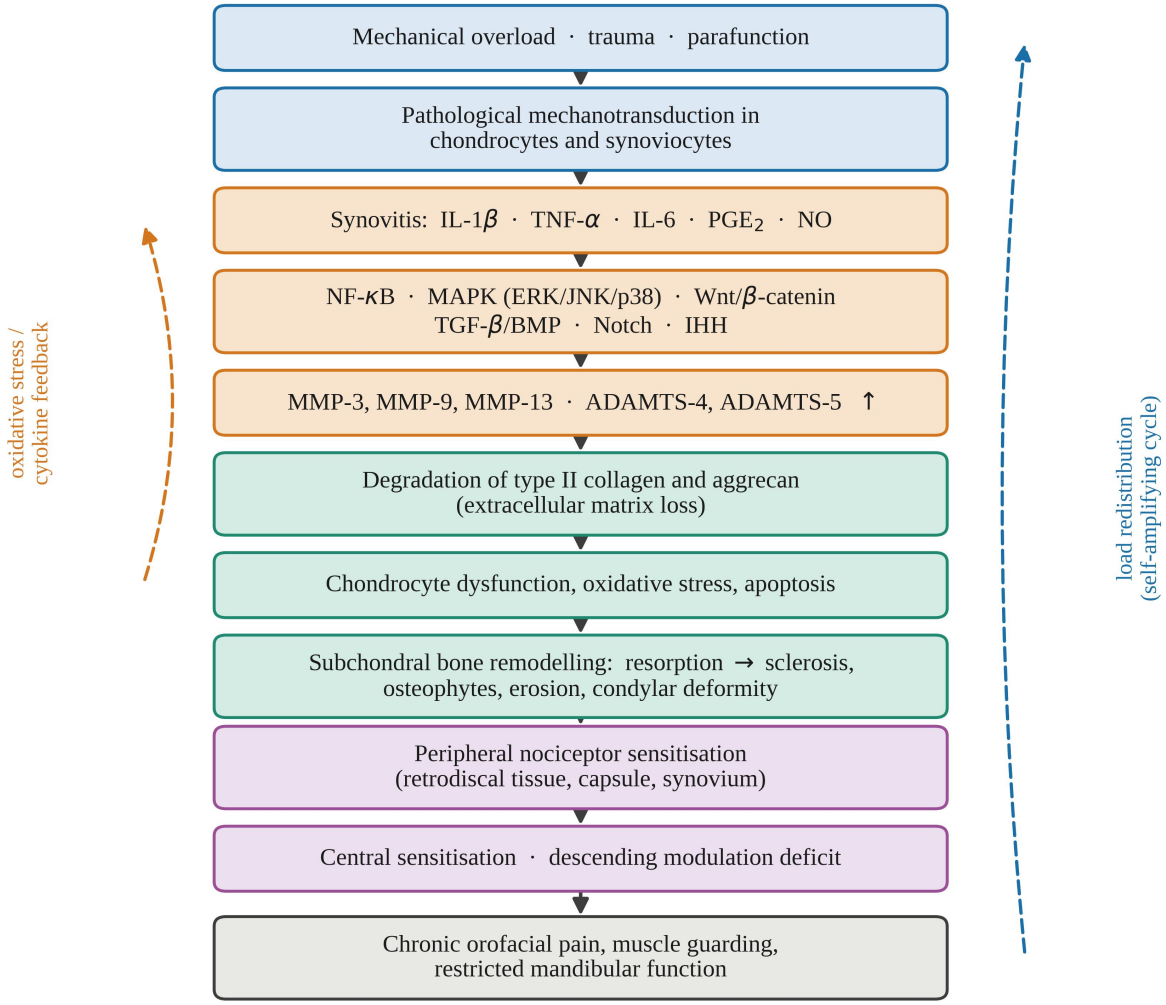


Figure 3. Integrated molecular and neurophysiological cascade in temporomandibular joint osteoarthritis. Mechanical overload initiates pathological mechanotransduction, synovial inflammation and activation of NF-κB and MAPK signalling, leading to matrix degradation, subchondral bone remodelling and, ultimately, peripheral and central sensitisation. Dashed arrows indicate self-amplifying feedback loops. Compiled from Lu et al. [10], Zhang et al. [15] and Cardoneanu et al. [16].

7. Pain Mechanisms

TMJ pain does not originate uniformly from all parts of the joint. The retrodiscal tissues, the capsule, the synovial membrane and the periarticular muscles are particularly rich in nociceptors. During inflammation, bradykinin, prostaglandins, cytokines and other mediators sensitise peripheral nociceptors, so that mechanical stimuli previously perceived as painless may provoke pain.

One of the most important issues in understanding TMD is that the duration of pain is not always proportionate to peripheral structural change. Prolonged nociceptive input may increase neuronal excitability within the central nervous system, a process termed central sensitisation,

which lowers pain thresholds, increases pain intensity, expands the painful area and may heighten sensitivity to pain in other body regions. In some patients with chronic TMJ pain, therefore, no direct correlation is observed between structural joint change and symptom severity [6,7].

This is clinically decisive: correcting a radiographic or magnetic resonance imaging abnormality does not necessarily abolish pain, and the presence of central sensitisation should be evaluated before any irreversible intervention is contemplated.

8. Clinical Forms and Course

TMDs are divided clinically into several principal groups. Myalgia is characterised by pain arising from the masticatory muscles. Arthralgia denotes pain localised to the joint itself, which may be aggravated by functional movement. Disc displacement with reduction involves temporary return of the disc to its normal position during mouth opening and commonly manifests as clicking or popping. Disc displacement without reduction, in which the disc fails to return to its normal position, restricts condylar movement and may produce the clinical state known as closed lock. Degenerative joint disease and osteoarthritis may present with pain, crepitus, restricted movement and structural change [6,9] (Table 5).

The onset of TMD is frequently slow and stepwise. Initially the patient may notice only a click or other sound on jaw movement. Muscle fatigue, pain on chewing or morning discomfort in the jaw region may subsequently appear. As the condition progresses, limitation of mouth opening, alteration of the mandibular movement trajectory, joint pain and concomitant headache may develop. In the chronic stage, pain may become constant or recurrent and may affect sleep and daily functioning.

The most frequently encountered clinical features are preauricular pain; pain on mastication; tenderness of the masseter and temporalis muscles; limitation of mouth opening; asymmetry of jaw movement; clicking, popping and crepitus; joint locking; headache; periauricular discomfort; and reduced masticatory efficiency. Otalgia or headache does not, however, automatically indicate TMD, and differential diagnosis is mandatory.

Table 5. Principal clinical forms of temporomandibular disorders and their characteristic presentation.

Clinical form	Characteristic features	Course and clinical significance
Myalgia	Pain of masticatory muscle origin, modified by function; tenderness of masseter and temporalis on palpation	Most frequent presentation; generally responsive to exercise and behavioural measures
Arthralgia	Pain localised to the joint, aggravated by functional movement and by palpation of the lateral pole	May accompany inflammatory or degenerative intra-articular change
Disc displacement with reduction	Transient return of the disc to its normal relation during opening; clicking or popping	Frequently asymptomatic; clicking alone is not an indication for intervention
Disc displacement	Episodic failure of reduction	Intermediate state; patient

Clinical form	Characteristic features	Course and clinical significance
with reduction and intermittent locking	with transient restriction	education and self-reduction manoeuvres are usually sufficient
Disc displacement without reduction, with limited opening	Persistent non-reduction; restricted condylar translation ("closed lock")	Acute presentations may warrant early mobilisation; many cases adapt functionally over time
Degenerative joint disease / osteoarthritis	Pain, crepitus, restricted movement, radiographic structural change	Chronic and fluctuating; pain severity correlates poorly with radiographic severity
Subluxation	Hypermobility with condylar excursion beyond the eminence and self-reducing open locking	Highest clinical diagnostic accuracy of all DC/TMD categories

Note. DC/TMD — Diagnostic Criteria for Temporomandibular Disorders. Adapted from Schiffman et al. [9] and Ohrbach and Dworkin [6].

9. Diagnosis

The diagnosis of TMD should not rest on instrumental findings alone. The most appropriate approach combines history-taking, clinical examination, functional assessment and, where indicated, imaging. The international DC/TMD system unites two axes: Axis I addresses clinical and biological diagnosis, while Axis II addresses psychosocial and functional factors [9] (Table 6).

Validation of the DC/TMD protocol against reference standards demonstrated high diagnostic accuracy for pain-related diagnoses — myalgia (sensitivity 0.90, specificity 0.99), arthralgia (0.89/0.98), myofascial pain with referral (0.86/0.98) and headache attributed to TMD (0.89/0.87) — and for subluxation (0.98/1.00). Sensitivity is substantially lower for intra-articular diagnoses established clinically: disc displacement with reduction 0.34, disc displacement with reduction with intermittent locking 0.38, disc displacement without reduction without limited opening 0.54 and degenerative joint disease 0.55 [9] (Figure 4). These figures justify the selective use of imaging: clinical criteria are sufficient for painful TMD, whereas structural diagnoses require imaging confirmation when the result will change management.

During the history, the onset, duration, localisation and intensity of pain, its relation to mastication and jaw movement, and diurnal variation are established. Information is also obtained regarding trauma, parafunction, bruxism, sleep disturbance, stress, headache, other chronic pain syndromes and rheumatic disease. On clinical examination the joint is palpated, maximum and pain-free mouth opening are measured, and lateral and protrusive excursions are assessed. Palpation of the masseter and temporalis muscles identifies muscular pain. The opening trajectory is evaluated for deviation and deflection, and joint sounds are characterised as clicking or crepitus.

Magnetic resonance imaging (MRI) is the principal method for evaluating the soft-tissue components of the joint, permitting assessment of disc position, disc displacement, the retrodiscal tissues, joint effusion and synovial change. Computed tomography (CT) and cone-beam computed tomography (CBCT) are used mainly to assess osseous components, including condylar erosion, osteophytes, subchondral sclerosis, deformity, bony ankylosis and cortical change. The two modalities are complementary rather than interchangeable (Table 7).

Table 6. Structure of the DC/TMD assessment protocol and the practical consequences of its measured diagnostic accuracy.

Axis	Domain assessed	Instruments and procedures	Consequence for practice
Axis I	Pain-related diagnoses: myalgia, local myalgia, myofascial pain with referral, arthralgia, headache attributed to TMD	Standardised history; palpation of masseter and temporalis with calibrated force; pain-free and maximum opening; provocation on movement	Clinical criteria are sufficient (sensitivity 0.86–0.90, specificity 0.87–0.99); imaging adds nothing to the diagnosis
Axis I	Intra-articular diagnoses: DDwR, DDwR with intermittent locking, DDwoR with and without limited opening, degenerative joint disease, subluxation	Joint sounds on opening, closing and excursion; deviation and deflection; measured opening; self-report of locking	Except for subluxation (0.98/1.00) and DDwoR with limited opening (0.80/0.97), clinical sensitivity is low (0.34–0.55); MRI for disc status and CBCT for osseous change are required when the structural diagnosis would change management
Axis II	Pain intensity and interference; jaw functional limitation; psychological distress; parafunctional behaviour	Graded Chronic Pain Scale; Jaw Functional Limitation Scale; PHQ-9; GAD-7; Oral Behaviours Checklist	Identifies patients at risk of chronification in whom cognitive behavioural and multimodal care should be prioritised

Note. CBCT — cone-beam computed tomography; DDwR — disc displacement with reduction; DDwoR — disc displacement without reduction; GAD-7 — Generalised Anxiety Disorder 7-item scale; MRI — magnetic resonance imaging; PHQ-9 — Patient Health Questionnaire 9-item scale. Accuracy values underlying the last column are shown in Figure 4; a pragmatic threshold for a stand-alone clinical diagnosis is sensitivity ≥ 0.70 with specificity ≥ 0.95 [9].

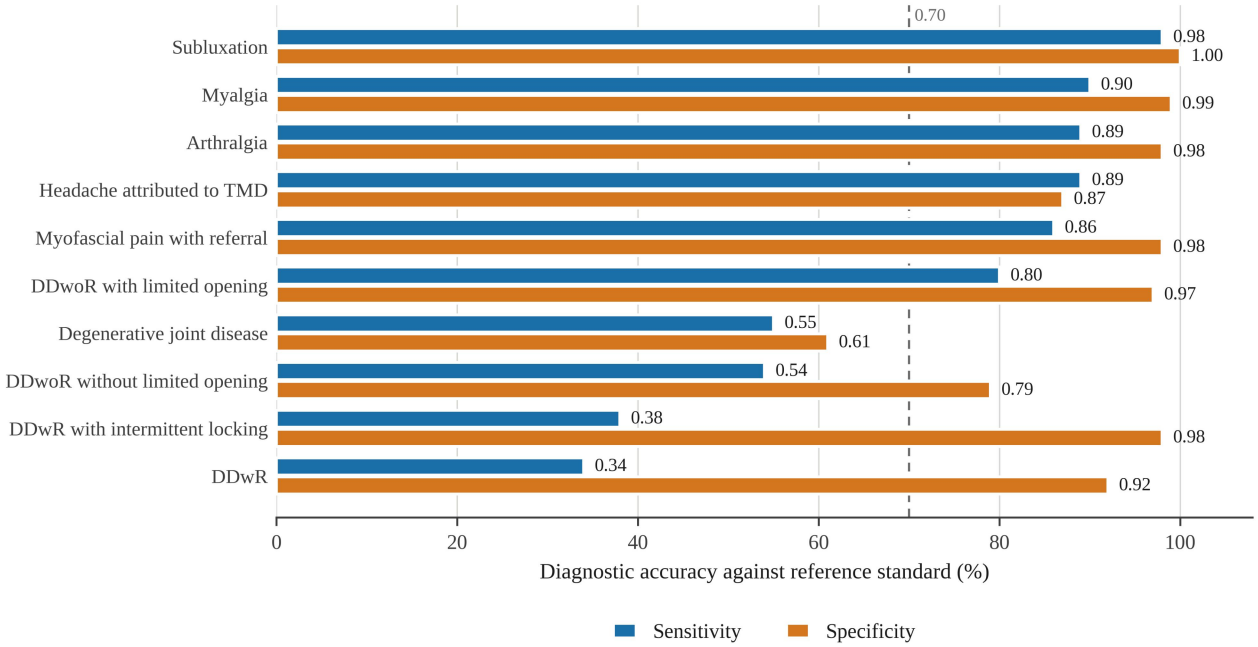


Figure 4. Diagnostic accuracy of DC/TMD Axis I criteria against reference standards. Pain-related diagnoses and subluxation show high sensitivity and specificity, whereas the sensitivity of clinically established intra-articular diagnoses is limited. The dashed line marks a sensitivity of 0.70. DDwR — disc displacement with reduction; DDwoR — disc displacement without reduction. Data from Schiffman et al. [9].

Table 7. Imaging modalities in the assessment of temporomandibular disorders.

Modality	Principal indication	Strengths	Limitations
MRI	Suspected disc pathology, joint effusion, retrodiscal inflammation	Reference standard for disc position and soft tissue; no ionising radiation; dynamic open/closed sequences possible	Cost and availability; limited depiction of fine cortical detail; motion sensitivity
CBCT	Suspected degenerative osseous change, erosion, osteophyte, ankylosis	High spatial resolution of cortical bone; lower dose than multislice CT; chairside availability	Does not visualise the disc or soft tissue; limited soft-tissue contrast
Multislice CT	Complex osseous pathology, trauma, neoplasia, preoperative planning	Superior assessment of extensive bone and adjacent structures	Higher radiation dose; rarely required for uncomplicated TMD
Panoramic radiography	Screening for gross osseous change and	Widely available; low dose	Low sensitivity for early degenerative

Modality	Principal indication	Strengths	Limitations
	exclusion of dental causes		change; geometric distortion of the condyle

Note. CBCT — cone-beam computed tomography; CT — computed tomography; MRI — magnetic resonance imaging. Imaging is indicated when the anticipated finding would change management; it is not required to establish a diagnosis of myalgia or arthralgia [9].

9.1. Differential diagnosis

Pain in the TMJ region does not invariably originate from the joint. Differential diagnosis must consider pulpitis, apical periodontitis, otitis, sinusitis, trigeminal neuralgia, migraine, tension-type headache, cervicogenic pain, rheumatoid arthritis, psoriatic arthritis, salivary gland pathology and neoplastic processes (Table 8). The diagnosis of TMD must therefore be clinically substantiated rather than assumed from the site of pain.

Table 8. Differential diagnosis of pain in the temporomandibular joint region.

Category	Conditions	Discriminating features
Odontogenic	Pulpitis; apical periodontitis	Pain reproduced by thermal or percussion testing; localised to a tooth; unrelated to jaw function
Otological and sinonasal	Otitis media and externa; sinusitis	Otoscopic or nasal findings; pain unrelated to mandibular movement; systemic signs
Neuropathic and primary headache	Trigeminal neuralgia; migraine; tension-type headache	Paroxysmal electric-shock quality with trigger zones (neuralgia); photophobia, nausea and typical episodic pattern (migraine)
Cervical	Cervicogenic pain; referred myofascial pain from the neck	Reproduced by cervical movement or palpation of cervical trigger points
Systemic inflammatory	Rheumatoid arthritis; psoriatic arthritis; other spondyloarthritides	Polyarticular involvement; morning stiffness; serological and radiographic markers
Salivary and neoplastic	Salivary gland pathology; benign and malignant tumours	Swelling, sialorrhoea or xerostomia; progressive pain, neurological deficit, weight loss; imaging is mandatory

Note. Otalgia or headache in the preauricular region does not in itself establish a diagnosis of temporomandibular disorder; differential diagnosis is mandatory before treatment [3,9].

10. Management

The governing principle of contemporary management is sequential: reversible and minimally invasive measures first, and invasive procedures only where clearly indicated. In chronic TMJ pain — that is, pain persisting for three months or longer — treatment should be individualised and multimodal.

A network meta-analysis supporting the 2023 BMJ Rapid Recommendations included 153 randomised trials with 8,713 participants and compared 59 interventions or intervention combinations, using a minimally important difference of 1 cm on a 10 cm visual analogue scale [11]. The largest reductions in pain relative to placebo or sham were observed with cognitive behavioural therapy combined with biofeedback or relaxation (mean difference −2.62 cm, 95% credible interval −3.03 to −2.20; moderate certainty), therapist-assisted jaw mobilisation (−2.57 cm, −3.18 to −1.96; moderate certainty) and manual trigger-point therapy (−2.08 cm, −2.31 to −1.84; moderate certainty). Supervised jaw exercise with stretching achieved −1.55 cm (−1.99 to −1.11) with high certainty, and usual care alone — education, reassurance and home exercise — achieved −1.47 cm (−1.69 to −1.25) (Table 9, Figure 5).

Accordingly, the guideline issued strong recommendations in favour of cognitive behavioural therapy with or without biofeedback or relaxation, therapist-assisted mobilisation, manual trigger-point therapy, supervised postural exercise, supervised jaw exercise and stretching, and usual care. Conditional recommendations in favour were made for manipulation, supervised jaw exercise with mobilisation, cognitive behavioural therapy combined with NSAIDs, manipulation with postural exercise and acupuncture. Conditional recommendations against were issued for reversible occlusal splints, arthrocentesis, low-level laser therapy, transcutaneous electrical nerve stimulation, gabapentin, botulinum toxin injection, hyaluronic acid injection, relaxation therapy as monotherapy, trigger-point injection, acetaminophen, topical capsaicin, biofeedback as monotherapy, corticosteroid injection, benzodiazepines and β-blockers. Strong recommendations against were issued for irreversible oral splints, discectomy and the combination of NSAIDs with opioids [11,17] (Figure 6).

10.1. Patient education and self-management

Explaining the mechanism of the disorder is the first therapeutic step. Patients should understand that TMD is generally not a life-threatening condition; that clicking alone does not always require treatment; that pain intensity does not always correspond to the degree of structural change on imaging; that reducing sustained clenching and excessive parafunction may be beneficial; and that temporarily limiting hard foods, reducing gum chewing and avoiding extreme mouth opening may reduce symptoms in some patients.

10.2. Therapeutic exercise and manual therapy

Therapeutic exercise is a core component of conservative management, with the objectives of reducing pain, restoring range of motion, improving muscular coordination, normalising mandibular movement and addressing postural factors. The network meta-analysis demonstrated that therapist-supervised mandibular mobilisation and manual trigger-point therapy are among the most effective interventions for pain reduction [11].

10.3. Cognitive behavioural therapy

The purpose of cognitive behavioural therapy in chronic TMJ pain is not to classify the pain as psychological but to reduce fear of pain, catastrophising, maladaptive stress responses and avoidance of movement. The 2023 guideline issued a strong recommendation in favour of cognitive behavioural therapy for chronic TMD pain [17].

10.4. Pharmacotherapy

Pharmacological treatment is selected according to the overall clinical status of the patient. In acute presentations or where an inflammatory component predominates, NSAIDs may provide short-term symptomatic relief, although the pooled effect in chronic TMD pain was modest (mean difference −0.76 cm, −1.15 to −0.38; low certainty) [11]. Gastrointestinal, renal and cardiovascular risks and interactions with other medications must be considered. Long-term and unsupervised pharmacotherapy is inappropriate in chronic TMJ pain, and a strong recommendation against the combined use of NSAIDs and opioids has been issued [17].

10.5. Occlusal splints

Reversible intraoral splints may be used to manage symptoms in selected patients, but they are not a universal treatment for all forms of TMD; the pooled estimate for reversible splints was −1.81 cm (−2.01 to −1.60) with low certainty and a conditional recommendation against routine use [11,17]. The cardinal principle is avoidance of irreversible occlusal change. This position reflects the transition from the older concept that TMD can be cured by altering the bite to an evidence-based model of care.

10.6. Arthrocentesis, arthroscopy and injectable therapies

Arthrocentesis is a minimally invasive procedure intended to lavage the joint space and to reduce mechanical and inflammatory components. It may be used in selected clinical situations, but its universal superiority in chronic TMJ pain has not been established, and the guideline issued a conditional recommendation against it [17]. Arthroscopy permits visualisation and, in some cases, treatment of intra-articular pathology and is considered in selected patients with a clear indication in whom conservative treatment has failed.

Botulinum toxin has been studied particularly in symptoms associated with masticatory muscle hyperactivity, but evidence on long-term efficacy and safety is inconsistent (mean difference −1.46 cm, −1.79 to −1.13; low certainty), and it is not regarded as a universal first-line treatment [11,17]. Hyaluronic acid and platelet-rich plasma are being actively investigated as biological and regenerative options in TMJ osteoarthritis, with the theoretical aims of reducing inflammation, improving the synovial environment, relieving pain and stimulating tissue regeneration. Results across studies are heterogeneous, and high-quality clinical evidence remains insufficient to establish platelet-rich plasma or mesenchymal stem-cell-based therapies as standard regenerative treatment [16].

10.7. Surgical treatment

Surgery is not a routine first-line treatment for TMD. It is considered in ankylosis, severe structural deformity, neoplasia, severe traumatic injury and selected structural pathologies unresponsive to conservative care. Assessment of both peripheral and central components of pain before surgery is essential, because in the presence of chronic central sensitisation the surgical correction of an anatomical abnormality may fail to abolish pain.

10.8. Outcome assessment

The efficacy of TMD treatment should not be judged by pain reduction alone. A composite assessment is required, covering pain intensity and duration; function, including mouth opening, mastication and mandibular movement; psychological status, including anxiety, depressive symptoms and catastrophising; sleep quality and duration; and quality of life in daily and social activity (Table 10). This is the practical expression of the biopsychosocial approach.

Table 9. Network meta-analysis estimates of pain reduction for chronic temporomandibular disorder pain, with the corresponding guideline recommendations.

Intervention	MD (95% CrI), cm	RD achieving ≥ MID	Certainty	Recommendation
CBT with biofeedback or relaxation	−2.62 (−3.03 to −2.20)	36%	Moderate	Strong for
Therapist-assisted jaw mobilisation	−2.57 (−3.18 to −1.96)	36%	Moderate	Strong for
Arthrocentesis	−2.12 (−3.06 to −1.17)	32%	Low	Conditional against
Manual trigger-point therapy	−2.08 (−2.31 to −1.84)	32%	Moderate	Strong for
Acupuncture	−2.04 (−2.38 to −1.71)	31%	Low	Conditional for
CBT alone	−1.88 (−2.42 to −1.35)	30%	Moderate	Strong for
Reversible occlusal splint	−1.81 (−2.01 to −1.60)	29%	Low	Conditional against
Supervised postural exercise	−1.56 (−2.33 to −0.79)	26%	Moderate	Strong for
Supervised jaw exercise and stretching	−1.55 (−1.99 to −1.11)	26%	High	Strong for
Usual care (education, home exercise)	−1.47 (−1.69 to −1.25)	25%	Moderate	Strong for
Botulinum toxin injection	−1.46 (−1.79 to −1.13)	25%	Low	Conditional against

Intervention	MD (95% CrI), cm	RD achieving ≥ MID	Certainty	Recommendation
NSAIDs	−0.76 (−1.15 to −0.38)	14%	Low	Conditional against

Note. CBT — cognitive behavioural therapy; CrI — credible interval; MD — mean difference; MID — minimally important difference; NSAIDs — non-steroidal anti-inflammatory drugs; RD — risk difference. Estimates versus placebo or sham on a 10 cm visual analogue scale, from the network meta-analysis of 153 trials and 8,713 participants comparing 59 interventions [11]; MID = 1.0 cm. Recommendation direction from the accompanying clinical practice guideline [17].

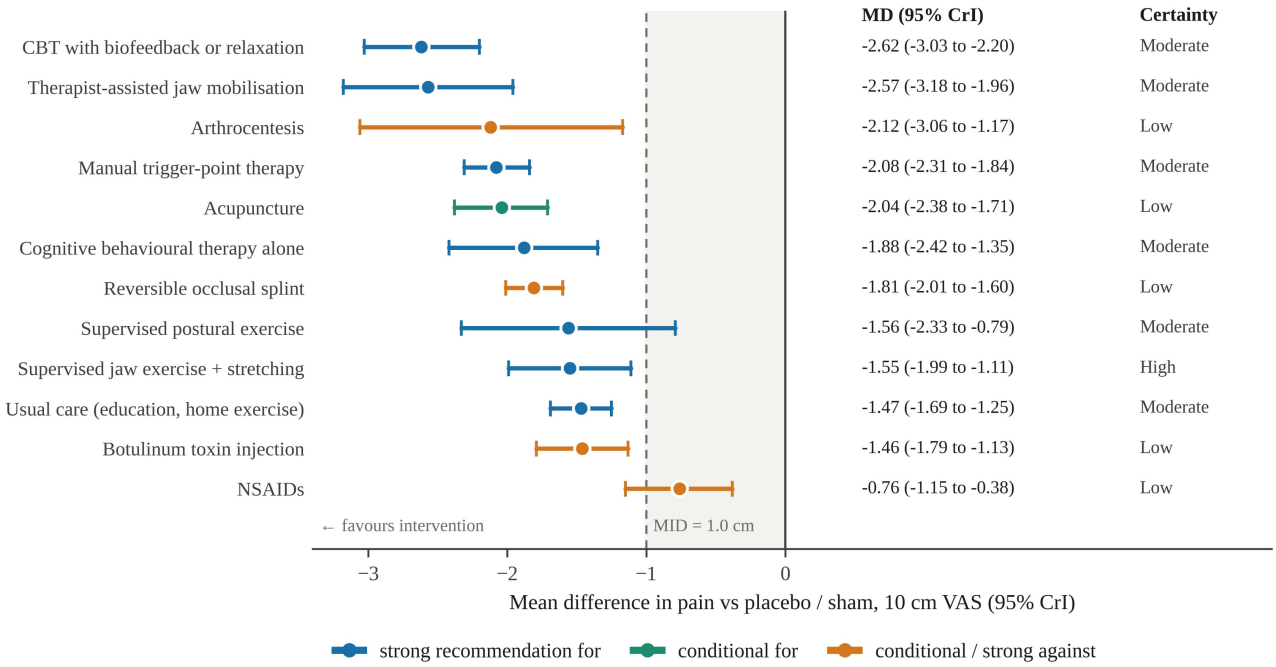


Figure 5. Effect of selected interventions on chronic temporomandibular disorder pain relative to placebo or sham. Points represent mean differences on a 10 cm visual analogue scale with 95% credible intervals; the dashed line marks the minimally important difference of 1.0 cm, and the shaded band denotes effects smaller than that threshold. Colour indicates the direction of the corresponding guideline recommendation. Data from Yao et al. [11] and Busse et al. [17].

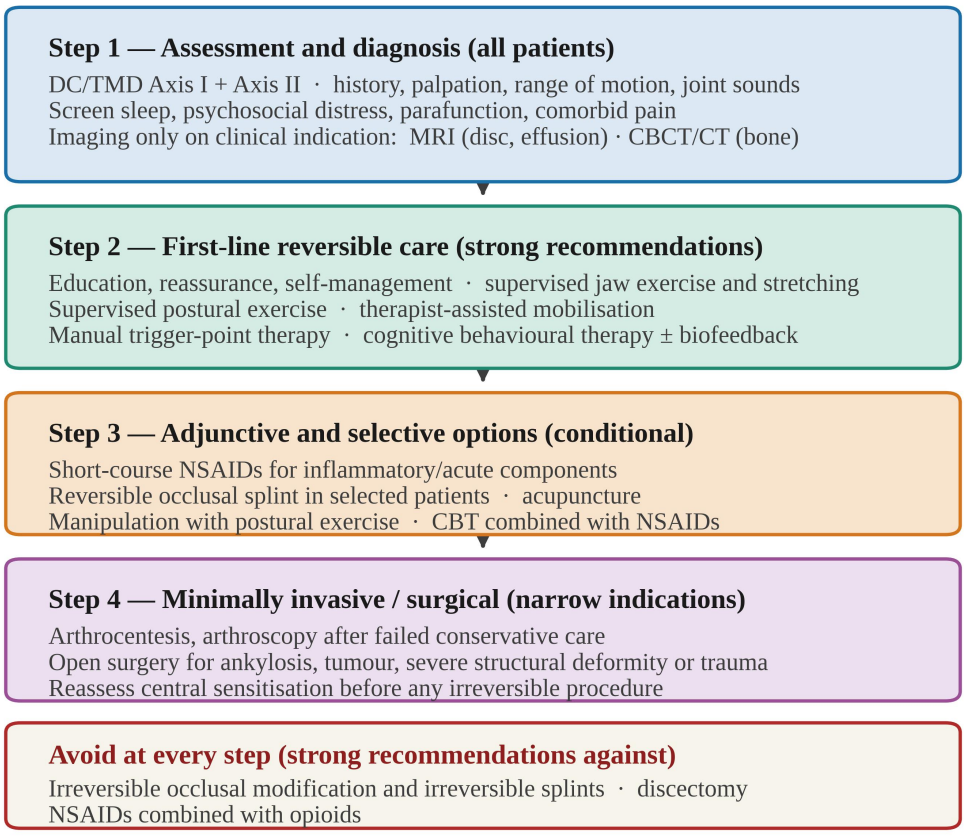


Figure 6. Stepped-care algorithm for the management of temporomandibular disorders, based on the 2023 clinical practice guideline and its supporting network meta-analysis [11,17]. Management proceeds from assessment and reversible first-line care to adjunctive and, exceptionally, invasive options; three interventions carry strong recommendations against use at any stage.

Table 10. Recommended outcome domains for the assessment of treatment response in temporomandibular disorders.

Domain	What is assessed	Practical measurement
Pain	Intensity, duration, frequency and interference	10 cm VAS or 0–10 numerical rating scale; improvement of ≥ 1.0 cm is clinically important
Function	Mouth opening, mastication, mandibular movement	Pain-free and maximum opening in millimetres; lateral and protrusive excursions; Jaw Functional Limitation Scale
Psychological status	Anxiety, depressive symptoms, catastrophising	DC/TMD Axis II instruments (PHQ-9, GAD-7, Pain Catastrophizing Scale)
Sleep	Quality and duration of sleep;	Structured sleep history; validated

Domain	What is assessed	Practical measurement
	nocturnal jaw activity	sleep-quality questionnaire; screening for obstructive sleep apnoea
Quality of life	Daily, occupational and social functioning	Oral Health Impact Profile or an equivalent generic instrument

Note. VAS — visual analogue scale. The minimally important difference for pain in chronic temporomandibular disorder is 1.0 cm on a 10 cm VAS [11].

11. Discussion

Temporomandibular disorders constitute a clinically and biologically complex group of conditions. The contemporary literature demonstrates that mechanical, inflammatory, immune, neurophysiological and psychosocial mechanisms interact in their development. The interaction between mechanical loading and inflammation is particularly important in TMJ osteoarthritis: mechanical stress activates pathological signalling pathways within cells, while inflammation alters the mechanical properties of cartilage and subchondral bone and reduces the load-bearing tolerance of the joint. The disease thereby becomes a self-amplifying pathological cycle [10,15,16].

At the same time, there is no consistent direct relationship between structural change and pain. In chronic pain, peripheral inflammation and central sensitisation may coexist, so that management based solely on MRI or CT findings is clinically inadequate. The value of the DC/TMD system lies precisely here: it permits assessment not only of joint or muscle pathology but also of the patient's pain experience and psychosocial state [9]. The accuracy data reinforce this point — the protocol performs well for pain-related diagnoses yet has limited sensitivity for intra-articular diagnoses, which is an argument for imaging when a structural diagnosis would alter management, and against imaging when it would not.

A paradigmatic shift is also evident in treatment. Where TMD was formerly addressed chiefly by occlusal alteration or surgical “mechanical correction”, current evidence prioritises patient education, exercise, physiotherapy, pain management and, where appropriate, psychological approaches [11,17]. The observation that usual care alone — education, reassurance and home exercise — exceeded the minimally important difference is arguably the single most consequential finding for routine dental practice, since it is deliverable in any clinical setting at negligible cost and without risk.

These results indicate that an individualised, multimodal approach is preferable to a “one procedure for one disease” model. They also carry a health-systems implication for settings in which access to specialist orofacial pain services is limited: the interventions with the strongest evidence base are predominantly behavioural and physical rather than technological, and can be implemented by trained general dental practitioners and physiotherapists.

12. Limitations

This review is narrative rather than systematic; although the search strategy was structured, no formal risk-of-bias assessment or quantitative pooling was performed, and selection bias cannot be excluded. The prevalence estimates cited derive from meta-analyses that differ in case definition, diagnostic instrument and population, which accounts for much of the variation between them and limits direct comparison. Evidence for the majority of interventions other than exercise-based and

psychological therapies remains of low certainty, and long-term outcome data beyond 12 months are sparse. Finally, no epidemiological data specific to the population of Uzbekistan were identified, and the generalisability of international estimates to this setting is therefore unknown.

13. Conclusions

Temporomandibular disorders are a group of multifactorial conditions characterised by biological, mechanical and neurophysiological alterations of the temporomandibular joint, the masticatory muscles and associated orofacial structures, classified under ICD-10 code M26.6. Their aetiology involves the interaction of mechanical loading, trauma, parafunction, bruxism, sleep disturbance, psycho-emotional factors, genetic predisposition and other chronic pain conditions; explaining the disease solely by occlusal disturbance or joint "wear" is inconsistent with current evidence.

In TMJ osteoarthritis, the interaction between mechanical stress and inflammation leads to elevation of IL-1β, TNF-α and IL-6, activation of the NF-κB and MAPK signalling systems, excessive expression of MMP and ADAMTS enzymes, degradation of the extracellular matrix, chondrocyte dysfunction and subchondral bone remodelling. These processes reinforce one another and create the substrate for degenerative joint change and chronic pain.

In chronic painful forms, central sensitisation operates alongside peripheral inflammation, and clinical symptoms therefore do not always correspond to the severity of radiological change. In diagnosis, the DC/TMD system standardises clinical assessment and allows pain and psychosocial components to be evaluated together; MRI and CT/CBCT are important complementary methods for soft-tissue and osseous components respectively.

Contemporary management must be reversible, minimally invasive and individualised. Patient education, self-management, therapeutic jaw and postural exercise, manual therapy and cognitive behavioural therapy occupy a central position in the management of chronic TMJ pain. Invasive procedures, irreversible occlusal alterations and surgery should be considered only where clear clinical indications exist. The essential condition for effective management of TMD is the implementation of the principle of treating the patient rather than the joint: anatomical change, functional impairment, pain mechanism, psychosocial factors and quality of life must be assessed as a single clinical system.

Declarations

Author contributions. Conceptualisation and supervision: U.A. Khatamov. Literature search and data extraction: D.Kh. Sunnatov, Y.A. Bekmirzayeva, N.Kh. Abdukhalimova. Table and figure preparation: A.A. Toyirova, S.S. Alikhodjaev, Z.Z. Rakhmiddinov. Drafting of the manuscript: D.Kh. Sunnatov, U.A. Khatamov. Critical revision: all authors. All authors have read and approved the final version of the manuscript.

Funding. This research received no specific grant from any funding agency in the public, commercial or not-for-profit sectors.

Conflicts of interest. The authors declare no conflicts of interest.

Ethical approval. Not applicable. This review synthesises previously published aggregate data and involved no human participants or animals.

Data availability. All data discussed in this review are available in the cited published sources.

References

1. Suvinen TI, Reade PC, Kemppainen P, Könönen M, Dworkin SF. Review of aetiological concepts of temporomandibular pain disorders: towards a biopsychosocial model for integration of physical disorder factors with psychological and psychosocial illness impact factors. Eur J Pain. 2005;9(6):613–33. doi:10.1016/j.ejpain.2005.01.012
2. Ohrbach R, Dworkin SF. The evolution of TMD diagnosis: past, present, future. J Dent Res. 2016;95(10):1093–101. doi:10.1177/0022034516653922
3. International Classification of Orofacial Pain, 1st edition (ICOP). Cephalalgia. 2020;40(2):129–221. doi:10.1177/0333102419893823
4. Zieliński G, Pająk-Zielińska B, Ginszt M. A meta-analysis of the global prevalence of temporomandibular disorders. J Clin Med. 2024;13(5):1365. doi:10.3390/jcm13051365
5. Alqutaibi AY, Alassaf MS, Radi MA, et al. Global prevalence of temporomandibular disorders: a systematic review and meta-analysis. J Oral Facial Pain Headache. 2025;39(2):48–65. doi:10.22514/jofph.2025.025
6. Ohrbach R, Dworkin SF. AAPT diagnostic criteria for chronic painful temporomandibular disorders. J Pain. 2019;20(11):1276–92. doi:10.1016/j.jpain.2019.04.003
7. Sharma S, Breckons M, Brönnimann Lambelet B, et al. Challenges in the clinical implementation of a biopsychosocial model for assessment and management of orofacial pain. J Oral Rehabil. 2020;47(1):87–100. doi:10.1111/joor.12871
8. Da-Cas CD, Valesan LF, do Nascimento LP, et al. Risk factors for temporomandibular disorders: a systematic review of cohort studies. Oral Surg Oral Med Oral Pathol Oral Radiol. 2024;138(4):502–15. doi:10.1016/j.oooo.2024.06.007
9. Schiffman E, Ohrbach R, Truelove E, et al. Diagnostic Criteria for Temporomandibular Disorders (DC/TMD) for clinical and research applications: recommendations of the International RDC/TMD Consortium Network and Orofacial Pain Special Interest Group. J Oral Facial Pain Headache. 2014;28(1):6–27. doi:10.11607/jop.1151
10. Lu K, Ma F, Yi D, Yu H, Tong L, Chen D. Molecular signaling in temporomandibular joint osteoarthritis. J Orthop Translat. 2022;32:21–7. doi:10.1016/j.jot.2021.07.001
11. Yao L, Sadeghirad B, Li M, et al. Management of chronic pain secondary to temporomandibular disorders: a systematic review and network meta-analysis of randomised trials. BMJ. 2023;383:e076226. doi:10.1136/bmj-2023-076226
12. Valesan LF, Da-Cas CD, Réus JC, et al. Prevalence of temporomandibular joint disorders: a systematic review and meta-analysis. Clin Oral Investig. 2021;25(2):441–53. doi:10.1007/s00784-020-03710-w
13. Lobbezoo F, Ahlberg J, Raphael KG, et al. International consensus on the assessment of bruxism: report of a work in progress. J Oral Rehabil. 2018;45(11):837–44. doi:10.1111/joor.12663
14. Warzocha J, Gadomska-Krasny J, Mrowiec J. Etiologic factors of temporomandibular disorders: a systematic review of literature containing diagnostic criteria for TMD. Healthcare (Basel). 2024;12(5):575. doi:10.3390/healthcare12050575

15. Zhang J, Mu XT, Zhang X, Li CY. Potential pathological and molecular mechanisms of temporomandibular joint osteoarthritis. J Dent Sci. 2023;18(3):959–71. doi:10.1016/j.jds.2023.04.002
16. Cardoneanu A, Macovei LA, Burlui AM, et al. Temporomandibular joint osteoarthritis: pathogenic mechanisms involving the cartilage and subchondral bone, and potential therapeutic strategies for joint regeneration. Int J Mol Sci. 2023;24(1):171. doi:10.3390/ijms24010171
17. Busse JW, Casassus R, Carrasco-Labra A, et al. Management of chronic pain associated with temporomandibular disorders: a clinical practice guideline. BMJ. 2023;383:e076227. doi:10.1136/bmj-2023-076227