Clinical Medicine and Diagnostics 2026, 14(1): 1-5
DOI: 10.5923/j.cmd.20261401.01

Dentistry in Ehlers–Danlos Syndrome: Temporomandibular Joint Dysfunction, Occlusal Instability, and Conservative Management

Christopher Brown DDS, MPS

Director of Innovation, Neuraxis Inc, Carmel, In. USA

Abstract Ehlers-Danlos syndrome (EDS) comprises a group of heritable connective tissue disorders caused by abnormalities in collagen structure or biosynthesis. The 2017 international classification recognizes 13 subtypes with variable genetic and clinical features. [1] Hypermobile EDS is the most prevalent subtype and is most commonly associated with temporomandibular disorders, reflecting generalized joint hypermobility and connective tissue laxity. [2] Classical and vascular subtypes are less frequently associated with TMD but may influence dental and temporomandibular management because of tissue fragility and altered healing responses. [1] Across subtypes, impaired connective tissue integrity contributes to joint instability and abnormal biomechanical loading, providing a mechanistic basis for temporomandibular dysfunction in affected individuals. [1] Temporomandibular disorders (TMD) are common in hypermobile phenotypes and may present as capsular laxity with recurrent subluxation, disc displacement, myofascial pain, and dynamic occlusal changes. [6,7,16] This manuscript provides a clinically oriented review of EDS-associated TMD emphasizing diagnostic pitfalls, conservative treatment sequencing, and stabilization orthotic design tailored to hypermobile patients. Using contemporary conceptual models of TMD that integrate instability, neuromuscular control, central pain processing, and sleep physiology. [7,10,11,12] Conservative care combining education, graded rehabilitation, sleep optimization, and stabilization orthotics—preferably lower-arch orthotics with careful monitoring—can reduce symptoms while minimizing iatrogenic occlusal change. [18,20,21] Recognizing the connective tissue phenotype and avoiding irreversible occlusal interventions during symptomatic phases are central to safe TMD management.

Keywords Ehlers–Danlos syndrome, Hypermobility, Temporomandibular disorders, Occlusal orthotic, Occlusal instability, Myofascial pain, Conservative management [16,11]

1. Introduction

Ehlers–Danlos Syndrome alters the mechanical environment in which the masticatory system operates. Capsular tissues, ligaments, and tendons demonstrate altered tensile properties, and many patients exhibit impaired proprioception and reduced ability to stabilize joints at the end of the normal range of motion. [1,2,4] In the temporomandibular joint (TMJ), stability is achieved through a balance of passive restraint and active neuromuscular control. When passive restraints are compliant, the burden of stability shifts toward sustained muscle co-contraction. While protective in the short term, this response can become maladaptive, producing muscular fatigue, myofascial pain, and increased joint compression. [3,8,9]

Temporomandibular disorders represent a spectrum involving the masticatory muscles, the TMJ, the capsule, and associated structures. Contemporary frameworks emphasize interacting drivers—nociceptive input, motor control patterns, central pain processing, psychosocial context, and sleep physiology—rather than a purely occlusal etiology. [7,10] In hypermobile phenotypes, instability is often a dominant driver. Patients may report an occlusion (bite) that feels different across several days or even within the same day, a feature that can be misinterpreted as primary occlusal pathology if connective tissue–mediated variability is not recognized. [8,21,22]

This presents a practical approach focused on pathophysiology, diagnostic strategy, and conservative management. Splint therapy is integrated as a vital aspect of a staged plan, with emphasis on reversibility, monitoring, and risk management.

2. Methods

This review integrates consensus statements and widely cited peer-reviewed literature addressing EDS classification

* Corresponding author:
drchrisbrown@etczone.com (Christopher Brown DDS, MPS)
Received: Dec. 19, 2025; Accepted: Jan. 6, 2026; Published: Jan. 9, 2026
Published online at http://journal.sapub.org/cmd

and clinical features, [12] TMD diagnostic criteria, [6] contemporary conceptual models of TMD, [7,10] and conservative interventions including rehabilitation and occlusal appliance therapy. [18,20] The objective is suggesting a conservative sequencing pathway for dental clinicians managing patients with hypermobility or diagnosed EDS.

3. Pathophysiology of TMJ Dysfunction in EDS

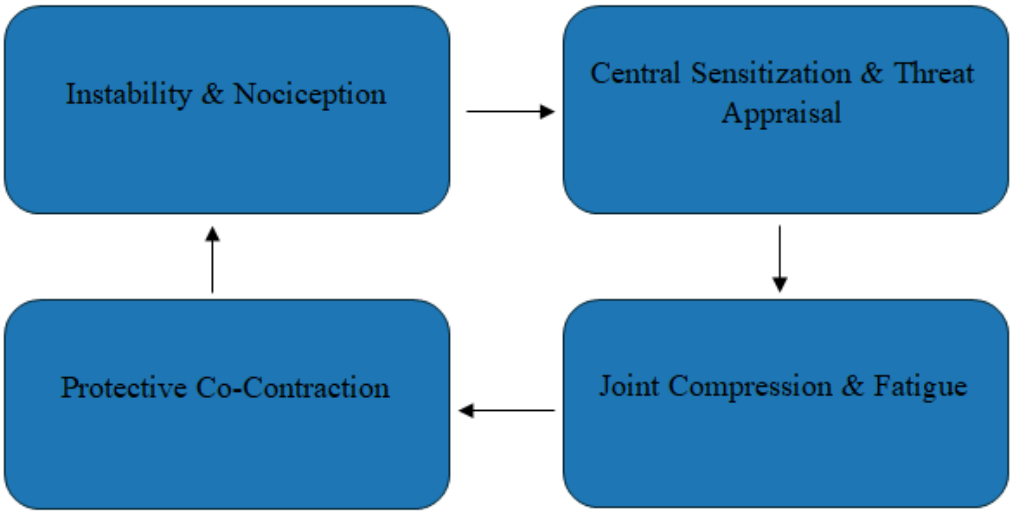

Figure 1. Neuromuscular Loop Sustaining Hypermobile TMD

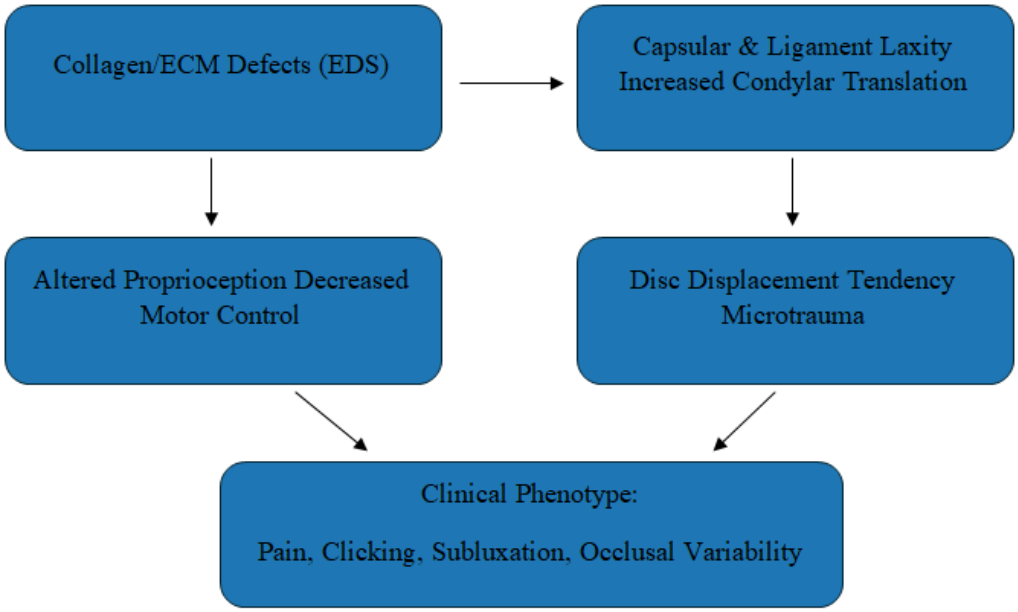

Figure 2. Drivers of TMJ Instability in EDS

EDS-associated TMJ dysfunction arises from the convergence of structural instability and altered sensorimotor control. Abnormalities of collagen and extracellular matrix reduce capsular stiffness and ligamentous restraint, permitting excessive translation of the condyle and increasing susceptibility to disc displacement and recurrent subluxation. [12,16] Instability is often most apparent during end-range opening—yawning, prolonged dental procedures, intubation, trauma, or following nocturnal parafunctional loading.

Sensorimotor control is frequently altered in hypermobility. Mechanoreceptor signaling from joint and muscle tissues contributes to proprioception and reflexive coordination; impaired afferent feedback can degrade fine motor control and promote protective co-contraction intended to stabilize the joint. [3,14,16] Over time, protective co-contraction may become persistent, elevating tonic activity in the masseter and temporalis, increasing joint compression, and provoking myofascial trigger points, headaches, and cervical muscle

involvement. [13,17]

Pain processing adds a second layer. Many patients report multisite pain and fatigue. Central sensitization can amplify symptom severity beyond what imaging suggests. [3] Psychosocial factors such as threat appraisal and fear-avoidance may further increase motor guarding and reduce adaptive movement variability. [24,25] Sleep physiology may also be relevant. Micro-arousals are often associated with increased jaw motor events, and consensus definitions of bruxism emphasize state dependence rather than a simple causal link to pain. [11,12] Together, these mechanisms support an instability–pain–motor control model that directly affects conservative management.

4. Clinical Presentation

Presentations vary by EDS subtype and comorbidity profile. Common TMJ features include clicking or popping, intermittent locking, pain with chewing, and a sense that the joint "slips" with wide opening. [6,9,17] Pain may be localized to the preauricular area or experienced predominantly as diffuse myofascial pain.

Variability in intensity and areas of symptoms is not uncommon. Symptoms and occlusal contacts may change daily with patients may report a bite that feels different after sleep or prolonged function. This variability increases the risk of iatrogenic harm from irreversible occlusal adjustments based on single time-point observations. [8,21,22] Headaches and otologic complaints may coexist even when primary pathology is muscular or instability-driven. [9]

In addition to TMD, generalized muscular fatigue, widespread pain, anxiety, and sleep disruption are common and influence tolerance for dental procedures. Recognizing these features supports shorter appointments, ergonomic positioning, and clear post-procedure guidance. [3,23,25]

5. Differential Diagnosis and Diagnostic Considerations

The differential diagnosis for TMD and peri-auricular pain includes odontogenic disease, cracked tooth syndrome, periodontal pathology, sinus disease, neuralgias, salivary gland disorders, and primary headache syndromes. Utilizing a progressive approach to TMD provides a standardized approach to differentiate myalgia, arthralgia, and disc displacement patterns. [6,9,13]

Screening for hypermobility using history prompts and brief tools can identify patients who may warrant formal evaluation for EDS. [25] Clinical examination should assess range of motion, deviation on opening, end-feel, joint sounds, and reproducibility of symptoms with loading and palpation. Direct muscle palpation assists in distinguishing myofascial pain from primary joint pain. [9,13]

Imaging is important to assist with a differential diagnosis. An MRI can assess disc position, effusion, and other soft tissues within the joint while panoramic and CBCT characterizes osseous remodeling. In hypermobile patients, imaging must be interpreted in the context of neuromuscular and central mechanisms, as symptom intensity often does not correlate with structural findings. [7,24]

6. Management Strategies

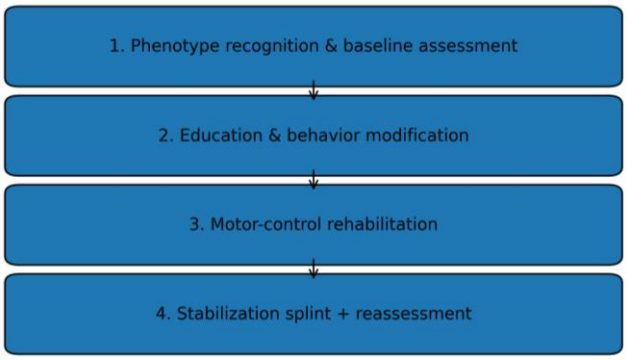

Figure 3. Conservative treatment algorithm for EDS-associated TMD

Initial management should be conservative, staged, and reversible, with objective reassessment. [9,10,17] Education focuses on avoiding end-range loading, reducing parafunctional activity, and dietary adaptation during flares. Daytime habit reversal strategies reduce awake clenching. [10,11]

Physical therapy prioritizes motor control and graded exposure rather than aggressive stretching. Improving cervical and scapular support and training controlled jaw movement can reduce guarding. [14,15,17]

Sleep and stress optimization is biomechanically relevant, as arousal and insomnia increase jaw motor activity and lower pain thresholds. [12,23,25]

7. Splint Therapy in EDS-Associated TMD

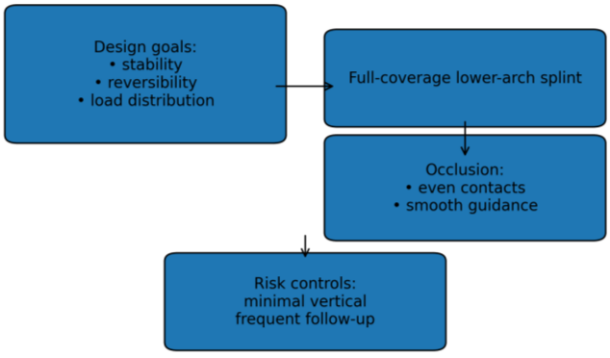

Figure 4. Lower-arch stabilization splint design principles

Stabilization splint therapy is often central to conservative management but must be adapted to the hypermobile phenotype. Evidence supports symptom reduction for subsets of patients, though protocols vary. [18,19,20] In EDS, the objective

is neuromuscular unloading and stability—not permanent mandibular repositioning.

Lower-arch stabilization orthotics are frequently advantageous due to tolerance and reduced risk of dental adaptation compared with prolonged maxillary orthotics. Regardless of arch, full coverage is often indicated to minimize tooth movement. Mandibular repositioning orthotics should be used cautiously as chronic repositioning can provoke occlusal changes and symptom variability. [8,21,22]

Occlusal scheme: Even, simultaneous contacts with smooth excursive guidance and minimal vertical increase are critical. Excessive vertical opening can strain joint tissues and increase guarding. Some designs incorporate features to discourage end-range opening in markedly unstable patients.

Wear and monitoring: Nighttime wear is typical when morning symptoms or sleep-related jaw motor activity are suspected. [11,12] Follow-up must be more frequent than routine appliance therapy: early adjustment at 2–4 weeks and reassessment at 6–8 weeks, with ongoing monitoring. At each visit, verify orthotic contacts and compare baseline and current occlusal contacts of the orthotic. If adaptation is suspected, reduce wear time, rebalance, or remake the orthotic as needed. [21,22]

Depending upon the patient's individual needs wearing an orthotic 24/7 for a limited time may be appropriate. The wearing time is based upon the patient's history, current symptoms, imaging indications, and response to treatment.

Integration: Orthotics are often more efficacious when combined with education and rehabilitation. As stability improves, tapering toward intermittent wear is often feasible.

Table 1. Clinical Use and Monitoring of Occlusal Orthotics in Patients with Ehlers-Danlos Syndrome

Indications-TMJ pain, joint instability, bruxism, occlusal variability-Ligamentous laxity and impaired proprioception increase joint loading
Splint type-Flat-plane stabilization splint (hard acrylic) Provides neuromuscular unloading without joint repositioning
Arch selection-Preferably mandibular; maxillary if retention or tolerance limits is an issue- mandibular splints often provide greater comfort and stability
Occlusal design- Bilateral contacts with shallow guidance-Reduces focal loading and risk of subluxation
Designs to avoid- Repositioning, anterior-only, or directive splints-May exacerbate instability and occlusal drift
Wear schedule- Primarily nocturnal; limited daytime use if symptomatic- Minimizes parafunctional forces while avoiding dependency
Adjustment interval- Every 4-6 weeks initially-Occlusal contacts may change due to ligamentous laxity
Monitoring parameters- Pain, joint sounds, range of motion, occlusal stability-Enables early identification of adverse changes
Duration of therapy-Symptom-guided with regular reassessment- Prolonged splint use may alter occlusion
Adjunctive therapies- Physical therapy, behavioral modification, stress management-Addresses neuromuscular and central sensitization components
Patient counseling - Emphasize symptom control rather than structural correction-Aligns expectations and improves adherence

8. Discussion

EDS challenges purely structural explanations of TMD. Capsular laxity, altered proprioception, central sensitization, and sleep-related motor activity interact to produce a variable phenotype in which irreversible occlusal interventions can be harmful. [3,7,10,25] Structured conservative care with monitoring aligns with contemporary standards and minimizes iatrogenic risk. [18,21]

Objective Outcome Measurements

Success of treatment can be best measured by the following:

1. Mandibular ROM (range of motion)
2. Report of findings of soft tissue palpation
3. Review of patient history
4. Review of medication consumption related to pain
5. Occlusal wear patterns on the orthotic

Patients are often seen on a 4-6 week period at the beginning of active treatment.

Interdisciplinary coordination is often beneficial given cervical involvement, migraine, dysautonomia, and widespread pain. Documenting objective outcomes and titrating reversible interventions provides a pragmatic pathway for complex patients.

Limitations

This review has several limitations. First, much of the available literature on dental and temporomandibular manifestations of Ehlers–Danlos syndrome consists of case reports, small observational studies, and expert opinion, which limits the strength and generalizability of conclusions. Second, EDS represents a heterogeneous group of disorders with varying genetic mechanisms and clinical severity; many studies do not clearly distinguish among subtypes, making it difficult to attribute findings to specific forms of the condition. Third, standardized diagnostic criteria for temporomandibular disorders and dental instability are inconsistently applied across studies, introducing variability in reported prevalence and treatment outcomes. Finally, there is a relative paucity of prospective, controlled trials evaluating conservative dental interventions in EDS populations, underscoring the need for higher-quality, subtype-specific research to inform evidence-based clinical guidelines.

9. Conclusions

EDS-associated TMD is best approached as an instability–pain–motor control syndrome. A conservative, staged plan incorporating stabilization orthotic therapy, rehabilitation, and sleep/stress optimization can reduce symptoms while preserving long-term function. [16,18,21]

REFERENCES

[1] Malfait F, Francomano C, Byers P, et al. The 2017 international classification of the Ehlers–Danlos syndromes. Am J Med Genet C Semin Med Genet. 2017; 175(1): 8-26.

[2] Tinkle B, Castori M, Berglund B, et al. Hypermobile Ehlers–Danlos syndrome (hEDS): clinical description and natural history. Am J Med Genet C Semin Med Genet. 2017; 175(1): 48-69.

[3] Castori M. Pain in Ehlers–Danlos syndromes: mechanisms, evaluation, and treatment. Am J Med Genet C Semin Med Genet. 2015; 169(1): 84-96.

[4] Grahame R. Hypermobility: an important but often neglected area within rheumatology. Nat Clin Pract Rheumatol. 2008; 4(10): 522-524.

[5] Hakim AJ, Grahame R. A simple questionnaire to detect hypermobility. Int J Clin Pract. 2003; 57(3): 163-166.

[6] Schiffman E, Ohrbach R, Truelove E, et al. Diagnostic Criteria for Temporomandibular Disorders (DC/TMD). J Oral Facial Pain Headache. 2014; 28(1): 6-27.

[7] List T, Jensen RH. Temporomandibular disorders: old ideas and new concepts. Cephalalgia. 2017; 37(7): 692-704.

[8] Okeson JP. Management of Temporomandibular Disorders and Occlusion. 8th ed. Elsevier; 2020.

[9] De Leeuw R, Klasser GD, eds. Orofacial Pain. 6th ed. Quintessence; 2018.

[10] Greene CS. Managing patients with temporomandibular disorders. J Am Dent Assoc. 2010; 141(9): 1086-1088.

[11] Lobbezoo F, Ahlberg J, Glaros AG, et al. Bruxism defined and graded. J Oral Rehabil. 2013; 40(1): 2-4.

[12] Kato T, Thie NMR, Huynh N, et al. Sleep bruxism and micro-arousals. Sleep Med Rev. 2003; 7(5): 395-414.

[13] Benoliel R, Svensson P, Heir GM, et al. Persistent orofacial muscle pain. Oral Dis. 2011; 17(Suppl 1): 83-94.

[14] Kraus S. Physical therapy management of TMD. J Bodyw Mov Ther. 2014; 18(3): 441-452.

[15] La Touche R, Fernández-de-las-Peñas C, et al. Manual therapy and exercise for TMD. Clin J Pain. 2009; 25(8): 705-712.

[16] Guarda-Nardini L, Manfredini D, Ferronato G. TMJ disorders and joint hypermobility. J Oral Rehabil. 2012; 39(10): 737-746.

[17] Wright EF, North SL. Management of TMD: a clinical perspective. J Man Manip Ther. 2009; 17(4): 247-254.

[18] Greene CS, Laskin DM. Splint therapy for TMD. J Am Dent Assoc. 2013; 144(6): e1-e9.

[19] Alajbeg I, Gikić M, Valentić-Peruzović M. Occlusal splints in TMD. Acta Clin Croat. 2015; 54(3): 331-338.

[20] Türp JC, Jokstad A, Motschall E, et al. Stabilization splints for TMD. J Oral Rehabil. 2004; 31(8): 777-786.

[21] Moghadam BK, Hartsfield JK. Adverse effects of occlusal appliances. Dent Clin North Am. 2007; 51(1): 225-240.

[22] Ishigaki S, Kurozumi T, et al. Occlusal changes during splint therapy. J Oral Rehabil. 2016; 43(5): 379-387.

[23] Baeza-Velasco C, Bulbena A, et al. Joint hypermobility and anxiety. Curr Psychiatry Rep. 2018; 20(4): 34.

[24] Sullivan MJL, Thorn B, et al. Fear and pain perspectives. Pain. 2001; 100(3): 289-299.

[25] Stone J, Waddell G. Biopsychosocial model of pain. J Psychosom Res. 2002; 53(1): 1-8.