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NARRATIVE REVIEW

Endometriosis and TMD as Phenotypes of the Same Systemic Disease: A New Paradigm for the Integrated Care of Pain in Women

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Abstract

Background: Endometriosis and temporomandibular disorders (TMD) represent debilitating chronic conditions affecting millions of women worldwide, imposing intense pain and a profound compromise on quality of life. Emerging evidence reveals a robust and non-fortuitous systemic connection between both pathologies. **Methods**: This integrative review critically analyses the literature published between 2000 and 2025 from the PubMed, Scopus, Web of Science, SciELO, and LILACS databases. The analysis focuses on the shared pathophysiological mechanisms hormonal, inflammatory, and neural that unify these conditions.

Results: The prevalence of TMD in women with endometriosis reaches levels of 45.2% to 77.3%, a value drastically higher than the 5-12% rate observed in the general population. This comorbidity is orchestrated by a unifying pathophysiological triad: hormonal dysfunction, systemic inflammation, and central sensitization, which act synergistically to amplify both pelvic and orofacial pain.

Conclusion: We propose a new paradigm of clinical care, integrated and multidisciplinary, based on bidirectional screening and multimodal therapies. Such an approach aims to catalyze early diagnosis and optimize the management of chronic pain in women, transforming the clinical trajectory of these patients.

Keywords

Endometriosis, Temporomandibular disorders, Comorbidity, Central sensitization, Chronic pain

Abbreviations

TMD: Temporomandibular Disorders; TMJ: Temporomandibular Joint; COPC: Chronic Overlapping Pain Condition; DC/TMD: Diagnostic Criteria for Temporomandibular Disorders; CSI: Central Sensitization Inventory; ERα/ERβ: Estrogen Receptors Alpha and Beta; TNF-α: Tumor Necrosis Factor-Alpha; IL-1β, IL-6, IL-8: Interleukins 1 Beta, 6, and 8; LPS: Lipopolysaccharides; GnRH: Gonadotropin-Releasing Hormone; PNE: Pain Neuroscience Education; CBT: Cognitive-Behavioral Therapy Introduction

This report addresses the paradigmatic clinical challenge represented by the coexistence of endometriosis and temporomandibular disorders (TMD). Far from being isolated pathologies confined to their respective specialties, these conditions emerge as interconnected phenotypic expressions of an underlying systemic process, marked by chronic inflammation, hormonal dysregulation, and sensitization of the central nervous system. Our central hypothesis posits that the

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high comorbidity observed is not a casual event, but rather the manifestation of a shared pathophysiological nexus, demanding a paradigmatic transformation in care: From a fragmented model to a holistic and integrated clinical approach. This introduction outlines the architecture of this work, guiding the reader from the robust epidemiological evidence, through the molecular and neural mechanisms that substantiate this connection, to the proposal of a new framework for clinical diagnosis and management.

Methods

This narrative review was conducted using an integrative approach, aiming to identify, synthesize, and critically analyze the scientific literature concerning the correlation between endometriosis and TMD. The investigation focused on shared pathophysiological mechanisms, their clinical implications, and emerging therapeutic strategies. The systematic search for articles covered the period from January 2000 to June 2025, in the electronic databases PubMed, Scopus, Web of Science, SciELO, and LILACS. Descriptors in English and Portuguese were used, including “endometriosis”, “temporomandibular disorders”, “comorbidity”, “central sensitization”, and “chronic pain”, and their correspondents. After removing duplicates, 632 records were screened by title and abstract. From this total, 110 articles were selected for full-text evaluation, resulting in the inclusion of 66 studies for the final synthesis, as detailed in the PRISMA flowchart (Figure 1).

Results

Rethinking the Clinical Landscape of Endometriosis and TMD

Endometriosis: Beyond the pelvis, a systemic inflammatory disease

Endometriosis, classically defined by the presence of endometrium-like tissue outside the uterine cavity [1], is now understood as a chronic, systemic, and estrogen-dependent inflammatory condition [2]. Its prevalence reaches about 10% of women of reproductive age [3], representing approximately 190 million individuals globally [4]. The clinical picture is dominated by

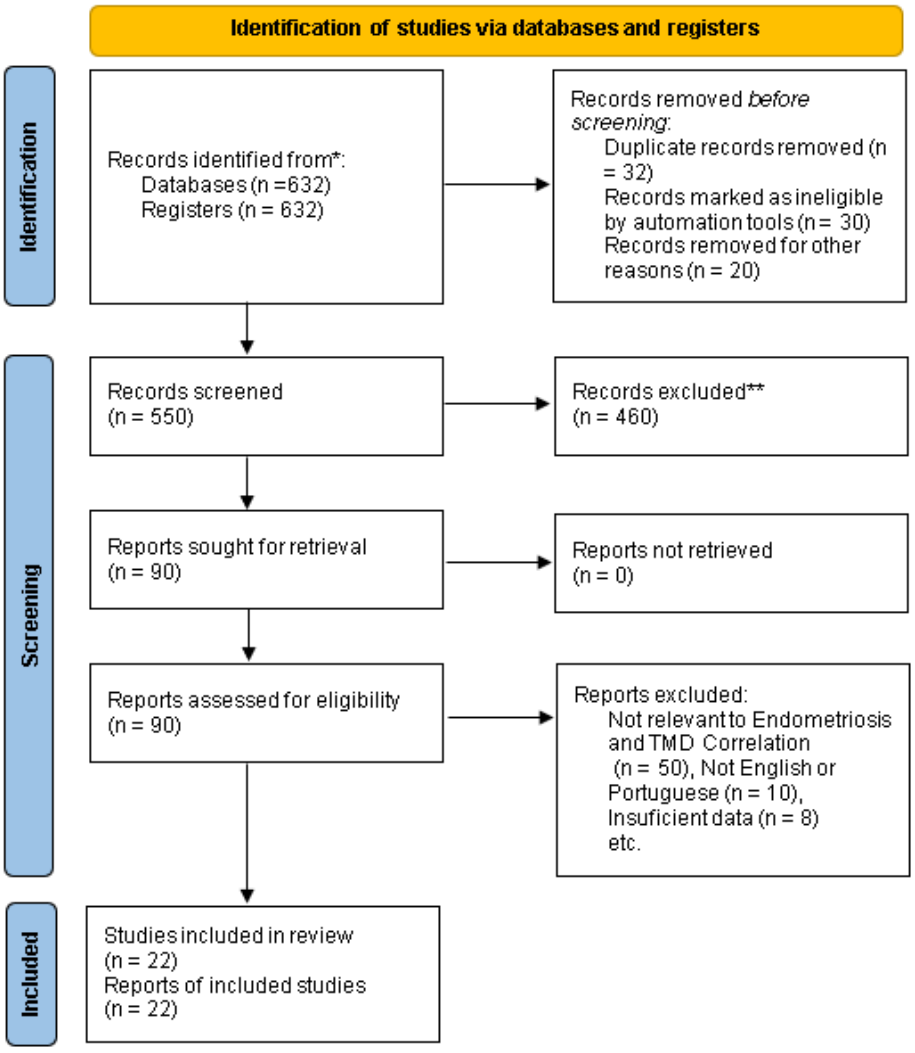

Figure 1: PRISMA 2020 flowchart detailing the literature selection process for the integrative review on the correlation between endometriosis and temporomandibular disorders (TMD).

debilitating symptoms such as chronic pelvic pain [5], severe dysmenorrhea [6], dyspareunia, and infertility (present in 30-50% of patients) [7], which profoundly impacts quality of life, mental health, and interpersonal relationships [8-10]. The socioeconomic impact of the disease is massive, comparable to that of conditions like type 2 diabetes, due to high healthcare costs and significant productivity loss [8]. Recent evidence challenges the view of endometriosis as a localized pelvic pathology, recognizing it as a systemic disease associated with various autoimmune conditions [11] and other chronic pain syndromes [12], thus framing it within the concept of a Chronic Overlapping Pain Condition (COPC).

TMD: A complex Orofacial pain syndrome

Temporomandibular disorders (TMD) constitute a heterogeneous group of musculoskeletal and neuromuscular conditions affecting the temporomandibular joint (TMJ), masticatory muscles, and associated structures [13]. The gold-standard diagnosis is made using the Diagnostic Criteria for TMD (DC/TMD), which employs a biaxial approach to assess both physical (Axis I) and psychosocial (Axis II) factors [14]. The prevalence in the general population ranges from 5% to 12% [15], with a notable predominance in females, reaching a ratio of up to 9:1 in clinical settings. Factors such as pain intensity, functional impairment, and catastrophizing [16] are determinants for seeking treatment, amplified by mechanisms of systemic inflammation and central sensitization [17,18]. The most common symptoms include orofacial pain, headache, limited mandibular movements, and tinnitus [19,20].

The Overlap: Quantifying the comorbidity and its functional burden

Recent studies reveal that between 45.2% and 77.3% of women diagnosed with endometriosis present with TMD symptoms [21,22], a prevalence dramatically higher than that of the general population (5-12%), suggesting a robust and non-random clinical association. This comorbidity not only adds but multiplies the burden on the patient, intensifying the psychological impact (higher rates of anxiety and depression) and socioeconomic consequences (increased use of health services and absenteeism). Electromyographic data elucidate significant functional deficits in women with TMD [21], indicating a protective neuromuscular inhibition in response to pain. The endometriosis-TMD nexus perfectly exemplifies a COPC, where shared pathophysiological mechanisms create a mutual predisposition and amplify the pain experience (Table 1).

The Pathophysiological Triad: Unifying Mechanisms

Hormonal Axis: Estrogen dominance and progesterone resistance

Estrogen acts as a potent pain amplifier by modulating the expression of ERα and ERβ receptors in the trigeminal ganglion and the TMJ [23]. Concurrently, progesterone resistance, a central feature of endometriosis [24], exacerbates the inflammatory response, perpetuating a vicious cycle of pain that manifests systemically [25].

Inflammatory cascade: Systemic effects and the Gut-Brain Axis

Pro-inflammatory cytokines (TNF-α, IL-1β, IL-6, IL-8), overexpressed in endometriosis [29], generate a state of low-grade systemic inflammation. This process is potentiated by gut dysbiosis and increased permeability of the intestinal barrier ("leaky gut") [30], which allow the translocation of inflammatory mediators, sensitizing peripheral nociceptors throughout the body, including those in the orofacial region.

Neural Amplifier: Central Sensitization and Nociplastic Pain

Chronic inflammation and persistent pain act as triggers for central sensitization, a process of maladaptive neuroplasticity that amplifies pain signals in the central nervous system. The result is nociplastic pain, characterized by hyperalgesia (exaggerated response to painful stimuli) and allodynia (pain in response to non-painful stimuli) [31,32]. Non-musculoskeletal symptoms like tinnitus [19] reinforce the presence of this mechanism, which functionally connects pelvic pain to orofacial pain.

A New Clinical Paradigm: An Integrated Approach

Bidirectional screening

It is imperative that gynecologists actively screen TMD in patients with endometriosis, and that dentists

Table 1: Comparative profile of endometriosis and TMD.

Feature	Endometriosis	Temporomandibular Disorder (TMD)
Definition	Chronic systemic inflammatory disease, estrogen dependent.	Musculoskeletal and neuromuscular disorders of the orofacial region.
Global Prevalence	About 10% of women of reproductive age.	It affects 5% to 12% of the general population.
Main Symptoms	Chronic pelvic pain, dysmenorrhea, dyspareunia, infertility.	Orofacial pain, headache, mandibular limitation, joint noises.
Standard Diagnosis	Laparoscopy with histological confirmation (history); Accepted imaging tests.	Diagnostic Criteria for TMD (CD/TMD).
ASSOCIATED CHARACTERISTICS	Systemic inflammation, hormone dysregulation, comorbid painful conditions (COPCs).	Central sensitization, psychosocial distress, presence of tinnitus.

Source: Authors

Table 2: Structure for integrated multidisciplinary management.

Clinical Objective	Primary Specialty	Specific Interventions	Primary Mechanism Targeted
Reduce pelvic nociceptive input and hormonal factors	Gynecology	Hormone suppression (progestins, GnRH agonists), surgical excision	hormonal, inflammatory
Reduce orofacial nociceptive intake and dysfunction	Dentistry / Orofacial Pain	Manual therapy, mandibular exercises, cautious use of reversible occlusal splints	Peripheral Neuromuscular
Modulate systemic inflammation	Nutrition, Physiotherapy	Anti-inflammatory diet (omega-3, antioxidants), targeted pelvic/cervical physical therapy	Neuromuscular, Peripheral, Inflammatory
Down regulation of central sensitization	Psychology / Pain Medicine / Physiotherapy	Pain Neuroscience Education (NEP), Cognitive Behavioral Therapy (CBT), graded exercises, centrally acting medications	Neural

Source: Authors

investigate the possibility of endometriosis in patients with chronic TMD. The application of tools like the DC/TMD [14] and the Central Sensitization Inventory (CSI) [33] is fundamental for a comprehensive diagnosis.

Multimodal Therapies

Effective management requires a synergistic and multimodal approach:

- **Gynecology/Dentistry**: Hormonal suppression therapies, surgical interventions, and the judicious use of occlusal devices [34,35].
- **Physiotherapy/Nutrition**: Specialized physiotherapy (pelvic and cervicocranial) and nutritional strategies with an anti-inflammatory focus (diet rich in omega-3 and antioxidants) [36-38].
- **Psychology/Pain Medicine**: Pain Neuroscience Education (PNE), Cognitive-Behavioral Therapy (CBT), and the use of neuromodulators to regulate central sensitization [35](Table 2).

Charting the Course for Future Research

Clinical studies

Conducting prospective longitudinal studies and randomized clinical trials is crucial to elucidate causality and validate the efficacy of integrated therapeutic approaches, with a focus on patient-centered outcomes.

Precision Medicine

The identification of biomarkers (genetic, inflammatory, neurobiological) [32] to stratify patients and the development of non-hormonal therapies targeting neuroinflammation [17] represent the next frontiers of research.

Discussion

This integrative review provides a solid foundation for a new clinical paradigm that recognizes the high comorbidity between endometriosis and TMD not as a coincidence, but as a manifestation of a shared pathophysiological process. The findings synthesized here validate the central hypothesis that both conditions are phenotypic expressions of the same systemic disease, marked by hormonal dysfunction, chronic inflammation, and central nervous system sensitization.

The reclassification of endometriosis as a systemic disease beyond its pelvic manifestations [1,2] is the starting point for this discussion. The disease's prevalence in approximately 10% of women of reproductive age [3,4] and its profound impact on quality of life [9,10] and the economy [8] establish it as a global public health issue. The literature already points to endometriosis's association with other inflammatory and autoimmune conditions [11], and our analysis frames it within the concept of a Chronic Overlapping Pain Condition (COPC) [12], a framework that allows for a better understanding of coexisting pain syndromes in different body regions.

The connection between pelvic pain and orofacial pain is mediated by systemic mechanisms, not isolated events. While the prevalence of TMD in the general population is 5-12% [15], this number dramatically increases to 45.2-77.3% in women with endometriosis [21,22]. This statistically significant difference is not coincidental. TMD, which affects the temporomandibular joint and masticatory muscles [13], is itself an example of a COPC, influenced by psychosocial and sensitization factors [16,17,18]. Its symptoms, such as orofacial pain and tinnitus [19,20], reinforce the systemic and interconnected nature of the problem.

The key to this interconnection lies in the pathophysiological triad that acts as a link between the two conditions. Hormonal dysfunction, marked by estrogen dominance and progesterone resistance [23-28], not only perpetuates inflammation in endometriosis but also sensitizes nociceptors in the orofacial region, amplifying pain perception in both areas. Simultaneously, low-grade chronic inflammation, generated by the overexpression of pro-inflammatory cytokines [29] and potentiated by gut-brain axis dysbiosis [30], creates a systemic environment that promotes the sensitization of neural pathways. Finally, central sensitization, a process of maladaptive neuroplasticity [31], acts as a neural amplifier, translating chronic inflammation and persistent pain into nociplastic pain [32], where non-painful stimuli are interpreted as painful.

The proposal for an integrated, multidisciplinary care model is the logical consequence of this understanding [34,35]. The current fragmented approach fails to recognize the shared etiology and, as a result, does not optimize chronic pain management in women. Implementing bidirectional screening between gynecologists and dentists, using tools like the DC/TMD [14] and the Central Sensitization Inventory (CSI) [33], can lead to earlier diagnoses and more effective management. The proposed multimodal therapy, which combines pharmacological interventions (hormonal suppression and neuromodulators), physical therapies (physiotherapy), and behavioral strategies (PNE and CBT), represents a promising path for treatment [35-38].

Although the correlation is strong, causality needs to be further investigated through prospective studies and randomized clinical trials [32]. The identification of biomarkers [17] and the search for personalized therapies are the next frontiers for precision medicine, which may finally offer more effective and compassionate treatment for millions of women suffering from these conditions. Ultimately, the paradigm shift proposed in this work is not just a matter of optimizing clinical practice, but of redefining the understanding of chronic pain in women.

Study Limitations

This review, while providing a robust conceptual framework, is subject to several limitations that warrant consideration and point to avenues for future research.

Methodological limitations

- **Heterogeneity of Included Studies:** This review synthesized findings from studies with diverse methodological designs (cross-sectional, cohort, case-control), varying populations, and inconsistent diagnostic criteria for both endometriosis and TMD. This heterogeneity limited our ability to perform a quantitative meta-analysis and may impact on the generalizability of our findings.
- **Selection Bias:** Our search was confined to specific databases and to articles published in English and Portuguese. This may have excluded relevant studies published in other languages or in regional databases. Furthermore, the possibility of publication bias, where studies with negative or null results are less likely to be published, might have artificially inflated the observed association.
- **Absence of Meta-analysis:** Due to the methodological heterogeneity and the variability in reported outcomes, a quantitative meta-analysis was not feasible. This limits the precision of our estimates regarding the association between the two conditions.

Limitations of primary studies

- **Observational Designs:** Most included studies were cross-sectional, which precludes the establishment of a causal relationship between endometriosis and TMD. The direction of the association remains uncertain.
- **Inconsistent Diagnostic Criteria:** We observed variability in the diagnostic criteria used for endometriosis (laparoscopy vs. ultrasound vs. MRI) and TMD (DC/TMD vs. various clinical criteria). This inconsistency can influence the reported prevalence rates and makes direct comparisons challenging.
- **Underreporting and Delayed Diagnosis:** Both endometriosis and TMD are known for delayed diagnosis and underreporting, particularly in healthcare systems with limited access to specialists. This likely results in an underestimation of the true comorbidity rates.

Conceptual limitations

- **Pathophysiological Mechanisms:** While we propose a unifying pathophysiological triad, the precise mechanisms linking endometriosis and TMD are still not fully understood. The current evidence is largely based on inferences from separate studies on each condition.
- **Confounding Factors:** Variables such as psychological stress, medication use, other comorbidities (e.g., fibromyalgia, irritable bowel syndrome), socioeconomic status, and lifestyle factors can act as confounders in the observed association. These were not adequately controlled in many of the primary studies.

Clinical limitations

- **Validation of the Integrated Model:** The proposed multidisciplinary care model, while theoretically sound, lacks validation through randomized clinical trials that could demonstrate its efficacy and cost-effectiveness compared to traditional fragmented care.
- **Practical Implementation:** We did not adequately address the barriers to implementing an integrated model, such as the availability of specialists, costs, organizational resistance, and professional training.
- **Cultural Generalization:** Most included studies were conducted in high-income countries, limiting the generalizability of the findings to populations with different socioeconomic contexts and healthcare systems.

Directions for future research

To overcome these limitations, we recommend the following:

- **Prospective longitudinal studies** to establish temporal and causal relationships.
- **Randomized clinical trials** to test the efficacy of multidisciplinary interventions.
- **Biomarker validation studies** enable patient stratification and precision medicine approaches.
- **Multicenter research** that includes diverse populations to improve generalizability.
- **Development of standardized instruments** for bidirectional screening.
- **Pharmacoeconomic analyses** of the integrated care model to assess its value.

Despite these limitations, our work provides a solid foundation for understanding the association between endometriosis and TMD, justifying the need for a more integrated clinical approach and more robust future studies.

Conclusion

The pathological synergy between endometriosis and TMD, evidenced by a robust epidemiological correlation and grounded in a shared pathophysiological triad, calls for a revolution in women's healthcare, centered on interdisciplinarity. The adoption of this integrated model not only promises earlier diagnoses and more effective treatments but also represents a fundamental step towards a more effective and compassionate therapeutic horizon for millions of women living with chronic pain.

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Conflicts of Interest

The authors declare no conflicts of interest.

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None declared.

Author Contributions

Metello Neves, L.B.: Conceptualized the study, conducted the literature research, and was the primary author in the writing of the manuscript. **Baldessarini, B.L.** and **Mello, R.C.M.:** Contributed to the critical review and editing of the manuscript. **Lima, B.C.:** Acted as advisor, supervised the project, and contributed to the critical review of the manuscript.

All authors have read and approved the final submitted version.

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