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VITAMIN D IN ENDOMETRIOSIS: MECHANISMS AND CLINICAL POTENTIAL – A LITERATURE REVIEW

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ABSTRACT

Objectives: Endometriosis is a chronic inflammatory condition affecting approximately 10% of women of reproductive age. Recent studies have highlighted vitamin D as a potential immunomodulator influencing disease progression. This review aims to synthesize current evidence regarding the mechanistic links between vitamin D and endometriosis, the correlation of serum 25(OH)D levels with disease severity, and the clinical efficacy of supplementation.
Methods: A systematic literature search was conducted using PubMed and the Cochrane Library. The analysis included randomized controlled trials (RCTs), observational studies, and molecular research published within the last eight years. A qualitative narrative synthesis was performed to categorize findings into mechanistic, observational, and interventional themes.
Key Findings: Molecular studies demonstrate that vitamin D inhibits endometriotic cell proliferation, inflammation, and angiogenesis by targeting the NF-κB and VEGF pathways. Observational data generally indicate lower serum 25(OH)D levels in women with advanced endometriosis compared to healthy controls. However, clinical supplementation trials yield heterogeneous results; while some RCTs report significant reductions in pelvic pain and inflammatory markers (hs-CRP), others find no statistical superiority over placebo, potentially due to variations in dosage and baseline vitamin D status.
Conclusions: Vitamin D serves as a biologically plausible adjunctive therapy for endometriosis. While not a definitive cure, its safety and anti-inflammatory properties justify its use in managing deficiency. Future research should prioritize personalized supplementation protocols considering genetic polymorphisms of the vitamin D receptor.

KEYWORDS

Endometriosis, Vitamin D, Vitamin D Receptor, Pelvic Pain, Immunomodulation, Inflammation

CITATION

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1. Introduction

1.1. Endometriosis

Endometriosis is a prevalent, estrogen-dependent chronic gynecological disorder characterized by the presence of functional endometrial-like tissue outside the uterine cavity, primarily within the pelvic peritoneum and ovaries. It affects approximately 10% of reproductive-age women, manifesting through symptoms such as chronic pelvic pain, severe dysmenorrhea, dyspareunia and infertility[1]. The pathogenesis is multifactorial, with the most widely accepted "Sampson's Theory" of retrograde menstruation being complemented by theories of immune dysfunction, where a failure in peritoneal immunosurveillance allows ectopic cells to survive, adhere, and proliferate. Current management relies on surgical excision of lesions and hormonal therapies (e.g. GnRH agonists, oral contraceptives) aimed at suppressing ovarian function. However, these treatments are often associated with significant side effects and high recurrence rates, necessitating the search for non-hormonal adjunctive therapies[2,3,4].

1.2. Vitamin D

Vitamin D, a fat-soluble secosteroid, primarily synthesized in the skin via UVB radiation (90%) and supplemented by dietary intake (10%), it undergoes two-step hydroxylation in the liver and kidneys to become its active form, 1,25(OH)2D[5]. While its classical role is the regulation of calcium-phosphorus homeostasis and bone mineralization, vitamin D is now recognized as a potent immunomodulator. It acts through the Vitamin D Receptor (VDR), which is expressed in immune cells and reproductive tissues, including the endometrium[6]. In the context of endometriosis, vitamin D exerts non-skeletal effects by inhibiting the production of pro-inflammatory cytokines (e.g., IL-1, IL-6, TNF-α), reducing angiogenesis, and promoting apoptosis in ectopic cells. Consequently, low serum levels of 25-hydroxyvitamin D have been hypothesized to correlate with disease risk and severity, suggesting that vitamin D status may significantly influence the inflammatory landscape of endometriosis[7].

2. Methodology

This study was conducted as a narrative review to synthesize current clinical and experimental evidence regarding the role of vitamin D in the pathophysiology and management of endometriosis. The analysis focused on the correlation between serum vitamin D levels and disease severity, as well as the efficacy of vitamin D supplementation in alleviating endometriosis-associated symptoms. A comprehensive literature search was performed using the PubMed and Cochrane Library databases. The search was limited to articles published between 2018 and 2026 to ensure the inclusion of the most recent scientific developments. Keywords and MeSH terms included: "endometriosis", "vitamin D", " vitamin D receptor (VDR)", " pelvic pain", " immunomodulation" and " inflammation". Studies were selected based on their relevance to the biochemical, immunological, and clinical aspects of the vitamin D-endometriosis axis. The inclusion criteria comprised:

- Interventional studies (Randomized Controlled Trials).
- Observational studies (case-control and cohort studies).
- Recent systematic reviews and meta-analyses.
- In vitro and animal models investigating molecular mechanisms.

Studies that focused exclusively on surgical techniques without considering metabolic or nutritional factors were excluded.

3. Results

3.1. Mechanisms of Action: Molecular and Experimental Evidence

Evidence from in vitro and animal studies suggests that vitamin D acts as a potent modulator of the biological pathways involved in the development and survival of endometriotic lesions. The primary mechanisms identified in recent research include:

- Anti-inflammatory and Immunomodulatory Effects: Vitamin D inhibits the activation of the NF-κB (nuclear factor kappa-light-chain-enhancer of activated B cells) pathway, which is a major driver of inflammation in endometriosis. By stabilizing the IκBα protein, vitamin D reduces the production of pro-inflammatory cytokines such as IL-1β, IL-6, IL-8, and TNF-α in both peritoneal fluid and endometriotic stromal cells.
- Inhibition of Proliferation and Migration: 1,25(OH)2D3 has been shown to significantly decrease the viability and DNA synthesis of ectopic endometrial stromal cells (ESCs). It targets the PI3K/AKT/mTOR and MAPK/ERK signaling pathways, which are responsible for the aggressive growth and invasion characteristic of endometriotic tissue.
- Regulation of Angiogenesis and Adhesion: Vitamin D reduces the expression of vascular endothelial growth factor (VEGF) and matrix metalloproteinases (MMP-2 and MMP-9). These enzymes are crucial for the formation of new blood vessels and the degradation of the extracellular matrix, which allows ectopic tissue to adhere and invade the peritoneal lining.
- Pro-apoptotic Activity: Recent studies have identified that vitamin D can trigger programmed cell death (apoptosis) in endometriotic lesions by modulating the balance between pro-apoptotic (e.g., Bax) and anti-apoptotic (e.g., Bcl-2) proteins.
- Animal Models: In mouse and rat models of induced endometriosis, the administration of vitamin D or VDR agonists (like elocalcitol) led to a significant regression of lesion volume and weight. This was often accompanied by a reduction in macrophage recruitment and fibrosis within the peritoneal cavity[8,9,10,11].

Table 1. Summary of Vitamin D mechanisms in the pathophysiology of endometriosis.

Biological Process	Effect of Vitamin D	Molecular Targets/Pathways
Inflammation	Reduction of pro-inflammatory response	↓ IL-1β, IL-6, IL-8, TNF-α; Inhibition of NF-κB
Cell Proliferation	Inhibition of ectopic endometrial growth	Suppression of PI3K/AKT/mTOR and MAPK/ERK
Angiogenesis	Decreased formation of new blood vessels	↓ VEGF (Vascular Endothelial Growth Factor)
Adhesion & Invasion	Reduced attachment and tissue remodeling	↓ MMP-2 and MMP-9 (Matrix Metalloproteinases)
Apoptosis	Induction of programmed cell death	↑ Bax (pro-apoptotic) / ↓ Bcl-2 (anti-apoptotic)

3.2. Serum Vitamin D Levels and Disease Severity

Clinical observational studies have extensively investigated the association between circulating levels of 25-hydroxyvitamin D [25(OH)D] and the prevalence or stage of endometriosis. The findings from recent meta-analyses and case-control studies can be summarized as follows:

- Evidence of Inverse Correlation: Several large-scale studies reported that women with endometriosis tend to have significantly lower serum 25(OH)D levels compared to healthy controls. Furthermore, a correlation between the degree of deficiency and disease severity has been observed, with lower vitamin D concentrations linked to advanced stages (III and IV according to the ASRM classification).
- Risk Assessment: A prospective cohort study involving over 70,000 women found that those in the highest quintile of predicted vitamin D status had a 24% lower risk of developing laparoscopically-confirmed endometriosis compared to those in the lowest quintile.
- Inconsistencies in Data: Despite the trends mentioned above, some studies have failed to find a statistically significant difference in vitamin D status between patients and controls. For instance, some researchers noted that while mean levels were lower in the endometriosis group, the overlap in confidence intervals prevented clear diagnostic differentiation.
- Phenotype Variations: Some evidence suggests that the association may be stronger for specific phenotypes, such as ovarian endometriomas, compared to superficial peritoneal lesions. However, results remains conflicting regarding whether vitamin D levels can serve as a reliable clinical biomarker for disease staging[12,13,14,15].

3.3. Supplementation Outcomes: Clinical Trials and Pain Management

The clinical efficacy of vitamin D supplementation in women with endometriosis has been evaluated primarily in the context of pain reduction and reproductive outcomes. Findings from recent randomized controlled trials (RCTs) and systematic reviews include:

- Impact on Pelvic Pain and Dysmenorrhea: Results regarding pain relief are mixed. Some studies reported a significant reduction in pelvic pain and dysmenorrhea after supplementation with high doses of vitamin D (e.g., 50,000 IU every two weeks for 12 weeks). Patients with baseline deficiency (<30 ng/mL) appeared to benefit more significantly from treatment compared to those with sufficient levels.
- Comparison with Placebo: However, other high-quality RCTs found that while vitamin D supplementation (e.g., 2,000 IU daily) led to a decrease in pain scores, the magnitude of the effect was not statistically superior to the placebo group. This suggests a strong placebo effect in pain management or that the dosage and duration used in some trials might be insufficient to produce a therapeutic response.

- Reproductive Outcomes and IVF: Data on fertility are limited. One significant study investigated the effect of a single high dose (600,000 IU) of vitamin D3 before IVF cycles in women with endometriosis. The results showed no significant improvement in clinical pregnancy rates compared to the placebo group, although the study noted that vitamin D remains essential for overall reproductive health.
- Anti-inflammatory Markers: Beyond clinical symptoms, supplementation has been shown to reduce systemic inflammatory markers, such as hs-CRP (high-sensitivity C-reactive protein) and total antioxidant capacity (TAC), which may indicate a stabilization of the chronic inflammatory state associated with the disease[16,17,18,19].

4. Discussion

The relationship between vitamin D and endometriosis represents a complex intersection of endocrinology, immunology, and clinical nutrition. While the molecular evidence strongly supports the role of vitamin D as a protective agent against the survival and proliferation of ectopic endometrial tissue, clinical findings remain heterogeneous. This discrepancy highlights several critical factors that must be considered when interpreting the data.

4.1. The Gap Between In Vitro and Clinical Findings

The most striking observation in this review is the robustness of vitamin D's effects in experimental settings compared to the variability in human trials. In laboratory models, 1,25(OH)2D consistently demonstrates anti-inflammatory and anti-proliferative properties by downregulating the NF-κB and VEGF pathways. However, the human body presents a far more complex environment. The systemic concentration of vitamin D measured in serum (25(OH)D) may not always reflect the local concentration or the metabolic activity within the endometriotic lesions themselves. Furthermore, the "biological insensitivity" of established lesions - potentially due to chronic fibrosis or altered VDR expression - might explain why vitamin D is more effective at preventing the establishment of endometriosis in animal models than at treating advanced stages in patients[20,21].

4.2. Methodological Heterogeneity in Supplementation Trials

The mixed results in supplementation outcomes can be attributed to several methodological variations:

- Dosage and Duration: Trials reviewed in this study utilized vastly different protocols, ranging from 2,000 IU daily to 50,000 IU bi-weekly. It is possible that low-dose supplementation is insufficient to overcome the high inflammatory burden of endometriosis.
- Baseline Vitamin D Status: Many trials included participants with varying baseline levels. Evidence suggests that supplementation is most effective in patients with profound deficiency (<20 ng/mL), whereas its impact on women with sufficient levels is negligible.
- Confounding Factors: Seasonal variations in sunlight exposure, BMI (as vitamin D is sequestered in adipose tissue), and dietary habits are often poorly controlled in clinical trials, which may mask the true effect of the intervention[22].

4.3. The Role of Genetic Polymorphisms

An emerging area of discussion is the role of genetic variations in the Vitamin D Receptor (VDR) and Vitamin D Binding Protein (VDBP). Certain polymorphisms (e.g., FokI, BsmI) may alter an individual's response to vitamin D, regardless of their serum levels. This suggests that "one-size-fits-all" supplementation may not be appropriate, and future therapeutic strategies might require a personalized approach based on genetic screening[23,24,25].

4.4. Clinical Implications and Safety

Despite the lack of a definitive "cure" status, vitamin D remains an attractive adjunctive therapy. It is cost-effective, has a high safety profile, and provides additional benefits for bone health and mood, which are often compromised in women with endometriosis due to hormonal treatments (e.g., GnRH agonists). The reduction in markers like hs-CRP suggests that even if vitamin D does not completely eliminate pain, it may significantly stabilize the chronic inflammatory environment, potentially slowing disease progression[26].

5. Conclusions

The evidence synthesized in this review confirms that vitamin D is a significant biological factor in the context of endometriosis, acting far beyond its traditional role in bone metabolism. Molecular and animal studies provide a strong mechanistic foundation, demonstrating that vitamin D can inhibit the key hallmarks of endometriosis: inflammation, proliferation, and angiogenesis. However, the translation of these laboratory findings into clinical practice remains incomplete.

While observational data frequently link low serum 25(OH)D levels with an increased risk and severity of the disease, clinical trials involving supplementation have produced inconsistent results. These discrepancies are likely due to variations in dosage, patient baseline status, and genetic factors that influence vitamin D metabolism.

Key Findings and Future Directions:

- Screening: Regular monitoring of vitamin D levels in women with endometriosis is advisable, particularly to address deficiency-related comorbidities and potentially stabilize the peritoneal inflammatory environment.
- Personalized Supplementation: Future research should move away from universal protocols and focus on "dose-response" studies that account for individual genetic polymorphisms (VDR/VDBP).
- Synergistic Therapies: There is a promising potential for vitamin D to be used as an adjunct to existing hormonal or surgical treatments, possibly reducing the required dosage of pharmacological agents and their associated side effects.
- Long-term Trials: Larger, multi-center randomized controlled trials are needed to determine whether long-term vitamin D optimization can prevent disease recurrence post-surgery.

In summary, while vitamin D is not a standalone "cure" for endometriosis, it represents a safe, inexpensive, and biologically plausible supportive therapy that warrants further integration into the holistic management of the disease.

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