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HORMONALLY INDUCED GINGIVAL ENLARGEMENT: FOCUS ON EPULIS - A NARRATIVE REVIEW

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ABSTRACT

Gingival enlargement and epulis are common reactive lesions of the oral cavity with a multifactorial aetiology. While dental plaque is considered the primary initiating factor, systemic hormonal influences play a significant modulatory role in their development and clinical expression. This narrative review aims to summarise current evidence regarding the effects of sex steroid hormones on gingival enlargement and epulis, with particular emphasis on pregnancy-associated lesions and hormonal fluctuations throughout life.

A literature search was conducted using PubMed and Google Scholar, focusing on studies investigating the effects of oestrogen and progesterone on periodontal tissues. The available evidence indicates that sex hormones influence inflammatory responses, vascular function, immune activity, microbial composition, and connective tissue metabolism within the periodontium.

The findings suggest that hormonal changes do not directly initiate gingival disease but act as modifying factors that increase tissue susceptibility to local irritants, particularly dental plaque. Understanding these mechanisms is essential for improving prevention and clinical management of hormonally influenced gingival conditions.

KEYWORDS

Gingival Enlargement, Epulis, Pregnancy Tumour, Oestrogen, Progesterone, Periodontium

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Introduction

Gingival enlargement (GE) and epulis are commonly encountered lesions in dentistry. However, accurate diagnosis of these conditions remains challenging due to multiple contributing aetiological factors influencing their development and progression (Rajanikanth i in., 2012).

The most prevalent cause of gingival overgrowth is biofilm-induced inflammation, which primarily affects the interdental papillae and may present in either localised or generalised forms. Gingival enlargement is further modulated by hormonal influences, particularly during puberty and pregnancy. It is also associated with certain systemic medications (Savage & Daly, 2010).

The term epulis refers to a localised gingival overgrowth, frequently resulting from chronic irritation, including dental plaque and mechanical trauma caused by poorly fitting prosthetic or orthodontic appliances. Clinically, these lesions are considered reactive hyperplastic responses. The incidence of epulis varies among populations, with higher prevalence reported in females and young adults, likely due to hormonal and behavioural factors increasing susceptibility to gingival irritation (Maya Madhuri i in., 2024).

The aim of this narrative review is to provide an overview of current knowledge regarding the influence of hormonal changes on gingival enlargement and epulis, with particular emphasis on pathophysiological mechanisms, pregnancy-associated lesions, and clinical implications. It is hypothesised that hormonal fluctuations do not directly cause these lesions but act as modifying factors that enhance the gingival response to local inflammatory stimuli.

Methodology

A comprehensive literature search was performed across electronic databases, including PubMed and Google Scholar, with a focus on publications addressing the relationship between hormonal variations and gingival enlargement, with particular emphasis on epulis and pregnancy-associated lesions. The search strategy utilised combinations of the following medical subject headings (MeSH) and keywords: "epulis", "gingival enlargement", "pregnancy tumour", "hormones", "estrogen", "progesterone", and "gingiva". A further series of relevant articles were identified through a manual screening of the reference lists of the retrieved publications.

The final inclusion criteria required articles to be published in English, in a peer-reviewed format, and to be directly relevant to the topic. Priority was given to reviews, clinical trials, observational studies, and case reports discussing the influence of hormonal fluctuations on periodontal tissues and the development of gingival overgrowths. Conversely, publications that were not related to hormonal factors or which did not directly address gingival enlargement and epulis were excluded from the analysis.

Classification of gingival enlargement and epulis

The classification of GE as a distinct clinical entity is based on the underlying etiological factors and/or pathological changes in the tissue. A comprehensive overview of this classification, categorized by primary etiology and clinical features, is presented in Table 1.

Table 1. Classification of type of GE (Bali i in., 2023).

I. Inflammatory enlargement	1. Acute 2. Chronic
II. Drug-induced enlargement	1. General information 2. Anticonvulsants 3. Immunosuppressants 4. Calcium channel blockers
III. Enlargements associated with systemic diseases or conditions	1. Conditioned enlargement a. Pregnancy b. Puberty c. Vitamin C deficiency d. Plasma cell gingivitis e. Non-specific conditioned enlargement (pyogenic granuloma) 2. Systemic diseases that cause GE a. Leukemia b. Granulomatous diseases
IV. Neoplastic enlargement (gingival tumours)	1. Benign Tumours 2. Malignant tumours
V. False enlargement	

In accordance with the 2018 classification of periodontal and peri-implant diseases, epulis is categorized as a reactive lesion of non-plaque-induced gingival diseases, including fibrous epulis, calcifying fibroblastic granuloma, vascular epulis (pyogenic granuloma, PG), and peripheral giant cell granuloma (PGCG). The most widely accepted histological classification of epulis currently includes four subtypes. The incidence of different types of epulis varies among regions and populations. Despite apparent similarities in aetiology, each subtype induces distinct clinical and histological alterations, consequently necessitating different treatment approaches and prognostic assessments, including consideration of recurrence rates. Therefore, classification is essential for the development of effective treatment plans („Epulis", 2025).

Physiology of Sex hormones and Periodontal Tissues

Sex steroid hormones (SSH) have numerous physiological functions. Besides sexual development and reproduction, SSH are active in practically every tissue of the body, including the oral cavity(P. E. Cornejo Ulloa i in., 2024).

Oestrogen and progesterone exert a significant impact on the periodontium via the designated receptors, namely the oestrogen receptor and the progesterone receptor. The localisation of these receptors in the human periodontium demonstrates that periodontal tissues are the target tissues for these hormones(Khodorovskyi i in., 2021).

Oestrogen demonstrates a significant influence on the cytodifferentiation of stratified squamous epithelium, in addition to the synthesis and maintenance of fibrous collagen. The action of this hormone on target cells results in a modification of the effectiveness of the epithelial barrier with regard to bacterial insult and collagen maintenance and repair(Jafri i in., 2015).

Progesterone is associated with increased microvascular permeability. This, in turn, modifies the rate and pattern of collagen production in the gingiva.

Furthermore, progesterone has been shown to increase folate metabolism, stimulate the production of prostaglandins and enhance the chemotaxis of polymorphonuclear leukocytes (Khodorovskyi i in., 2021).

Oestrogen and progesterone directly modulate microcirculation, inducing the swelling of endothelial cells and pericytes of the venules, while promoting the adherence of granulocytes and platelets to vessel walls, which may lead to microthrombi formation. The disruption of perivascular mast cells enhances vascular permeability and proliferation. Collectively, these cellular alterations can significantly exaggerate the inflammatory response of periodontal tissues during periods of hormonal fluctuation, such as puberty, pregnancy and postmenopause (Jafri i in., 2015).

Mechanisms of Hormonally Influenced Gingival Enlargement

Sex steroid hormones, primarily oestrogen and progesterone, act on specific receptors within gingiva, the periodontal ligament, and various immune cells, thereby altering how these tissues respond to dental plaque. The pathophysiological effects of these hormones can be categorized into four distinct, interconnected pathways (Jafri i in., 2015; Jawed & Tul Kubra Jawed, 2025).

5.1 Inflammatory Response

Dental plaque serves as the primary etiological factor in periodontal diseases. Although the presence of pathogenic bacteria is necessary, host susceptibility ultimately determines disease progression. Systemic factors, particularly oestrogen levels, can profoundly modify this susceptibility. Consequently, the periodontium - a complex structure surrounding and supporting the teeth, is composed of the gingiva, periodontal ligament, cementum, and alveolar bone exhibits a heightened inflammatory response to microbial plaque under the influence of female sex steroid hormones during distinct life stages, including puberty, pregnancy, oral contraceptive therapy and postmenopause (Jafri i in., 2015).

Similarly to oestrogen, progesterone modulates the periodontal tissues either independently or synergistically. This hormone enhances vascular permeability, reduces glycosaminoglycan synthesis, and alters collagen production, while high concentrations downregulate the expression of interleukin-6 (IL-6). Furthermore, in conjunction with oestrogen, progesterone accelerates folate metabolism in oral mucosa, consequently affecting tissue repair mechanisms and the synthesis of prostaglandin E2(PGE2)(10,11).

The modulatory effects on oestrogen and progesterone on the host immune response are characterized by the regulation of pro-inflammatory cytokine production, including interleukin-1 beta (IL-1B), tumor necrosis factor-alpha (TNF-A), and IL-6. This regulatory process contributes to alterations in the inflammatory balance within periodontal tissues, thereby increasing susceptibility to plaque-induced inflammation (Palanisamy, 2025).

This mechanism is particularly active during pregnancy, when elevated concentrations of oestrogen and progesterone are associated with increased susceptibility to gingival inflammation. Consequently, this predisposes patients to pregnancy-associated GE, particularly pregnancy epulis („Epulis”, 2025; Tilakaratne i in., 2000).

5.2 Angiogenesis and Vascular Alterations

Hormonal fluctuations exert a profound influence on a woman’s periodontal health by altering the vasculature and subsequently affecting the inflammatory process. Specifically, oestrogen drives gingival tissue proliferation, whereas progesterone exhibits a vasodilatory effect and stimulates the creation of new blood vessels. These cellular modifications can promote the clinical onset of gingivitis, with a distinct potential for progression to periodontitis (Kulkarni i in., 2022; Sathish i in., 2022).

Elevated hormone levels significantly contribute to the development of Granuloma Gravidarum (GG). Oestrogen and progesterone concentrations peak during late pregnancy profoundly affecting periodontal tissues and their microvascular networks. Specifically, oestrogen upregulates vascular endothelial growth factor (VEGF) expression in macrophages, which triggers gingival vasodilatation and subsequent lesion growth. The vascular effects of progesterone appear to be even more pronounced than those of oestrogen. Consequently, these pregnancy tumours characteristically reach their peak size during second and third trimesters, while their incidence remains lowest during the first trimester („Epulis”, 2025; Reeves, 2025; Whitaker i in., 1994).

5.3 Microbiological changes

The oral microbiome has been identified as a potential indicator of oral pathologies and hormonal imbalances (Ionaș i in., 2025).

Sex hormones interact directly with specific oral pathogens; for instance, Prevotella intermedia can utilise estradiol and progesterone as growth substitutes, particularly under conditions of vitamin K limitation. This utilization shifts the microbial balance, favouring the proliferation of Prevotella species during periods of elevated hormone levels. Furthermore, Porphyromonas gingivalis significantly alters gingival tissues and modulates the local inflammatory response. Given its dual capacity to simultaneously activate and inhibit pro-inflammatory cytokine production and cellular immunity, fluctuating female hormones likely alter the tissue microenvironment to facilitate enhanced colonization at the periodontal level (P. Cornejo Ulloa i in., 2021; Ionaș i in., 2025).

Importantly, hormone levels alone do not induce gingival changes; rather, they alter periodontal tissue susceptibility to microbial plaque, thereby indirectly contributing to the progression of periodontal disease (Di Stefano i in., 2022; Ionaș i in., 2025).

5.4 Connective Tissue Metabolism

While gingival connective tissue is destroyed due to collagen degradation in periodontal lesions, collagen accumulates in gingival overgrowth lesions. Therefore, periodontitis and gingival enlargement can both be characterised by an imbalance in collagen metabolism. Gingival overgrowth clinically manifests as fibrosis and inflammation due to poor plaque control resulting from the formation of pseudopockets formed by the overgrown gingiva (Dieterle i in., 2021; Naruishi, 2022; Trackman & Kantarci, 2004).

The homeostasis of the periodontal ligament (PDL) is influenced by oestrogen, which modulates collagen turnover within the extracellular matrix. This hormone enhances the synthesis of collagen types I and III, thereby ensuring the necessary strength and flexibility of the ligament. Moreover, tissue architecture and remodeling are maintained through oestrogenic control over matrix metalloproteinases (MMPs) (Dieterle i in., 2021; Palanisamy, 2025).

On the other hand, progesterone contributes to these tissue changes by increasing vascular permeability and fluid accumulation, which enhances gingival edema and inflammatory enlargement within periodontal tissues. Ultimately, gingival fibroblasts play a crucial role in extracellular matrix remodelling; under hormonal influence, these cells can drive excessive collagen deposition, contributing directly to reactive gingival overgrowth (Naruishi, 2022; Sathish i in., 2022).

Pregnancy-associated epulis

A distinct clinical variant of epulis is pregnancy epulis- otherwise known as pyogenic granuloma or granuloma gravidarum. It is a benign, inflammatory reactive hyperplasia of the connective tissues, which manifests as a nodular formation on the oral mucosa. It is most frequently observed on the gingiva and alveolar crest, followed by the buccal mucosa, tongue, and lips. Such changes are commonly attributed to constant, low-grade local irritation, traumatic injury, or fluctuating hormonal factors. From a histological perspective, the overlying surface epithelium may be characterised by hyperkeratosis, focal ulceration, or complete structural intactness (Goh i in., 2025; Meshram i in., 2023).

This clinical entity is observed with an incidence ranging from 1.7% to 8.5% in the pregnant population („Epulis”, 2025).

Clinically, GG presents as soft, erythematous masses that are highly prone to bleeding and may develop surface ulceration. These lesions typically grow rapidly but vary in size, usually remaining under 2 cm. Importantly, due to postpartum hormonal stabilisation, these lesions frequently tend to regress spontaneously after delivery. Consequently, the primary focus of management emphasises early diagnosis and conservative intervention, with non-surgical treatment and the maintenance of meticulous oral hygiene serving as the first-line therapy. Surgical intervention is generally reserved for symptomatic cases or lesions that fail to resolve after childbirth (Agrawal, 2015; „Epulis”, 2025; Goh i in., 2025).

Hormonal changes beyond pregnancy

Fluctuations in sex steroid hormones throughout a woman's lifespan—spanning puberty, pregnancy, the use of oral contraceptives, and menopause—profoundly modulate the immune response, thereby altering periodontal tissue susceptibility to inflammatory diseases. During puberty, elevated levels of sex hormones exacerbate the gingival inflammatory response to dental plaque, a clinical phenomenon recognized as puberty-associated gingivitis (P. E. Cornejo Ulloa i in., 2024; Kanmaz i in., 2025; Mariotti, 1994).

Similarly, the administration of oral contraceptives can heighten gingival sensitivity to bacterial biofilms. Although modern low-dose formulations have significantly mitigated this risk, a predisposition to gingival inflammation persists in the absence of meticulous plaque control. Conversely, the postmenopausal period is characterized by a profound decline in oestrogen levels. This hormonal deficiency impairs the regenerative capacity of the oral mucosa and diminishes tissue barrier integrity, rendering the periodontium more susceptible to degradation (Castro i in., 2021; Mariotti, 1994).

In general, while hormonal shifts do not initiate periodontal disease independently, they significantly increase tissue reactivity. Consequently, even in the presence of stable plaque levels, hormonal fluctuations can modulate the degree of gingival enlargement and inflammation, highlighting the important role of endocrine homeostasis in maintaining periodontal health.(Castro i in., 2021; P. E. Cornejo Ulloa i in., 2024; Kanmaz i in., 2025; Mariotti, 1994).

Discussion

The findings of this review highlight the multifactorial nature of gingival enlargement and epulis, with sex steroid hormones acting as important modulatory factors in disease expression. Although dental plaque remains the primary aetiological factor in periodontal inflammation, hormonal influences significantly alter host response, thereby affecting disease severity and progression.

Oestrogen and progesterone exert their effects through specific receptors in periodontal tissues, influencing vascular permeability, immune regulation, inflammatory mediator expression, and fibroblast activity. These changes result in an exaggerated inflammatory response to bacterial plaque, particularly during periods of hormonal fluctuation such as puberty, pregnancy, oral contraceptive use, and menopause.

Pregnancy-associated epulis (granuloma gravidarum) represents a clear example of hormonally influenced gingival overgrowth. Elevated levels of oestrogen and progesterone during pregnancy promote angiogenesis, vascular proliferation, and immune modulation, leading to rapid lesion growth. The frequent spontaneous regression after childbirth further supports the role of hormonal modulation rather than autonomous neoplastic development.

Hormonal influences also extend to microbial composition, as certain periodontal pathogens, such as *Prevotella intermedia*, can utilize sex hormones as growth factors. This contributes to microbial imbalance and increased periodontal susceptibility during periods of elevated hormone levels.

Additionally, sex hormones influence connective tissue metabolism by regulating collagen turnover and extracellular matrix remodelling. Oestrogen enhances collagen synthesis, while progesterone contributes to vascular permeability and oedema formation. These combined effects promote gingival enlargement in susceptible individuals.

Overall, the evidence supports the concept that sex hormones do not directly cause gingival disease but act as modifying factors that amplify tissue response to local irritants.

Conclusions

Gingival enlargement and epulis are reactive lesions primarily associated with local inflammatory factors, particularly dental plaque, but significantly modulated by systemic hormonal influences. Sex steroid hormones, mainly oestrogen and progesterone, affect periodontal tissues through multiple mechanisms, including inflammatory modulation, vascular changes, microbial shifts, and connective tissue metabolism.

Hormonal fluctuations during key life stages such as puberty, pregnancy, contraceptive use, and menopause increase periodontal tissue susceptibility rather than directly inducing disease. Recognition of these interactions is important for improving prevention and management strategies in hormonally susceptible patients.

The authors declare that they have no conflict of interest.

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All authors contributed to the study conception, literature review, and manuscript preparation. All authors read and approved the final version of the manuscript.

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