CASE REPORT

Insights into Localized and Generalized Gingival Overgrowth - A Report of 15 cases and Concise Review of Literature

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Abstract

Background: Gingival overgrowth, characterized by an increase in the size of the gingival tissue, can be either localized or generalized and often arises from diverse etiological factors.

Case Presentation: This report discusses 15 such cases, localized and generalized, to provide insights into their varied presentations and etiopathogenesis. Localized overgrowth often results from factors like chronic irritation, poor oral hygiene, and systemic influences. Generalized overgrowth, frequently associated with drug therapy, systemic diseases, and hormonal changes, presents broader tissue involvement, impacting aesthetic and functional aspects of oral health.

Management: Histopathological examination revealed common inflammatory patterns across different cases, underscoring the role of plaque and secondary irritants. This report further highlights the challenges in diagnosis and managing gingival overgrowth, emphasizing the importance of identifying underlying causes to tailor effective treatment strategies. The findings underscore the need for comprehensive oral hygiene and regular monitoring, especially in patients on long-term medications or with systemic conditions.

Clinical Implications: This review, thus, synthesizes current knowledge and presents clinical insights, contributing to improved understanding and management of gingival overgrowth in diverse patient populations.

Keywords: Gingival overgrowth, oral lesion, etiopathogenesis, histopathology, oral hygiene

Introduction

Gingival overgrowth, a multi-faceted clinical condition, refers to the enlargement of the gingival tissue- either localized or generalized.[1] Localized gingival overgrowth refers to the enlargement of the gingiva confined to a specific area, typically affecting the gingival tissue around one or a few teeth. These overgrowths can be further categorized into isolated, discrete and regional types. Generalized gingival overgrowth affects the gingiva adjacent to nearly all teeth in the mouth. This type of enlargement is more diffuse and widespread, often linked to systemic factors or medications that induce hyperplastic changes across the entire gingival tissue.[2]

Etiopathogenetically, enlargements may be inflammatory, traumatic, drug-induced, associated with chronic habits or systemic conditions, neoplastic, or pseudo-enlargements.[1,3] Localized gingival overgrowth is relatively common and often results from local irritants such as plaque, calculus, or specific mechanical factors. Conditions like gingival abscesses or localized inflammatory responses due to trauma or infection are typical examples.[3,4] Generalized gingival overgrowth is less common but can be seen in specific populations, such as patients on certain medications or those with systemic conditions like granulomatous diseases or hormonal imbalances. The prevalence of generalized gingival overgrowth also varies with the patient's adherence to oral hygiene practices. Certain genetic and hereditary conditions also lead to generalized overgrowth of the gingival tissue.[3,5]

Understanding the distinctions between localized and generalized gingival overgrowth is essential for clinicians to develop targeted treatment plans and address the specific etiological factors contributing to each condition.

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This knowledge helps in optimizing patient outcomes and preventing long-term periodontal complications. The purpose of this article is to present some of the frequently encountered cases in a clinical setup and to discuss the possible causes that leads to these appearances.

Localized Gingival Overgrowth

This non-neoplastic condition, usually plaque-induced, might often result from a combination of systemic health issues, medication use, genetic predisposition, chromosomal anomalies, hormonal fluctuations, patient lifestyle and habits, and certain ethnic factors.[6] The gingival mucosa is frequently subjected to mild irritation due to mechanical forces during mastication, entrapment of food particles, substandard dental restorations and appliances, and the diverse microbiota present in the oral cavity, which may undergo pathogenic transformation under specific conditions. Consequently, the gingiva may exhibit hyperplastic responses, leading to various reactive gingival lesions.[7] Environmental factors are believed to exert a significant influence on the progression of these lesions, and their diverse clinical presentations may be attributed to multiple factors beyond mere plaque accumulation.[8]

In the present article, we report 7 cases of localized gingival overgrowth, viz fibroepithelial hyperplasia, pyogenic granuloma, peripheral ossifying fibroma, inflammatory fibrous hyperplasia, periodontal abscess, and gingival abscesses [table 1, figures 1a-d and 2a-c]. Etiological factors contributing to these lesions range from localized trauma to systemic influences, with poor oral hygiene identified as the primary instigator.

Fibroepithelial hyperplasia

The patient diagnosed with fibroepithelial hyperplasia had a medical history including medications for type II diabetes mellitus and alcoholic liver cirrhosis, indicating potential systemic contributions to gingival pathology. It has been suggested that serum levels of endocrine hormones may play a secondary role in modulating reactive hyperplasia of the gingival tissue.[9]

Pyogenic granuloma

Pyogenic granuloma, one of the most commonly encountered reactive gingival lesions, results from an exaggerated connective tissue response to localized trauma or irritation due to calculus, habits, restorations, or certain infections. Areca nut chewing history in the pyogenic granuloma case underscores the role of tobacco use as an exacerbating factor in gingival pathology.[10]

Peripheral ossifying fibroma

The occurrence of peripheral ossifying fibroma in an older male patient deviates from its typical presentation in young to middle-aged females, suggesting potential variations in disease manifestation across demographic groups.[11,12] The patient gave a history of trauma, that is in accordance with one of the proven etiologies of this condition.[3]

Inflammatory fibrous hyperplasia

The occurrence of inflammatory fibrous hyperplasia has been attributed to poor oral hygiene along with chronic local irritations.[13] The case reported presently complies with the proven etiology of poor oral hygiene.

Gingival and Periodontal abscesses

Abscesses, categorized as gingival or periodontal, are localized painful overgrowths having primary etiology as poor oral hygiene, further superimposed by factors ranging from subgingivally impacted foreign objects and improper restorations to extensive damage of the deeper periodontal structures.[14]

Localized overgrowths can cause significant discomfort, interfere with oral hygiene, and potentially lead to periodontal complications if left untreated. The varied presentations of reactive gingival lesions aid in their diagnosis, thus guiding treatment strategies aimed at reducing the recurrence. They are often indicative of localized factors that need to be addressed, such as eliminating the local irritants and proper supportive care. Additional surgical interventions might be required to excise the enlarged and persistent or recurrent lesions, thus enhancing the patient's ability to maintain oral hygiene.

Generalized Gingival Overgrowth

Systemic diseases such as diabetes mellitus, granulomatous conditions, and immunosuppressive disorders have been significantly correlated with gingival hypertrophy. Furthermore, medications including anticonvulsants are well-documented culprits in drug-induced gingival overgrowth. Additionally, hormonal fluctuations, particularly during puberty, pregnancy, and menopause, can exacerbate gingival tissue responses to local irritants, promoting hypertrophic changes. Genetic predispositions and chromosomal abnormalities further complicate the etiopathogenesis, suggesting a polygenic influence on tissue proliferation and inflammatory responses.[2,3,5]

In the present article, we report 8 cases of generalized overgrowth, viz inflammatory gingival overgrowth (dental plaque biofilm-induced), conditioned gingival overgrowth (hormonal), hereditary gingival fibromatosis, granulomatous inflammatory overgrowth (Crohn's disease), plasma cell gingivitis, drug-induced gingival overgrowth (Phenytoin and Amlodipine), and plasma cell granuloma of the gingiva (chronic generalized lipodystrophy), [table 2, figures 3a-d and 4a-d]. The amalgamation of various systemic factors with poor oral hygiene practices compounds the severity of gingival overgrowth.

Inflammatory gingival overgrowth (dental-plaque biofilm induced)

Plaque-induced gingival overgrowth, a reversible inflammatory response of the gingiva due to microbial overload, is prevalent in almost all age groups. Although it does not directly lead to damage of the periodontal structures and tooth loss, controlling the progress of gingival inflammation is a crucial primary preventive measure against periodontitis and a secondary preventive approach to prevent its recurrence.[15,16]

Conditioned gingival overgrowth (hormonal)

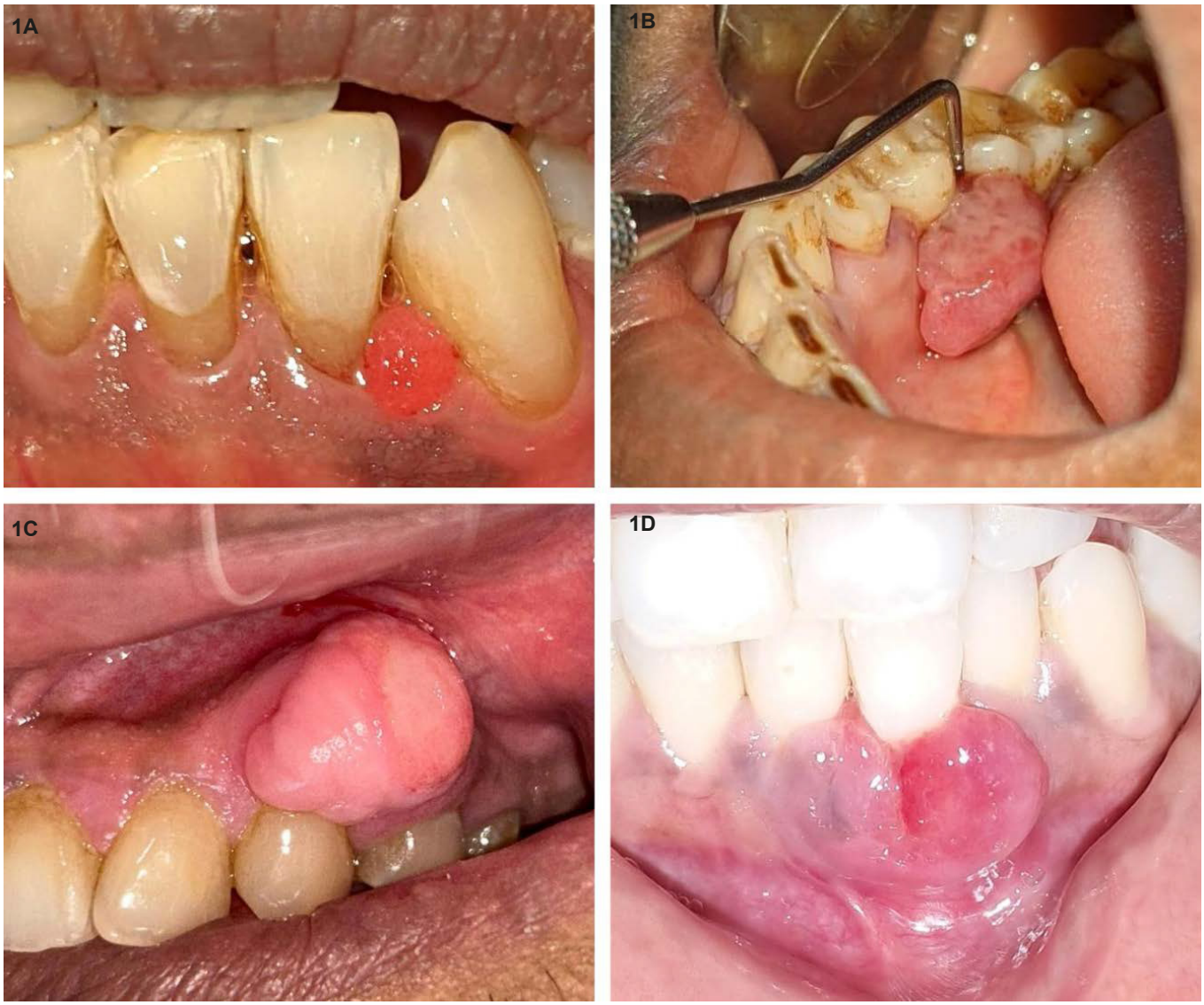
The incidence and severity of gingival overgrowth in

Table 1: Localized Gingival Overgrowth

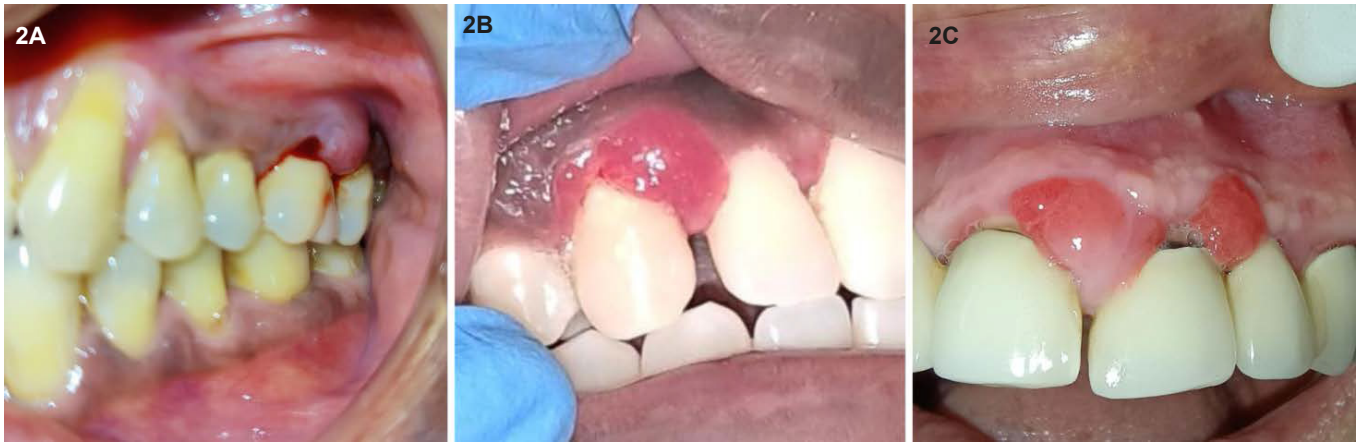
Sl. No.	PATIENT DETAIL and HISTORY	CLINICAL FEATURES	INVESTIGATIONS/ TREATMENT	DIAGNOSIS
1.	62-year-old male; history of medications for type II diabetes mellitus and alcoholicliver cirrhosis; gingival overgrowth in the left lower front teeth region.	well-circumscribed, soft, fixed mass, encompassing the interdental papilla, exhibiting bleeding on probing	- stratified squamous epithelium with parakeratosis and hyperplasia - connective tissue with densely packed collagen fibers - chronic inflammatory infiltrate composed of lymphocytes and plasma cells - blood vessels and extravasated red blood cells	Fibroepithelial hyperplasia (figure 1a)
2.	39-year-old female; medical history of anemia and a habit of areca nut chewing for the past 15 years; gingival overgrowth on the lingual aspect of the right lower back teeth region	well-demarcated, soft, pedunculated mass with a probing depth of 8mm, profuse bleeding upon palpation	- stratified squamous epithelium with parakeratinization, focal hyperplasia and ulceration - connective tissue demonstrated numerous, newly formed endothelial cells lining vascular channels, with plump endothelial cells scattered throughout the tissue - inflammatory infiltrate composed of extravasated cells	Pyogenic granuloma (figure 1b)
3.	systemically healthy 59-year-old male; swelling in the upper left front gingival area	asymptomatic, oval in shape, smooth, pink, firm, single, fixed mass; history of trauma concerning the left lower half of his face 3 months back, gradual increase in size	-thin-layered epithelium composed of squamous cells with a keratinized surface and ulceration in some areas - connective tissue with plump, actively reproducing fibroblasts scattered among dense collagen fibers and interconnected web-like calcification - numerous blood vessels and extravasated red blood cells	Peripheral ossifying fibroma (figure 1c)
4.	systemically healthy 35-year-old male, swelling in the lower front gingival area	well-circumscribed, pink, fibrous, single fixed mass exhibiting bleeding on palpation	- stratified squamous parakeretanised epithelium, hyperplastic in few areas and atrophic in other areas - myxomatous and fibrovascular connective tissue consisting of blood capillaries filled with red blood cells, abundant fibroblasts along with mixed inflammatory cells	Inflammatory fibrous hyperplasia (figure 1d)
5.	42-year-old male, history of medications for type II diabetes mellitus for past 5 years, swelling and loose tooth in left upper back gingival region	reddish, erythematous overgrowth involving the interdental papilla and marginal gingiva and extending beyond the mucogingival junction, associated with deep probing depth (buccal- 8mm, distobuccal- 7mm, grade 2 furcation involvement)	- regressed after initial non-surgical periodontal therapy, followed by open-flap debridement and guided tissue regeneration	Periodontal abscess (figure 2a)
6.	systemically healthy 45-year-old male, swollen and reddish gingiva in the upper right front tooth region, habit of using toothpick	reddish, erythematous overgrowth involving the interdental papilla and marginal gingiva	- overgrowth subsided after non-surgical periodontal therapy and counselling for habit cessation	Gingival abscess due to local irritation (figure 2b)
7.	71-year-old female, history of medication for hypertension (Telmisartan 40mg) for past 25 years	soft, friable, bright reddish fixed mass with profuse bleeding, neighbouring areas were pink but had minutely-lobulated texture with no bleeding on probing in those areas, completely edentulous lower arch, partially edentulous upper arch, fixed prosthesis supported maxillary anterior teeth	- overgrowth subsided after non-surgical periodontal therapy and recontouring of the prosthesis	Gingival abscess due to ill-fitting prosthesis/drug induced (figure 2c)

Table 2: Generalized Gingival Overgrowth

Sl. No.	PATIENT DETAIL and HISTORY	CLINICAL FEATURES	INVESTIGATIONS/ TREATMENT	DIAGNOSIS
1.	25-year-old systemically healthy female, bleeding while brushing and bad breath; initial slight ballooning of the papilla or marginal gingiva, progressively increased in size and extent	deep red, friable, and edematous gingiva, with a smooth surface, profuse bleeding on probing	- gingival overgrowth subsided after non-surgical periodontal therapy	Inflammatory gingival overgrowth (dental plaque biofilm-induced) (figure 3a)
2.	15-year-old systemically healthy female, swollen gingiva and bleeding gums for the past few months	soft and friable, pink and bright red in colour and smooth, shiny surface, along with bleeding on mild stimulation	- gingival overgrowth did not subside after multiple sessions of non-surgical periodontal therapy; patient put on recall visits for symptomatic treatment	Conditioned gingival overgrowth (hormonal) (figure 3b)
3.	15-year-old female, swollen gums in upper front tooth region since 7 years and unerupted upper anteriors; the size of the gingival mucosa had increased gradually covering more than half of tooth structure; family history	generalized fibrous gingival enlargement of tuberosities, anterior free/attached gingiva and retro-molar pads, unerupted teeth	- stratified squamous parakeratinised epithelium with elongated test tube-shaped rete ridges - connective tissue having large areas of fibrosis packed with dense bundles of collagen fibers and fibroblasts with blood vessels and few chronic inflammatory cells - small focal calcifications	Hereditary gingival fibromatosis (figure 3c)
4.	17-year-old male, swollen and bleeding gums for past 1 year, gingival mucosa had grown to the current size gradually	pink in colour, firm and leathery in consistency, with minutely pebbled surface; swollen lips	- abdominal pain, chronic diarrhoea and fever - endoscopy revealed patchy distribution of inflammation with skip lesions and longitudinal ulcers. - stratified squamous epithelium and connective tissue with inflammatory cells predominantly lymphocytes; Langerhan's giant cell with horseshoe-shaped nucleus and eosinophilic cytoplasm; caseous necrosis evident	Granulomatous inflammatory overgrowth (figure 3d) The patient was diagnosed to be suffering from Crohn's disease
5.	40-year-old female, complaining of swollen and bleeding gums and burning sensation on eating hot and spicy food	mild-reddish, friable, and edematous gingiva with smooth-shiny and granular surface texture, overgrowth encompassing marginal and attached gingiva, bleeding on slight provocation, swollen lips	- hyperplastic, spongiotic parakeratinized stratified squamous epithelium with areas of thin long rete pegs - lamina propria revealed extremely dense chronic inflammatory cell infiltrate, composed predominantly of plasma cells, and numerous dilated vascular channels - plasma cells had eccentric round nuclei with cartwheel chromatin pattern and an abundant cytoplasm	Plasma cell gingivitis (figure 4a)
6.	17-year-old male, history of epileptic seizures and taking medicine (Phenytoin) for the same for past 1 year, swollen gingiva covering the tooth structure	pink-colored, mulberry shape, firm and resilient with minute lobulations and no bleeding on probing	- hyperplastic para keratinized stratified squamous epithelium with long rete ridges - fibroreticular connective tissue with dense bundles of collagen fibers showing varying numbers of fibroblast and chronic inflammatory cell infiltrate	Drug-induced gingival overgrowth (figure 4b)
7.	40-year-old male, history of medication for hypertension (Amlodipine) for past 2 years, multiple sites of swollen gingiva	lobulated gingiva involving interdental papilla along with marginal and attached gingiva	- non-surgical periodontal therapy followed by referring the patient to physician for opinion regarding drug substitute	Drug-induced gingival overgrowth (figure 4c)
8.	17-year-old female, swollen gums, loose teeth, bad breath since 3 years; the size of the gingival mucosa had increased gradually	reddish, friable and generalized edematous overgrowth of gingiva with ulceration and exudation seen in few areas, pathologically migrated teeth, missing teeth, spontaneous bleeding upon slight provocation, swollen lips	- dry, pigmented skin, underweight, short stature - Glycated HbA1C 13.2%, RBS 332 mg/dl, Platelet count 6,39,000/ml, high Cholesterol and LDL level - connective tissue stroma consisted of dense chronic inflammatory cell infiltrate and abundant plasma cells - Plasma Cells had eccentrically placed nucleus, cartwheel shaped, along with hyperchromatic and clear golgi zone	Plasma cell granuloma of gingiva (figure 4d) The patient was diagnosed to be suffering from Congenital generalized lipodystrophy or Berardinelli-Seip congenital lipodystrophy



Figs. 1A - fibroepithelial hyperplasia **1B** - pyogenic granuloma **1C** - peripheral ossifying fibroma **1D** - inflammatory fibrous hyperplasia



Figss 2A - periodontal abscess, **2B** - gingival abscess, **2C** - gingival abscess

adolescents are influenced by various factors, with the significant rise in steroid hormone levels during puberty having a temporary effect on gingival inflammation.[15,17] Historically, there has been an observed increase in gingival inflammation in circumpubertal age in both genders, even without a corresponding rise in plaque levels. Although puberty-associated overgrowth shares many clinical characteristics with plaque-induced overgrowth, it is distinguished by the tendency to exhibit pronounced gingival inflammation in the presence of relatively small amounts of plaque during puberty.[17,18]

Hereditary gingival fibromatosis

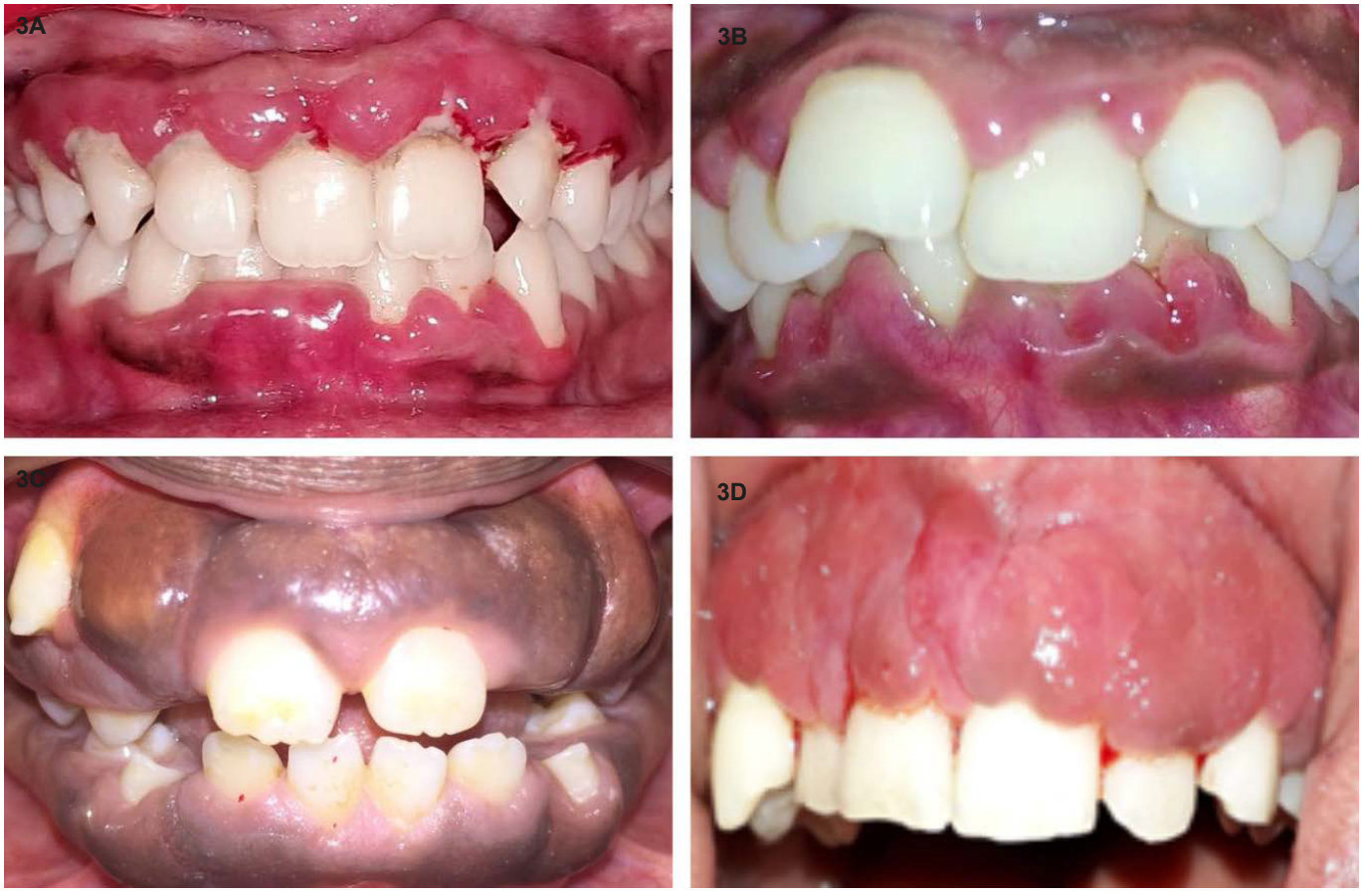
In the case of gingival fibromatosis, while the exact mechanism remains unclear, hereditary factors are predominantly implicated.[19] The diagnosis in this case was based on clinical presentation, family history, and histopathological features. Hereditary gingival fibromatosis can be inherited through autosomal dominant or recessive patterns.[20] This case is likely autosomal dominant, given that the patient's father and grandfather also had the same condition.

Granulomatous inflammatory overgrowth (Crohn's disease)

Gingival overgrowth due to granulomatous inflammation has a possible etiology of Crohn's disease, further confirmed by endoscopy. Crohn's disease, a multisystem inflammatory disorder, is believed to result from an inappropriate mucosal inflammatory response to intestinal bacteria in genetically predisposed individuals. It is hypothesized that immune system changes and environmental triggers are necessary for disease onset. Lesions can affect any part of the alimentary tract, including the mouth, with oral involvement (oral Crohn's disease) having a prevalence rate of 0.5-80%.[21-23]

Plasma cell gingivitis

The etiology of plasma cell gingivitis is challenging, but it is widely regarded as a hypersensitivity reaction.[3] Gargiulo described it as an immunological response that various factors, including allergens, neoplastic conditions, and of unknown origins can trigger.[24] Identifying the precise cause can be complex due to the diverse potential triggers, which underscores the importance of thorough clinical evaluation and patient history.[25] Epstein et al. speculated on the role of psychological disorders in the etiopathogenesis of plasma cell gingivitis.[26] In this context, it can be suggested that mental health conditions, such as stress, anxiety, or other psychological conditions, could manifest physically as plasma cell gingivitis.[27] This condition, thus, underscores the complex interplay between mental and



Figs. 3A - Dental-plaque biofilm-induced gingival overgrowth, **3B** - Hormone-influenced gingival overgrowth, **3C** - Hereditary gingival fibromatosis, **3D** - Crohn's disease-influenced granulomatous inflammatory overgrowth

physical health, highlighting the need for a holistic approach to diagnosis and treatment.

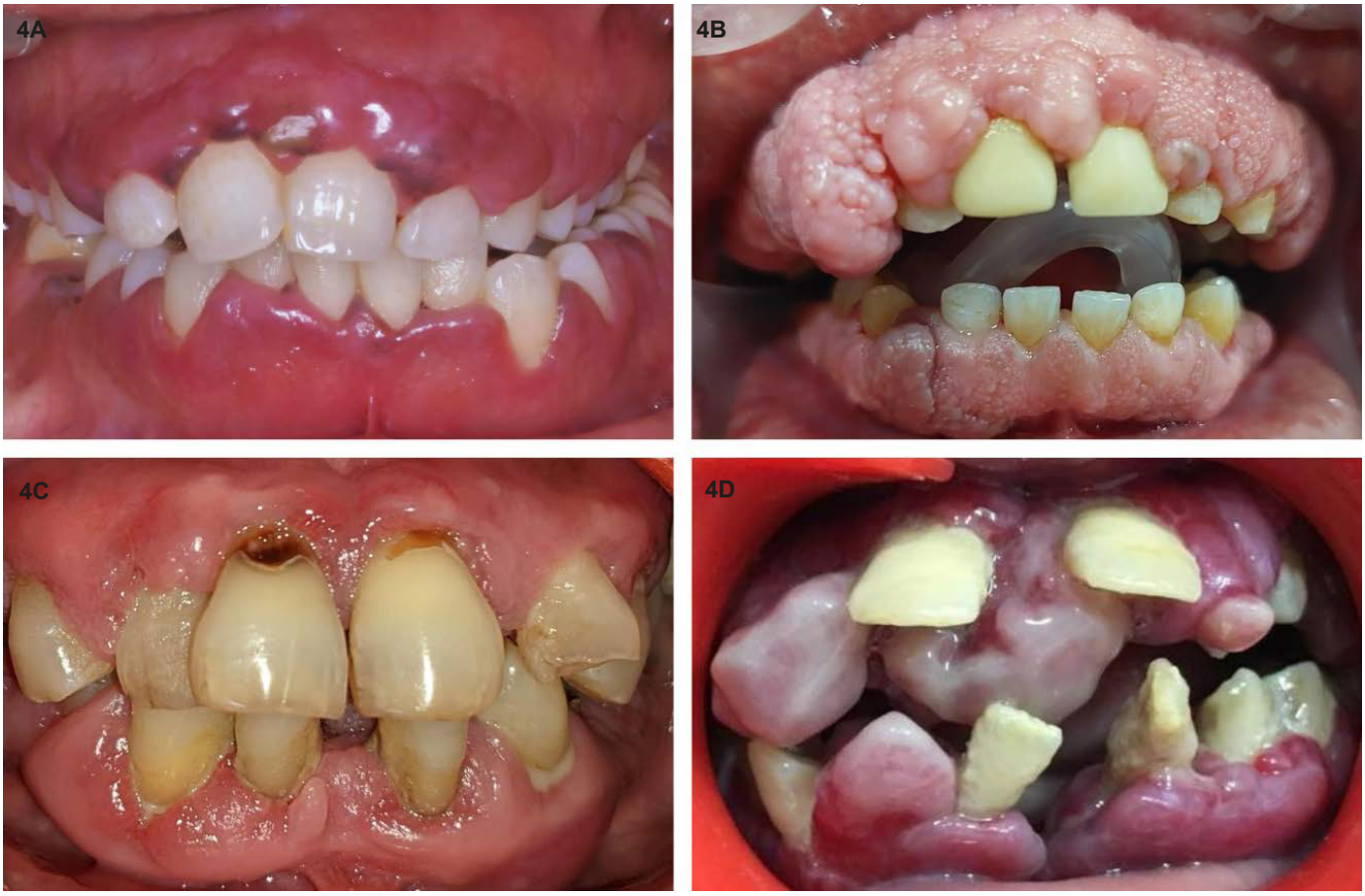
Drug-induced gingival overgrowth (Phenytoin and Amlodipine)

Drug-induced gingival overgrowth is an adverse effect of certain medications that are not intended to target the gingival tissue in the first place. The clinical manifestation of gingival overgrowth due to phenytoin (anti-epileptic) and amlodipine (calcium channel blocker) is majorly similar, with enlargement typically beginning in the papillary region. The presence of plaque deposits influences this overgrowth.[28] Phenytoin-induced overgrowth begins within the first month of treatment, becomes noticeable within three months, and peaks within the first year of medication use.[29] It often presents a granular or pebbly surface, with the facially and lingually enlarged inter-dental papillae connecting with adjacent papillae, resulting in pseudo-clefts. Amlodipine-induced overgrowth, on the other hand, presents a nodular or lobulated morphology, usually confined to the attached and marginal gingiva, particularly on the facial surfaces of the teeth.[28]

Plasma cell granuloma of the gingiva (Congenital generalized lipodystrophy)

Lipodystrophy syndromes encompass a diverse range of conditions marked by either complete or partial absence of adipose tissue. Metabolic dysfunctions largely underlie the significant comorbidities seen in these syndromes, including chronic complications stemming from poorly managed diabetes.[30] While oral manifestations are not typically linked with congenital generalized lipodystrophy, systemic disorders can occasionally manifest with unusual or uncommon oral symptoms due to underlying metabolic and endocrine disruptions.[31,32] Plasma cell granuloma, recognized as an inflammatory pseudotumor, is a non-neoplastic lesion with an uncertain etiology that is rarely observed in the oral cavity. Although the precise cause remains unidentified, various proposed theories suggest potential origins including localized irritation, an impacted foreign body, idiopathic antigenic stimulation, adverse effects of certain systemic medications, hormone therapy, or autoimmune mechanisms.[33-35] Therefore, it can be speculated that the patient's compromised systemic health may have contributed to the generalized occurrence of this particular lesion.

Generalized gingival overgrowth poses a broader clinical



Figs. 4A - plasma cell gingivitis, **4B** - drug-induced gingival overgrowth (phenytoin), **4C** - drug-induced gingival overgrowth (amlodipine), **4D** - congenital generalized lipodystrophy-influenced plasma granuloma of gingiva

challenge due to its widespread nature. It can significantly impact oral function, aesthetics, and overall oral health. Patients may experience difficulties in maintaining proper oral hygiene, leading to an increased risk of periodontal disease and dental caries. Management often requires a comprehensive approach, addressing underlying systemic conditions or modifying drug regimens in collaboration with medical professionals. In severe cases, surgical intervention may be necessary to reduce the gingival mass and improve the patient's ability to maintain oral hygiene.

Conclusion

This case report underscores the interplay of local and systemic factors in the development of gingival overgrowth and aims to highlight these distinctions, providing valuable insights for dental practitioners in managing various forms of gingival overgrowth. The comparison between localized and generalized gingival overgrowth is crucial for understanding the different etiological factors, clinical presentations, and treatment strategies. Localized overgrowth often results from specific local irritants such as plaque or trauma, whereas generalized overgrowth may be influenced by systemic conditions, medications, or genetic factors. Recognizing these differences helps clinicians tailor their diagnostic and therapeutic approaches, ultimately improving patient outcomes. Further research is warranted to elucidate the complex mechanisms underlying these lesions and to develop targeted therapeutic interventions for successful management and prevention of recurrence.

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