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A scoping review of plasma cell and eosinophil rich lesions of the oral cavity

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Abstract
This review aims to summarize the main features, potential causes, and diagnostic methods of plasma cell- and eosinophil-rich oral lesions, and to provide information on their clinical manifestations, pathogenesis, and treatment strategies. A comprehensive review of the literature was performed, examining studies of oral lesions characterized by significant plasma cell and eosinophil infiltration. Articles were analyzed for common conditions associated with these cell types, histopathological findings, clinical manifestations, and treatment methods. Plasma cell- and eosinophil-rich oral lesions are commonly associated with conditions such as plasma cell gingivitis, eosinophilic granulomatosis, oral manifestations of systemic diseases such as eosinophilic granulomatosis with polyangiitis, and allergic reactions such as eosinophilic stomatitis. Histopathological examination usually reveals an inflammatory infiltrate dominated by plasma cells and eosinophils, with different patterns depending on the underlying cause. Clinically, these lesions often present as painful, swollen, or ulcerated areas of the oral mucosa, requiring careful diagnostic evaluation.

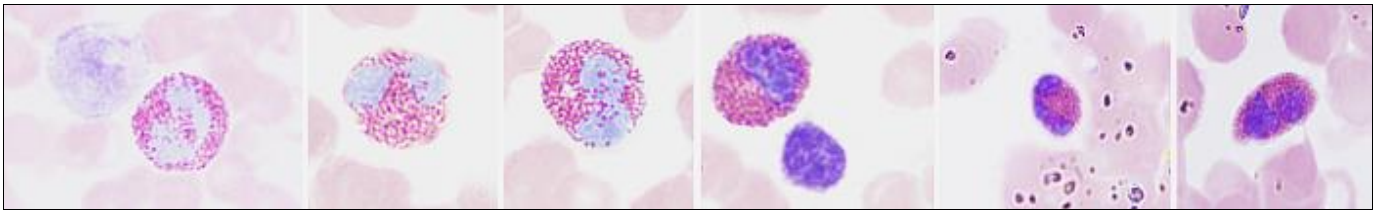
Keywords: Plasma cells, eosinophils, oral lesion, granuloma, mucositis

Introduction

Plasma cells and eosinophils are inflammatory cells that can help in the diagnosis of the lesion. Plasma cells are differentiated antibody-producing B lymphocytes, and their presence in oral lesions may be a sign of chronic inflammation or an immune response. When plasma cells are found in the oral cavity, they may be associated with conditions such as plasma cell gingivitis, chronic periodontitis, or oral lichen planus. In some cases, they are found in conditions such as plasma cell tumors or plasma cell granulomas, where there is an abnormal proliferation of plasma cells.

The role of plasma cells in these lesions is often related to chronic inflammation and immune dysfunction. Eosinophils are white blood cells involved in the body's response to parasitic infections and allergic reactions. Eosinophil-rich lesions in the oral cavity may occur in conditions such as eosinophilic granuloma, allergic reactions, or eosinophilic stomatitis. These conditions typically present as painful, swollen, or ulcerated lesions, and eosinophils contribute to the inflammation and tissue damage. In allergic reactions, such as drug reactions, eosinophils may accumulate as part of the immune system's response to an allergen or irritant. [1, 2, 3]

Overview of eosinophils features



Anatomy

Eosinophils have several vital biological roles, such as maintaining homeostasis, protecting the host from infections, regulating the immune system through canonical Th1/Th2 balance modulation, and having anti-inflammatory and anti-tumorigenic properties, in addition to their crucial role in the pathophysiology of eosinophil-associated diseases. [5]

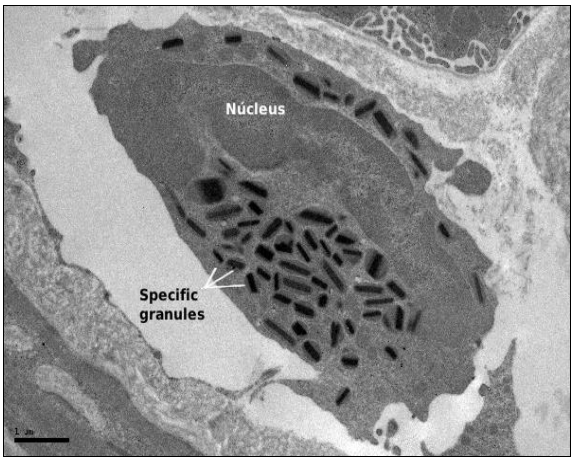
Leukocytes are present in connective tissues and blood. Eosinophils are granulocytes, a subset of leukocytes, along with neutrophils and basophils, due to the large number of granules in their cytoplasm. [11]

The name "eosinophil" is derived from their strong affinity for the dye eosin, which stains the acid molecules in these granules a pink-red colour. Under healthy conditions, eosinophils make up about 2% to 4% of the total leukocytes in the blood. Eosinophils can exit the bloodstream and migrate to the connective tissues of body organs, where their proportion may be significantly higher. [1, 4]

Morphology

Eosinophils are rounded cells with a diameter of about 15 µm, making them larger than other blood cells such as erythrocytes, lymphocytes, and basophils. Under light microscopy, eosinophils exhibit a nucleus with two lobes connected by a thin nuclear bridge. The cytoplasm contains numerous granules, referred to as specific granules, which stain orange-red with acidic dyes like eosin. [4, 5]

The specific granules of eosinophils display a crystallized central structure arranged in parallel layers, surrounded by a more or less electron-dense matrix. Additionally, light microscopy reveals numerous azurophilic granules in the cytoplasm, also known as non-specific granules. These granules are actually lysosomes containing acid hydrolases and other hydrolytic enzymes, which contribute to eosinophil functions. The cytoplasm also contains lipid bodies and tubule-vesicular structures known as eosinophil sombrero vesicles (EoSV). [1, 5]



Pathologic significance

The typical number of eosinophils in normal tissues is low. However, they become more numerous when recruited by healthy tissues, developing tissues, and pathological tissues, sometimes influenced by the immune system. Eosinophils make up 1–3% of circulating blood leukocytes, with a blood half-life of 8–18 hours, but they can survive for several weeks in tissues. As mature cells in the periphery, eosinophils are characterized by a large basophilic cytoplasm filled with numerous coarse granules containing cationic proteins; the major basic protein, eosinophil cationic protein, eosinophil-derived neurotoxin, and eosinophil peroxidase are included. [5, 6]

When stimulated, eosinophilic granules are released from eosinophils, serving as the first line of immune defence against infectious agents such as microbes, parasites, and allergens, which are associated with blood and solid cancers. While providing a defensive immune response against viral, bacterial, and helminth pathogens, eosinophils also contribute to tissue destruction by initiating inflammation through the release of eosinophil-derived cytotoxic mediators, playing a particularly harmful role in allergic disorders. [2, 6]

Eosinophil rich lesions in oral cavity

- **Oral eosinophilic ulcer**

An oral eosinophilic ulcer is an inflammatory reactive lesion of unknown aetiology, with trauma suggested as a potential cause; therefore, it is not frequently seen in clinical practice. Various terms have been used to describe this lesion, including "eosinophilic ulcer," "eosinophilic granuloma of soft tissue," and "traumatic ulcerative granuloma with stromal eosinophilia (TUGSE)." It was first described by Popoff in 1956 and later by Shapiro and Juhlin. Clinically, TUGSE appears as a painful single ulcer with indurated borders, primarily located on the surface of the tongue, but it can affect any site of the oral mucosa and is a self-limiting lesion that typically heals spontaneously. [4]

Due to its clinical presentation and prolonged duration, TUGUE should not be overlooked as it closely resembles OSCC. Microscopic observation revealed a specimen of oral mucosa partially coated by a stratified squamous epithelium with an extensive ulcerated area covered by fibrin and neutrophils. The lamina propria was constituted by a dense connective tissue with an intense chronic inflammatory infiltrate constituted by lymphocytes and plasma cells that spread diffusely deep into muscle bundles. Eosinophils were frequently seen amidst the inflammatory infiltrate. [5]

Eosinophilic ulcers can occur in individuals of any age and do not show a preference for sex. The tongue is the most common site for these lesions in the oral cavity, with an average size of 1.6 cm^2 at the time of diagnosis. These lesions are frequently ulcerated, may be tender, and can sometimes appear in multiple forms. The histological characteristics are distinctive but likely indicate a range of related disorders. Most eosinophilic ulcers tend to resolve on their own within a month, and recurrences are rare, occurring in less than 15% of cases. Eosinophilic ulcers are benign, self-limiting, reactive processes of the oral mucosa with an unknown origin. [4]

A histopathological examination of these ulcerative lesions shows that the underlying connective tissue deep into muscle bundles is chronically infiltrated with inflammatory cells such as neutrophils and lymphocytes, with eosinophils often observed as part of a mixed infiltration. Inflammatory cytokines and chemokines released from eosinophils have been proposed to play a role in the pathogenesis of this condition; however, the exact mechanism remains unclear. [7]

While their histological features are distinctive, they may be mistaken for atypical histiocytic granuloma, Angio lymphoid hyperplasia with eosinophilia, or, more critically, lymphoma. This condition likely represents a spectrum of related disorders with overlapping clinical and histological characteristics. Once the diagnosis has been confirmed histologically, conservative management is recommended. [4, 7]

Eosinophilic granuloma

Eosinophilic granuloma is a rare benign bone lesion that accounts for less than 1% of bone tumours. It primarily affects children under the age of 10, with the mandible being the most commonly affected site. This condition represents a mild localized form of oral Langerhans cell histiocytosis (LCH) without malignant transformation. Radiologically, it presents as tooth resorption, giving the appearance of teeth floating in the air. A histopathological examination shows scattered sheets of eosinophilic infiltrates. [3, 6]

Eosinophilic granuloma (EG) is a rare and mildest form of Langerhans cell histiocytosis. The presentation of EG can be monostotic, polyostotic, or can involve multiple organs. EG is characterized by the abnormal proliferation of Langerhans cells. Langerhans cells originate from mononuclear cell and dendritic line precursors. They are typically located in the bone marrow and have the capability to migrate into tissues, where they function as antigen-presenting cells to T lymphocytes. The proliferation of Langerhans cells can be triggered by viral infections (such as Epstein-Barr virus and Human Herpes virus-6), bacteria, and immune dysfunction, which leads to an increase in cytokines like interleukin-1

and interleukin-10. Additionally, EG of the lung is strongly associated with cigarette smoking. [7, 8]

The disease primarily affects the axial skeleton, including the skull, jawbone, spine, pelvis, ribs, and long bones. Lesions in the long bones are mainly found in the diaphysis. It often involves the soft tissues surrounding the bone. EG constitutes less than 1% of all bone tumours. The presentation of EG can be solitary, which rarely necessitates treatment, or multisystem, which requires aggressive therapy. The incidence rate of eosinophilic granuloma is about 70 %, but the jaws are involved twice as frequently as the oral soft tissues. [4, 6]

Histopathological Findings: The findings reveal Langerhans cells, which are mononuclear histiocyte-like cells characterized by prominent nuclear grooves resembling coffee beans, along with admixed eosinophils that contribute to the pink appearance of the cytoplasm. There are scattered multinucleated Touton-like giant cells, inflammatory cells, and areas of necrosis. The cells stain positively for CD1 antigen, S-100 protein, CD207 (Langerin), cyclin D1, PNA (peanut agglutinin), and BRAF VE1 in 50% of cases. There is a lack of nuclear atypia and atypical mitoses, which helps differentiate these findings from malignant conditions. [6, 8]

Electron Microscopy: The electron microscopy reveals Birbeck granules within Langerhans cells. These granules are cytoplasmic inclusions that have a "tennis racquet" shape and a zipper-like appearance. [9]

Pemphigus vegetans

Pemphigus vegetans is an autoimmune illness that is vesiculobullous is distinguished by the development of vegetative plaques in the oral mucosa and intertriginous space. Autoantibodies against the transmembrane protein desmosomes, which are in charge of keratinocyte cell-to-cell adhesion, are the reason. Patients usually have stomatitis at first, followed by mucosal blisters, cutaneous pustules, or flaccid bullets that usually affect the flexor, arms, legs, and trunk. [3]

Hyperkeratosis, pseudo epitheliomatous hyperplasia, and papillomatosis with acanthosis that forms a suprabasal cleft are histopathological characteristics. The tombstone appearance" is caused by intact hemidesmosomes connecting the basal cells to their basement membrane. Eosinophilic spondylosis, an inter epidermal eosinophilic micro abscess, and a thick eosinophilic dermal infiltration are all present in this lesion because of its high eosinophilic reaction. [10]

Bullous pemphigoid

The most prevalent autoimmune subdermal blistering condition is called bulbous pemphigoid, and it is brought on by autoantibodies directed against proteins found in the dermal-epidermal interface. It could occasionally be caused by drugs. Tense bullae and severe widespread pruritus are among its clinical manifestations. Bullous pemphigoid most commonly affects elderly patients between the ages of 60 to 80 years. [3]

A biopsy for hematoxylin and eosin staining will show a subepidermal split with eosinophils, and direct immunofluorescence will highlight the autoantibodies against the basement membrane zone. Urticarial lesions may present with spongiosis and eosinophils infiltrating the epidermis, also termed eosinophilic spongiosis, with an absence of subepidermal clefting. Peripheral eosinophilia is present in around 50% of treated patients. Subcutaneous split and superficial perivascular inflammatory infiltrates are among the histopathological characteristics seen. This condition is caused by an increase in perivascular eosinophils. [1, 6]

Oral lichenoid reactions

Oral lichenoid reactions usually commence by contact with pharmaceuticals, medications, or dental restorative materials, among other things. Histologically and clinically, oral lichenoid lesions are similar to oral lichen planus. Despite their similarities, oral lichenoid lesions might have different causes. An oral lichenoid reaction brought on by systemic medications is the well-known Grinspan syndrome, which is linked to diabetes and hypertension. White lacy lines, erythematous patches, plaque, erosion, and occasionally papules are among the clinical characteristics. [2, 5]

Histopathological characteristics include diffuse subepithelial infiltration of chronic inflammatory cells and eosinophils, perivascular inflammatory cell congregation, and white lacy lines (Wickham's striae), which frequently have a reticular appearance. The presence of cytoid structures in the granular and keratinized layers, focal parakeratosis, and focal disruption of the granular layer are examples of epithelial alterations. [2, 6]

Overview of plasma cells features

Anatomy

The Golgi apparatus is clearly visible and appears as a clear acidophilic area under a light microscope, which contrasts with the basophilic cytoplasm.

Plasma cells are relatively large, ovoid, antibody-producing cells that are derived from activated B lymphocytes. They are found in loose connective tissue and are especially prevalent in areas where antigens enter the body. They are also a natural component of salivary glands, lymph nodes, and hematopoietic tissue. Large clusters of peripheral heterochromatins are scattered throughout clear regions of euchromatin in the spherical, typically eccentrically oriented plasma cell nucleus, giving it the appearance of a cartwheel or an analogue clock face. [11, 20]

Morphology

The size of plasma cells ranges from 14 to 20 micrometres. They are ovoid to spherical cells with a pale perinuclear region that represents the Golgi apparatus and a large amount of deep blue cytoplasm. [12, 15]

Their coarse chromatin is organized in a clock face (art wheel) pattern around a spherical, eccentrically positioned nucleus. Few plasma cells are binucleate or multinucleate; the majority are uninucleate. They may have several spherical inclusions packed in their cytoplasm (Mott, grape, or morular cells) or cytoplasmic inclusions known as Russell bodies. Condensed immunoglobulins are found in dilated endoplasmic reticulum cisternae, which are seen in Russell bodies and Mott cell inclusions. [1, 13]

Pathologic significance

The synthesis, modification, and secretion of antibodies are the main tasks that plasma cells due to their endoplasmic reticulum and Golgi apparatus.

A variety of clinical diseases that fall under the categories

of plasma cell neoplasms and plasma cell immunodeficiency can result from deviations from appropriate plasma cell growth. The hallmark of plasma cell neoplasms is the abnormal growth of clonal plasma cells that produce monoclonal, heavy-chain, class-switched immunoglobulin, or M-protein. Plasma cell neoplasms can be categorized into a number of distinct pathological states due to their excessive growth and the accompanying symptoms that result from them. [2, 13]

A thorough diagnostic evaluation is necessary to determine the extent of the neoplasm. This evaluation includes the following specific tests and observations: a bone marrow biopsy to determine the percentage of clonal plasma cells and possible cytogenetic analysis; serum protein electrophoresis to assess for M protein; immunofixation to determine the amount and type of immunoglobulin; serum-free light chain analysis; a complete skeletal survey to determine the presence of lytic lesions; and a complete CBC with differential. [14]

Plasma cell rich – lesions in oral cavity

- **Plasma cell gingivitis**

Plasma cell gingivitis (PCG) is a rare benign condition of the gums, characterized by sharply defined red and swollen gingivitis that often extends to the mucogingival junction. As the name implies, it involves a diffuse and extensive infiltration of plasma cells into the sub-epithelial gingival tissue. This condition is a hypersensitivity reaction to certain antigens, often flavouring agents or spices found in chewing gums, toothpastes, and lozenges. [14]

The significance of this lesion lies in its potential to cause severe gingival inflammation, discomfort, and bleeding, and it may resemble more serious conditions. PCG is referred to by several other names, including atypical gingivostomatitis, plasmacytosis, idiopathic gingivostomatitis, and allergic gingiva-stomatitis. [14, 20]

Plasma cell gingivitis is a rare condition characterized by the extensive infiltration of plasma cells into the sub-epithelial gingival tissue. Clinically, the condition manifests as a diffuse reddening accompanied by edematous swelling of the gingiva, with a sharp demarcation along the mucogingival border. The cause of plasma cell gingivitis is not well understood, but the significant presence of plasma cells has led many authors to suggest that it may be an immunological reaction to allergens, which can be found in toothpaste, chewing gum, mint pastels, and certain foods. It has been proposed that strong spices and some herbs, such as chili, pepper, clove, and cardamom, may play a significant role. [20]

The addition of flavouring agents to chewing gum and dentifrices can lead to an inflammatory reaction in both attached and free gingiva, characterized by intense hyperaemic and erythematous changes. It is common for patients to report "bleeding from the mouth." Some authors categorize plasma cell gingivitis into three types: (1) caused by an allergen, (2) neoplastic, and (3) of unknown cause. The current case falls under Type 1, as the changes developed after prolonged use of herbal tooth powder. [14]

Microscopic analysis showed significant squamous hyperplasia accompanied by localized ulceration and a widespread dense infiltrate of plasma cells beneath the epithelium, consistent with PCG [Figure 7]. Observations at higher magnification revealed plasma cells that lacked cellular atypia. Each plasma cell exhibited an eccentric round nucleus featuring a cartwheel pattern of chromatin and a rich cytoplasmic presence. [14, 20]

Plasma cell mucositis

It is an uncommon, benign, inflammatory condition of the upper aerodigestive tract that is similar to plasma cell (Zoon) balanitis/vulvitis in both histology and clinical presentation. A localized hypersensitivity reaction of the gingivae to flavouring compounds (mostly cinnamon and cinnamon aldehydes) in toothpaste and chewing gums appears to be a unique kind of plasma cell gingivitis. Plasma cell mucositis has no recognized aetiology. It has been hypothesized that contact sensitization, actinic injury (of the lips), or an as-yet-unidentified viral trigger (such as Candida albicans or herpes simplex virus) may cause an immunological response in susceptible individuals. [15]

In the early stages of the disease, clinical symptoms include voice hoarseness, sore throat, dysphagia, itching, and burning mouth discomfort. Surface alterations could be cobblestone, granular, warty, or nodular. Gingival involvement can resemble granulomatosis with polyangiitis and look "strawberry-like." Patients frequently report both spontaneous and prompted gingival bleeding (i.e., to toothbrushing). The buccal and palatal mucosa are two more intraoral regions that are frequently impacted. Contact bleeding linked to tissue hyperaemia or a superimposed candidal infection can also happen, as might diffuse, sloughing, or vegetative oral ulceration. Strictures may result from involvement of the upper and lower airways. The mucosa of the nose, throat, larynx, trachea, bronchi, and oesophagus may also be impacted. [15]

A dense subepithelial infiltration of mature plasma cells is revealed by histopathological characteristics, which may also show pseudo epitheliomatous hyperplasia, Russell body development, and subsequent micro abscess formation. Although some eosinophils may be found in the inflammatory infiltrate on biopsy, they are not a predominant cell type; instead, plasma cells, lymphocytes, and macrophages make up the majority of cells in plasma cell mucositis, so eosinophil counts are typically not significantly elevated in this condition. [11, 13]

Plasma cell granuloma

The non neoplastic lesions known as plasma cell granulomas (PCGs) or inflammatory pseudotumor are mostly composed of innate immune cells such neutrophils, macrophages, and eosinophils as well as plasma cells that secrete antibodies. (PCGs) are benign inflammatory growths that are rarely observed in the oral cavity and frequently occur in the lungs. These lesions' precise aetiology and natural history are still unknown. According to some writers, it falls under the category of diseases linked to IgG4. The gastrointestinal system and lungs are the most commonly reported organs, but any area of the body may be affected. [16]

Microscopic analysis reveals an inflammatory proliferation with a majority of cells identified as polyclonal plasma cells by immunohistochemistry. A polytypic population of mature plasma cells, plasmacytoid, and small non transformed lymphocytes populate plasma cell granulomas, which are surrounded by fibroblasts, entrapped leptomeningeal cells, and variable fibrosis. There are Russell bodies and rare binucleate plasma cells, but no overtly dysplastic plasma cells. There are also dispersed

histiocytes that do not exhibit any S 100 protein immunoreactivity. The eosinophil count is usually low or absent (less than 1%) in a plasma cell granuloma. [17, 18]

Solitary polycytoma

An uncommon type of plasma cell dyscrasia known as solitary plasmacytoma manifests as a single mass of intraosseous or extramedullary monoclonal plasma cells. Although the exact cause of SPB is still unknown, a number of theories have been put forth that link radiation, chemical exposure, viruses, and genetic factors to the condition. According to cytogenetic research, chromosomes 13, 1p, and 14q were lost, while 19p, 9q, and 1q were gained. Interleukin-6 is thought to be a key growth factor in pathogenesis. Depending on the location and size of the lesion, symptoms may include paraesthesia (numbness), swelling, mobility of the teeth, localized pain, and occasionally a soft tissue mass. [19]

According to histopathological characteristics, plasmacytoid mononuclear cells in contact with amyloid material express anti-Lambda light chain antibody (polyclonal antibody) exclusively and mono-typically, while anti-Kappa light chain antibody (polyclonal antibody) is not expressed. Eosinophil granulomas are typically small, rarely measuring more than two to three centimetres. Polycytoma may have an eosinophilic count of 3–5%. [19]

Clinical implications

Eosinophils are a type of granulocyte found in numerous diseases. These versatile cells carry out a range of functions. Eosinophilia can occur in a broad range of conditions, from common allergies to cancers with metastasis. In individuals experiencing eosinophilia, it is essential to conduct comprehensive investigations to identify and exclude any underlying diseases. Eosinophils play a role in tissue destruction by initiating inflammation through the release of eosinophil-derived cytotoxic mediators, with a particularly detrimental role to play in allergic disorders.

Plasma cell lesions represent a diverse range of conditions, extending from reactive to immune-mediated and neoplastic lesions, each with distinct etiological mechanisms. Accurate identification of these lesions is crucial due to their implications for treatment and prognosis. Among reactive plasma cell lesions is plasma cell gingival lesion (PCG), a rare condition marked by the widespread infiltration of plasma cells within the gingival connective tissue. This lesion is believed to result from a hypersensitivity response to certain allergens, and some researchers categorize PCG into three types: those caused by allergens, those linked to neoplasms, or those with an indeterminate origin. Numerous case reports describe plasma cell granuloma as an isolated lesion with a similar cause or as a localized variant of PCG.

Conclusion

Plasma cell- and eosinophil-rich oral lesions represent a variety of inflammatory and immunologic processes. Early recognition of these lesions is essential to differentiate between benign conditions and those potentially more serious. A comprehensive approach including clinical, histopathologic, and immunologic evaluation is essential for accurate diagnosis and appropriate management. Further research is needed to clarify the mechanisms underlying these lesions and improve treatment strategies.

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