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CASE REPORT

ORAL PLASMA CELL GRANULOMA- A DIAGNOSTIC ENIGMA

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ABSTRACT

Plasma cell granuloma is a non neoplastic lesion classified and described under pseudoinflammatory tumor category. Frequently these lesions are found in lungs and rather uncommonly seen in the oral cavity, making diagnosis and treatment actually complicated. They can arise in areas such as brain, orbit, paranasal sinuses, head, neck, larynx, tonsils, ears, liver, kidney, stomach and heart. Intraoral plasma cell granulomas are seen involving tongue, lip, oral mucosa and gingiva. Its etiology, biological behavior, ideal treatment and prognosis are still unclear and rather controversial. Both clinically and histopathologically, it may be misinterpreted as various pathological entities thus necessitating the complete evaluation of patient and proper histopathological and immunohistochemical analysis of the tissue to rule out other lesions with poor prognosis. This case presents a 35 year old female with an enlargement in the right buccal mucosa masquerading as carcinoma and the histological evaluation revealed plasma cell infiltrates in the connective tissue. The immunohistochemistry revealed kappa and lambda light chains with a polyclonal staining pattern, which confirmed the diagnosis of plasma cell granuloma.

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INTRODUCTION

Plasma cell granuloma (PCG) is an idiopathic inflammatory pseudotumor (Bansal *et al*., 2013). This rare, nonneoplastic lesion consists of proliferation of inflammatory cells predominantly plasma cells in a fibrovascular background. It was first described in 1973 by *Bahadori and Liebow* (Jawanda *et al*., 2014). Since then it has been described by many authors using different terms as inflammatory myofibroblastic tumor, inflammatory pseudotumor, fibrocystic inflammatory proliferation, xantomatosis pseudotumor, benign myofibroblastoma and inflammatory fibrosarcoma (Ajay and Lahari, 2013; Reyes *et al*., 2015). It manifests mostly in the lung, but nearly any other organ may be involved, including the head and neck region (Phadnaik and Attar, 2010). It is very rare in the oral cavity and more so, on the gingiva. (Manohar and Bhuvaneshwari, 2011) In the gingiva, the lesion was first described in 1968 by *Bhaskar, Levin and Firch* (Amita *et al*., 2015). In the head and neck region it has been reported in the oral mucosa, temporal bone, tonsil, sub-mandibular region, paranasal sinuses, tongue and on the gingiva. (Manohar and Bhuvaneshwari, 2011) The etiopathogenesis, prognosis and treatment of this rare reactive lesion is unclear. It may arise due to periodontitis, periradicular inflammation due to the presence of a foreign body or may be due to an idiopathic antigenic cue (Sunil *et al*., 2013). An autoimmune mechanism has also been implicated. It is also associated with alteration of blood flow imposing congestive vasodilatation (Pandav *et al*., 2015). These lesions have no sex predilection and may occur at any age (Bansal *et al*., 2013). This benign inflammatory lesion requires biopsy and histopathological, immunohistochemical study to rule out possible neoplastic and plasma cell dyscrasias and thus establish a differential diagnosis with other similar diseases such as multiple myeloma. This paper presents a peculiar case of oral PCG that clinically mimicked an epithelial malignancy. Thus this case highlights the need to biopsy for unusual lesions to rule out potential neoplasms.

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Case report

A 35 year old female patient reported with a chief complaint of pain in the right upper back Tooth region since 10 days. There was no history of trauma or surgery. No relevant past dental history and the medical history was not contributory. On examination no extraoral swelling was noticed. On intraoral examination, a solitary swelling was seen on the right buccal mucosa, measuring approximately 3 x 3 cm in size. The growth was well circumscribed, erythematous and covered by pseudomembranous slough. On palpation the inspectory findings were confirmed. It was firm in consistency, borders of the lesion were indurated and fixed to the underlying structures. All laboratory investigations were normal. Based on the clinical findings, a provisional diagnosis of carcinoma was given. An incicional biopsy was done and the tissue was sent for histopathological examination. Routine haematoxylin and

eosin staining was done. Immunohistochemical staining for kappa and lambda immunoglobulin light chains was done. Histopathological examination of the specimen revealed a parakeratinised stratified squamous epithelium with rete ridges. No dysplastic changes were seen in the epithelium. The underlying connective tissue showed mixed inflammatory cell infiltrate composed of abundant plasma cells with typical eccentrically placed hyperchromatic, cart wheel nucleus arranged in sheets without any atypia. Other cellular elements namely lymphocytes, neutrophils, eosinophils and histiocytes were usually surrounded by connective tissue septa. Abundant blood vessels were observed. The histopathological appearance when correlated with clinical features was suggestive of PCG of the buccal mucosa. Immunohistochemical stain for kappa and lambda light chains showed a polyclonal plasma cell population, thereby a confirmatory diagnosis of PCG was made.

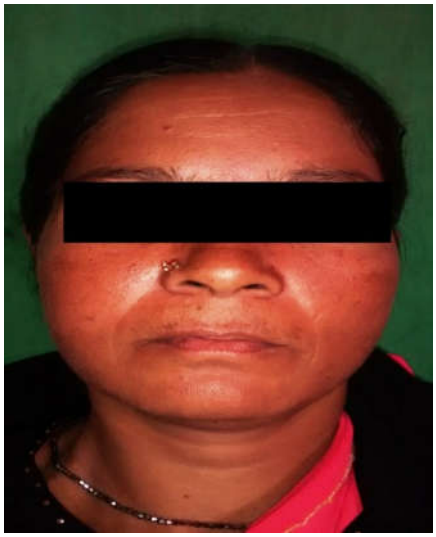

Fig.1. Extraoral picture showing no abnormality

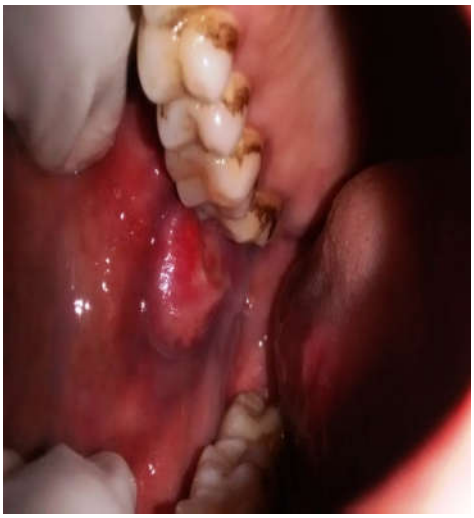

Fig.2. Solitary swelling on the right buccal mucosa, measuring approximately 3x3cm in size

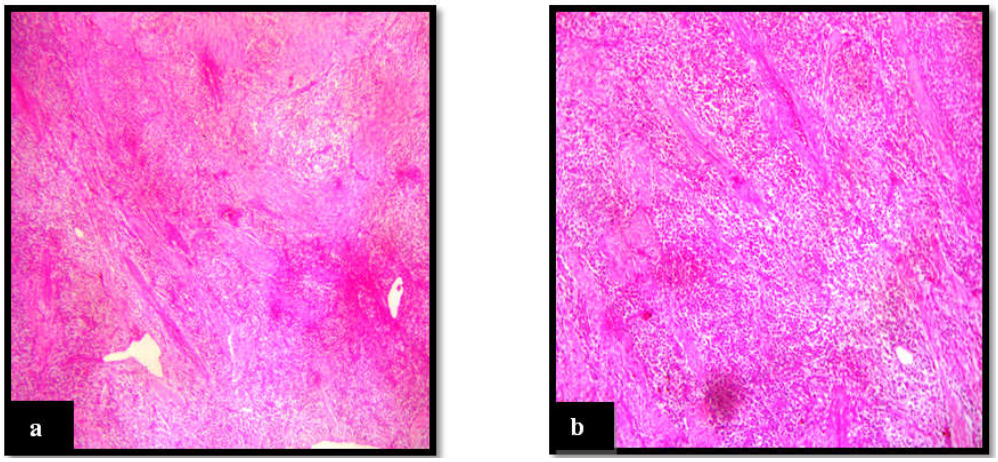

Fig.3. (a) Scanner view photomicrograph of the biopsy specimen, which reveals foci of plasma cells separated by connective tissue septa (H and E, 4X). (b) Low power photomicrograph of the biopsy specimen depicting plasma cells separated by connective tissue septa (H and E, 10X)

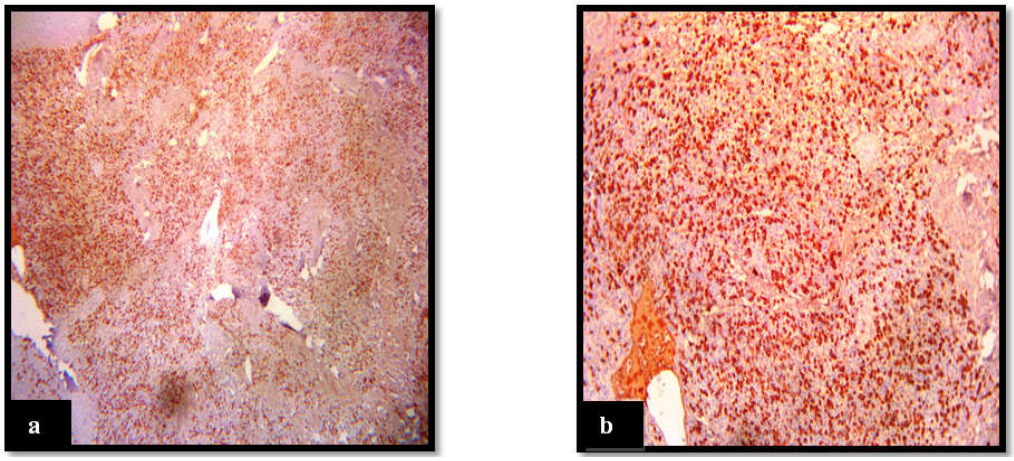

Fig.4. (a) (b) Immunohistochemical photomicrograph showing Kappa Lambda chains immunoreactivity present in the cytoplasm of plasma cells. (4x, 10x)

DISCUSSION

Plasma cells are a prominent constituent of loose connective tissue where antigens tend to enter the body (Jeyraj *et al*., 2015). These terminally differentiated B lymphocytes are typically found in the red pulp of the spleen, medulla of the lymph nodes, tonsils, lamina propria of the entire gastrointestinal tract, mucosa of the nose and upper airway and sites of inflammation. These cells are characterized by basophilic cytoplasm with an eccentrically placed nucleus. They range in size from 14-20µm (Jeyraj *et al*., 2013). The phenomenon of plasma cell infiltrate was first described by Zoon in 1952 when he described balanitis plasmacellularis. Since then, plasma cell infiltrates have been found on the vulva, buccal mucosa, palate, nasal aperture, gingiva, lips, tongue, epiglottis, larynx and other orificial surfaces (Ajay *et al*., 2013; Sunil *et al*., 2013; Anila *et al*., 2008). PCGs were considered to be highly uncommon, non-neoplastic, reactive lesions, which were first brought to the attention of health care practitioners during the late 1960s and early 1970s. (Ajay and Lahari, 2013) During this period, cases of plasma cell infiltrates of the lips, gums, and tongue were described primarily in the dental literature under the names of atypical gingivostomatitis, idiopathic gingivostomatits, and allergic gingivostomatitis. The lesions were thought to be a result of a reaction to chewing gum, dentrifices, and other foreign substances, although extensive allergy testing had been inconclusive (Manohar and Bhuvaneshwari, 2011; Jeyraj *et al*., 2013). Later this lesion has been described as inflammatory myofibroblastic tumor, which was classified by world health organization (WHO) in the year 2002 under intermediate (rarely metastasizing) fibroblastic/myofibroblastic tumors. It has been described by WHO as a distinctive lesion composed of myofibroblastic spindle cells along with inflammatory infiltrate of plasma cells, lymphocytes and eosinophils (Ajay and Lahari, 2013). Clinically, PCG takes at least two morphological types in the oral mucosa: exophytic/tumor or unilateral ulcerative (Reyes *et al*., 2015). In this case it adopted unilateral ulcerative type. According to the limited literature reviewed there was not another case with clinical ulcerative morphology in the buccal mucosa, if so, this would be the first case with this type of manifestation. If the lesion looks like a tumor, differential diagnosis must be performed first because of its frequency with squamous cell carcinoma, with extramedullary plasmacytoma and multiple myeloma in the oral cavity. If the lesion looks like an ulcer, differential diagnosis will be performed with autoimmune processes such as pemphigus vulgaris, bullous pemphigoid, mucous membrane pemphigoid and erosive lichen planus, and systemic lupus erythematosus. (Reyes *et al*., 2015)

The PCG should be distinguished from other plasma cell rich lesions like osseous solitary plasmacytoma, multiple myeloma and soft tissue plasmacytoma (Bansal *et al*., 2013; Amita *et al*., 2015). As PCG may be benign, but plasmacytoma may show early stages of multiple myeloma differentiating the type of soft tissue tumor is mandatory (Bansal *et al*., 2013). Histologically plasmacytoma shows monomorphic population of plasma cells with presence of plasmablast, bi and multinucleation and many Russell and Dutcher bodies. In contrast, however, PCG though shows predominance of plasma cells, there will be intermingling of other inflammatory cells like lymphocytes, mast cells and eosinophils (Amita *et al*., 2015). Hematologic diseases like multiple myeloma may mimic this condition, but they are composed of neoplastic monoclonal plasma cells. Thus a light chain restriction can be demonstrated by immunohistochemistry or, preferentially by insitu hybridization. On the contrary PCG at any site always consists of polyclonal plasma cells (Laco *et al*., 2015).

Conclusion

Thus this article laid an emphasis on submission of every tissue excised for microscopic examination regardless of their clinical impression so that proper and accurate diagnosis can be made. PCG is a rare pathology and the main contributing pathways for the pathogenesis are hard to pin down, so documentation of cases will help in understanding its etiology and nature.

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