

## Plasma Cell Granuloma of Gingiva: A Case Report

Firdos Saba<sup>1\*</sup>, Mohammed Mustafa Samir<sup>2</sup>, Aseel alhindi<sup>3</sup>, Ehab Alsayyed<sup>4</sup>

<sup>1</sup> Associate Consultant, Department of Anatomic Pathology, King Abdullah Medical City, Makkah, Saudi Arabia.

<sup>2</sup> Associate Consultant, Department of Intensive Care Unit, King Abdullah Medical City, Makkah, Saudi Arabia.

<sup>3</sup> General Dentist, Umm Alqura University, Makkah, Saudi Arabia.

<sup>4</sup> General Dentist, Umm Alqura University, Makkah, Saudi Arabia.

**Received date:** 30 January 2024; **Accepted date:** 23 February 2024; **Published date:** 29 February 2024

**Corresponding Author:** Firdos Saba, Associate Consultant, Department of Anatomic Pathology, King Abdullah Medical City, Makkah, Saudi Arabia.

**Citation:** Firdos Saba, Mohammed Mustafa Samir, Aseel alhindi, Ehab Alsayyed. Plasma Cell Granuloma of Gingiva: A Case Report. Journal of Medical and Clinical Case Reports 1(1). <https://doi.org/10.61615/JMCCR/2024/FEB027140228>

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### Abstract

Plasma Cell Granuloma (PCG) is an uncommon tumor that's usually located in the lung. In rare events, it can occur in the oral cavity. However, the gingiva is the least common site. Here, we present a rare case of a 57-year-old man with a gingival mass. It had been growing for a year and measured 1 cm. His medical history includes hypertension and kidney transplant. The patient was on nifedipine and tacrolimus. The tumor was treated by excision and requested pathological evaluation. The lesion displayed heavy plasma cell infiltration in the sub-epithelium. The diagnosis was confirmed through immunostains. Eventually, gingival PCG was reported.

**Keywords:** Plasma Cell Granuloma, Granuloma, Gingival Lesion, Reactive lesion, pseudotumor.

### Introduction

Plasma Cell Granuloma (PCG) is an inflammatory lesion that could affect any region of the body. Its' exact incidence is controversial, but it is generally considered a rare lesion [1]. It is known as a "pseudotumor" that mainly consists of polyclonal plasma cells, histocytes, lymphocytes, and blood vessels [3]. Due to the histological familiarity with Plasma Cell Gingivitis, it was described as a variant of this inflammatory process [11].

PCG of the intraoral region has been reported in different sites in the oral cavity; however, the least common site is believed to be the gingiva [12]. Oral

PCGs affect patients in their 30s to 40s with a female predilection. They are usually radiographically concerning and may be mistaken for malignancy as they appear with infiltrative borders [10].

A chronic immune response to an antigen aids in the formation process. Such as periodontitis, radicular inflammation, or foreign substance reaction [4]. This article reports a rare case of PCG in the gingiva in an adult male. To the best of our knowledge, this is the first reported case of PCG in the oral cavity with a history of organ transplantation.

### Case presentation

A 57-year-old male patient with a history of hypertension, ischemic heart disease, and renal transplant five years ago. The patient is currently on nifedipine and immunosuppression: Mycophenolate, Mofetil, Prednisolone, and Tacrolimus. His thyroid gland revealed multiple non-toxic benign

thyroidal nodules. His most recent blood work shows a slight increase in monocytes. Urine sample testing was carried out to detect Bence Jones protein and exclude multiple myeloma. Urine testing was found to be normal.

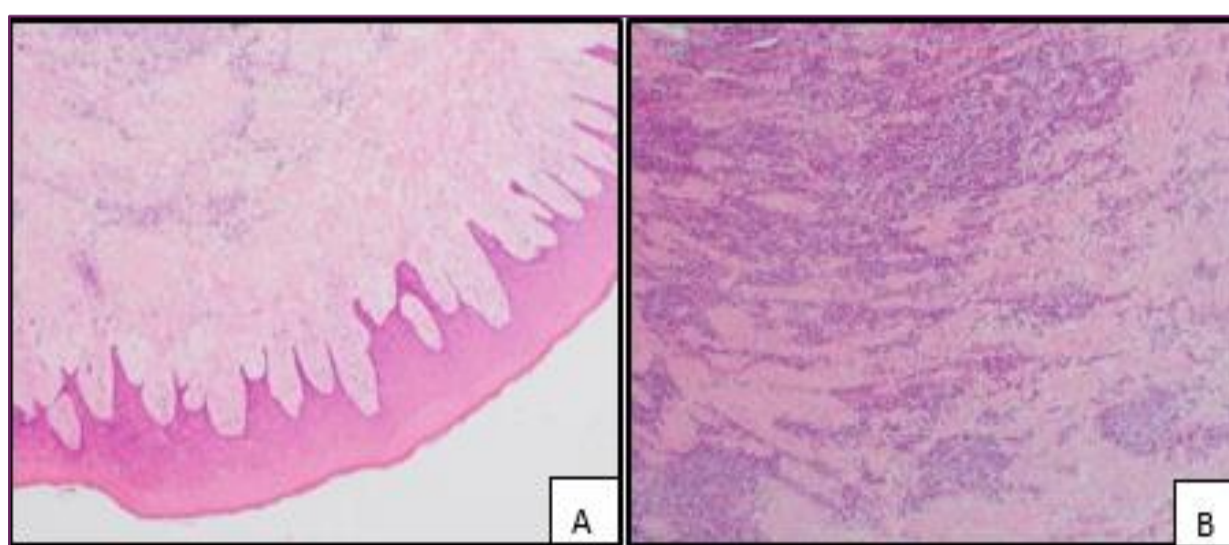
Test	Result	Reference Range	Unit
WBC	7.83	3.9-11	10 <sup>9</sup> /L
RBC	4.95	4.5-5.9	10 <sup>12</sup> /L
HGB	15.2	13.5-17.5	Gm/dl
HCT	46.7	46.7	%
MCV	94.3	80-100	FL
MCH	30.7	27-37	pg
MCHC	32.5	31-37	gm/dl
Platelet Count	216	150-400	10 <sup>9</sup> /L

<b>Neutrophils</b>	5.22	2.5-7	$10^9/L$
<b>Lymphocyte</b>	1.33	1.3-2.9	$10^9/L$
<b>Monocyte</b>	1.09	0.2-0.8	$10^9/L$
<b>Eosinophil</b>	0.15	0.04-0.4	$10^9/L$
<b>Basophil</b>	0.04	0-0.1	$10^9/L$

**Table 1.** Complete blood count of the patient. Monocytes were slightly elevated.

He presented to the surgical dental clinic complaining of a painless intra-oral mass that had been growing for over a year. On examination, an exophytic pedunculated soft mass related to the upper left alveolar mucosa was found. The mass was covered with normal mucosa, which measured 1cm. The lesion was attached to the keratinized gingiva and reported no discharge or bleeding. A tentative diagnosis of Drug-Induced Gingival Hyperplasia was suggested.

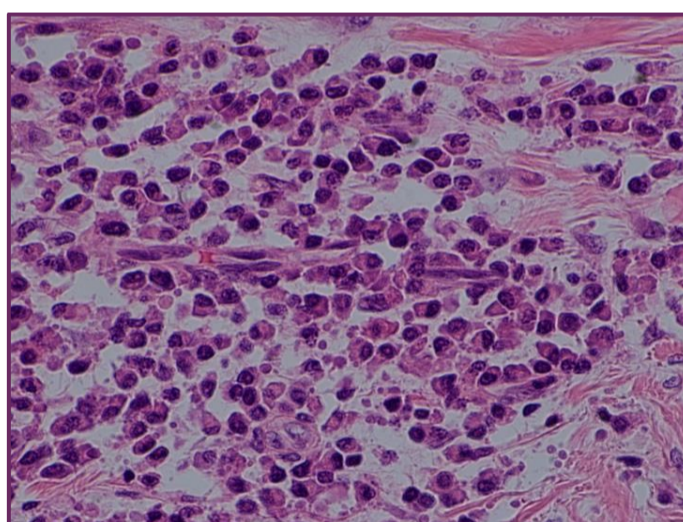
The whole lesion was excised in a dental setting under local anesthesia and sent for pathologic interpretation. It measured 0.8x0.4x0.3 cm. Sections from the lesion revealed polypoidal mucosa lined by acanthotic stratified squamous epithelium.



**Figure 1.1** A) Polypoidal lesion lined by stratified squamous epithelium. B) Plasma cells in clusters and focally as sheets

No nuclear atypia or mitotic figures were seen. Subepithelial stroma showed fibrocollagenous stroma with many plasma cells along with few blood

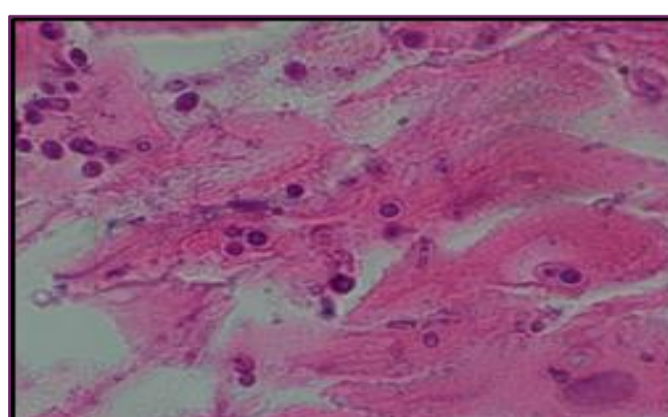
vessels. The small separate fragment shows few squamous cells with few budding forms of yeast.



**Figure 2.1** Plasma cells with a peripheral pushed nucleus.

The confined clinical presentation, along with the heavy plasma cells intermixed with minimal collagen in histology, eliminates Drug Induced

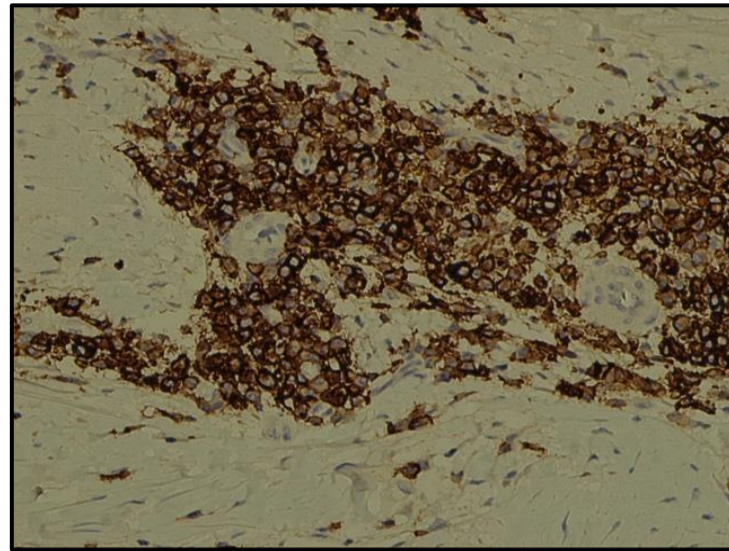
Gingival enlargement (in such cases, there would be an increase in dense collagen bundles along with fibroblasts)



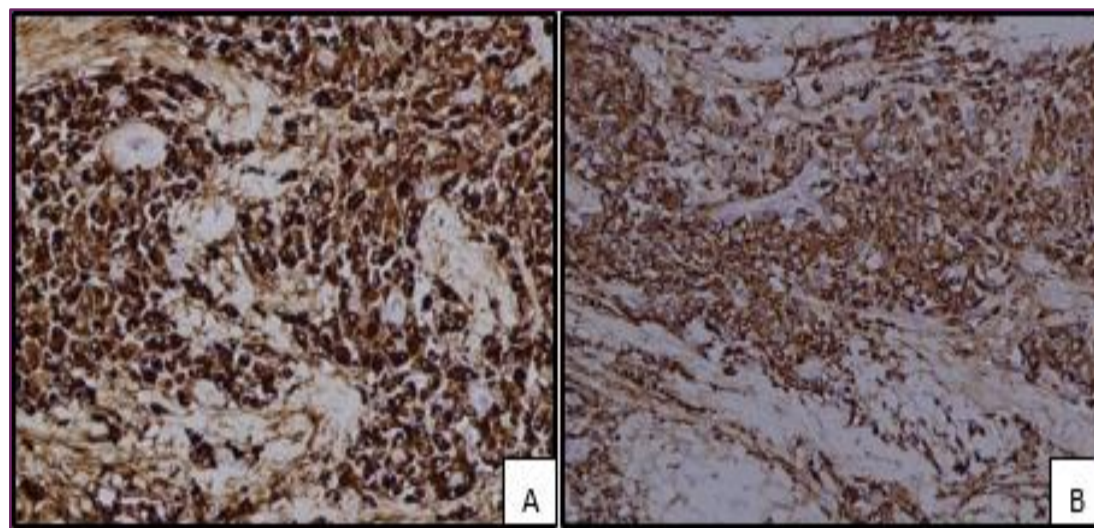


**Figure 3.1.** Budding forms of fungi. (highlighted by arrow)

Immunostaining was ordered for further investigation. Results obtained highlighted plasma cells (CD38 positive) and no restriction of Kappa and Lambda light chains.



**Figure 4.1.** CD38 Positive plasma cells.



**Figure 5.1.** A) Lambda light chain IHC positive. B) Kappa IHC positive.

The polyclonal nature of expressed plasma cells precludes the suspicion of tumors consisting of monoclonal plasma cells such as myeloma or plasmacytoma. Other lymphoid markers, such as CD 45, were negative. The

final diagnosis of Plasma Cell Granuloma was made. Upon follow-up, the lesion showed complete healing with no recurrence.

Diagnosis	Histology	Immunohistochemistry	others
<b>Plasma cell granuloma</b>	Polypoidal lesion covered by mucosa with sheets of plasma cells. Fibrocollagenous stroma.	CD38 + Kappa+ Lambda+	
<b>Plasmacytoma/Multiple myeloma</b>	Sheets of plasma cells.	CD38+ Kappa/lambda restricted	Serum/urine M protein Osteolytic bone lesions.
<b>Drug-induced gingival hyperplasia</b>	Pseudoepitheliomatous hyperplasia with dense collagen bundles, fibroblasts, and blood vessels.		Drug history
<b>Lymphoid neoplasms</b>	Sheets of lymphoid cells.	Lymphoid markers positive Kappa/lambda restricted	

<b>Giant cell epulis</b>	Mixed inflammatory cells, fibroangiomatic stroma, and giant cells.		
<b>Fibrous epulis</b>	Polypoidal lesion with increased collagenous stroma.		History of chronic irritation.

**Table 2:** List of Differential diagnosis

### Discussion

The majority of PCGs occur in the respiratory system. However, various unusual locations were reported, like the skin and CNS. As mentioned previously, only a few cases have been reported in the gingiva [8]. The exact cause remains a conflict; nevertheless, the hypothesis that suggests that a non-specific inflammation to an unknown antigen stimulates immunity and the proliferation of plasma cells is the most accepted explanation [1]. The inflammatory process can be a consequence of anything from minor trauma to malignancy [6].

Clinically, the lesion appears as a smooth, painless mass. As the lesion grows, it becomes larger and, therefore, ulcerated. At that time, the patient may complain of pain [7]. Herein, the lesion was only 1cm, and the patient reported no pain. PCGs do not have a unique clinical presentation. Thus, they can be easily misdiagnosed at first, particularly in patients with complex medical histories. A paper that reported a case of PCG in a hypertensive patient on Amlodipine had initially favored the diagnosis of Drug-Induced Gingival Overgrowth [5]. That was certainly in the top differentials in our patient who was on Nifedipine and immunosuppressors. Tacrolimus is another drug that is popular for its effect on the gingiva. A study reported overgrowth in patients who have been taking it for as little as three months

### Conclusion

PCGs of gingiva have been reported more frequently in the past few years. Unfortunately, the lack of interlapping variables in these patients' histories or backgrounds represents a challenge, as no exact causative factor can be

from transplant [9]. The patient in this paper had received his transplant five years ago. The impact of this drug cannot be ruled out.

In isolated advanced cases, bone destruction can be evident radiographically as an ill-defined radiolucency [13]. The role of histologic examination of these lesions is of great importance to exclude drug-induced lesions as well as neoplasms like multiple myeloma and plasmacytoma, especially in patients around their 60s [11]. Our patient is a 57-year-old, which puts him under the light.

Microscopically, plasma cells in sheets and aggregates are seen. They have an eccentric nucleus with a basophilic cytoplasm. These cells are arranged along with fibroblasts, histocytes, or other types of mesenchymal cells [2]. Several microorganisms have been associated with PCGs; these are mycobacteria, Epstein–Barr virus, actinomycetes, Nocardia, and mycoplasma [7]. Interestingly, in our case, budding yeast has been observed.

Management with surgical resection of the lesion is the treatment of choice with no recurrences in follow-up [14]. Along with the removal of the underlying cause if identified. The line of treatment with PCG is simple as opposed to plasmacytoma, which can require chemo or radiotherapy [13]. With our patient, excision of the nodule was found ideal.

proposed. Careful history-taking and diagnosis are vital in intraoral PCGs as these lesions may mimic many other oral lesions. This is achieved only through accurate histological analysis.

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