Case Report

Pyogenic granuloma of unusual size with alveolar resorption in a 75-year-old patient

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ABSTRACT

Pyogenic granuloma is an overzealously proliferative non-neoplastic lesion of connective tissue origin, found commonly in oral cavity and is secondary to chronic low grade local irritation, poor oral hygiene, and hormonal disturbances. The term is misnomer because a lesion is unrelated to infection. It is characterized by excessive and exuberant tissue repair response with varied inflammatory component. Since it is a benign lesion, choice of treatment is surgical excision with removal of underlying cause if any. This article aims at presenting a case of pyogenic granuloma in an extremely old patient which is unusual as it attained a very large size and also has caused mild resorption of underlying alveolar bone of mandible.

Key words: Inflammatory hyperplasia, proliferative lesions, pyogenic granuloma

Introduction

The term inflammatory hyperplasia is used to describe a large range of growths of oral cavity that histologically depicts inflammatory and granulomatous tissue.[1,2] Pyogenic granuloma (PG) is a kind of inflammatory hyperplasia. PG or granuloma pyogenicum is a common tumor like growth of the oral cavity or skin that is considered to be of non-neoplastic nature, arising commonly as a result of constant low grade trauma and poor oral hygiene and in few instances because of hormonal disturbances.[3,4] The term is absolute misnomer contrary to what the name implies, as the lesion does not contain pus and not strictly speaking a granuloma. It was first thought to be mycotic infection contracted from horses to human.[5] Clinical behavior and appearance of the lesion depend upon the total duration of the lesion. PGa of early state are usually highly vascular as they are consisting of hyperplastic granulation tissue, and the PG of long duration exhibit more collagenization.[6] To avoid possibility of a recurrence lesion must be excised down to the underlying periosteum and predisposing irritants must be removed.

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Case Report

A 75-year-old female patient reported to our maxillofacial surgical services with the chief complaint of intra-oral painless growth since one and half year. She also had a complaint of difficulty in closing the mouth since past 2–3 months.

The patient was relatively asymptomatic one and half year back, then the patient started noticing small painless growth inside the oral cavity in the right mandibular alveolar region, and growth gradually started increasing in size. Few months later she also started having fetid odor from the oral cavity. Because of large size of the growth the patient started having difficult in closing the mouth completely and inability to chew the food. The patient had a history of exfoliation of mandibular teeth and few maxillary teeth due to extensive mobility. During the dental education camp held in patient's village, the patient turned up for consultation. The patient was then referred to outpatient

department of maxillofacial surgery at Sardar Patel Institute of Dental and Medical Sciences, Lucknow. Past history and medical history did not reveal any relevant information. She was taking no medication and had no history of known drug allergy. Her general physical examination revealed no abnormality other than those related to the chief complaint except for moderate visual and hearing impairment.

On examination growth was slightly visible when the patient protruded the tongue. The patient was unable to close the mouth completely due to size of the lesion [Figure 1]. On intra-oral examination smooth surfaced, and lobulated growth of approximately 5 × 4 × 2 cm in size was present covering the alveolar ridge from the midline to molar region on completely edentulous alveolar ridge. Growth was pedunculated and was freely movable over peduncle which was present on the alveolar ridge of the premolar region. Surface of growth was smooth and showing normal color in certain areas whereas superior surface of the growth in certain area was covered by yellowish plaque like material. Growth was partially divided into two large lobules covering both buccal and lingual vestibules [Figure 2]. Mild indentation of maxillary teeth was observed on the superior surface of the growth. On palpation, growth was nontender, nonfluctuant, hard and fibrous in consistency without tendency for excessive bleeding upon probing under topical anesthesia. While the mandibular ridge was completely edentulous, the maxillary arch showed the presence of supra eruption of maxillary teeth from first molar to first molar on both sides. All maxillary teeth were periodontally compromised and showed grade 3 mobility. Tongue movements were unrestricted. There was no evidence of submandibular lymphadenopathy or paresthesia/anesthesia in the region of inferior alveolar nerve.

Provisional diagnosis of benign irritational hyperplasia/fibroma was made. Supra-eruption and the presence of sharp edges of maxillary teeth were considered responsible for providing low grade chronic irritation required for development o growths.

On radiographical examination of the mandibular ridge a moderate resorption of the alveolar ridge was observed which was biconcave in shape, which might be suggestive resorption due to pressure from the growing lesion. The presence of small root piece of about 3–4 mm was seen in the right mandibular posterior region [Figure 3].

Since the lesion was though unusual in size but having pedunculated base, treatment plan comprising of excisional biopsy of the growth and total teeth extraction was formulated and explained to the patient and her relatives and written and informed consent was procured. Routine hematological examination was advised and values were found to be within normal limits. An excisional biopsy was performed under local anesthesia [xylocaine 2% with adrenaline 1:200000]. After securing local anesthesia, growth was gently lifted to make the peduncle visible from both buccal and lingual. A sharp bone touching elliptical incision was made around the peduncle and on the alveolar ridge. With the help of periosteal elevator it was lifted along with the underlying periosteum from the bone surface and removed. Hemostasis was achieved and single layer closure was performed with the help of 3–0 silk suture. Excised specimen was preserved and sent to Dept. of oral and maxillofacial pathology for histopathological examination which confirmed it as Pyogenic granuloma [Figures 4 and 5]. The patient revisited after 7 days for suture removal [Figure 6]. The patient is currently under follow up without any signs of the recurrence of a lesion. The time of the total follow up till date is 1 year.

Discussion

The incidence of PG among other reactive lesions has been described as between 26.8% and 32%.[7,8] Although it has been reported to occur in almost all age groups, it occurs mainly between 11 and 40 years with the peak incidence in 30 years.[9] A study by Skinner *et al.* revealed that females are affected more than male with the female:male ratio of 3:2.[10]

Initial misconception about the etiology of PG was that it is a mycotic infection contracted from horses.[5] Later on without any scientific evidence it was labeled as a condition resulting from purulent change within benign intra oral tumors. Recently, angiogenesis-associated factors Tie2, angiopoietin-1, angiopoietin-2, ephrin B2, and Eph B4 have been discovered through immunochemistry from PG.[11] Now PG is a considered as a lesion developed as an exaggerated localized connective tissue reaction to minor injury or irritation. Sites of its occurrences are gingiva, lips, tongue, buccal mucosa, and palate. It is however well-circumscribed benign soft tissue tumor arising from connective tissue of skin and mucous membrane rather than of neoplastic nature.[12]

Few studies revealed that traumatic conditions occurring initially is the primary etiologic factor behind PG.[13,14] Almost 80% of patients while taking case history revealed initial trivial traumatic conditions before the appearance of the intra-oral lesion at the site.[4] Irritation of mucosa as a result of calculus, sharp margins of restoration, sharp cusps, and incisal edges leads to inflammation as a result of mechanical trauma.

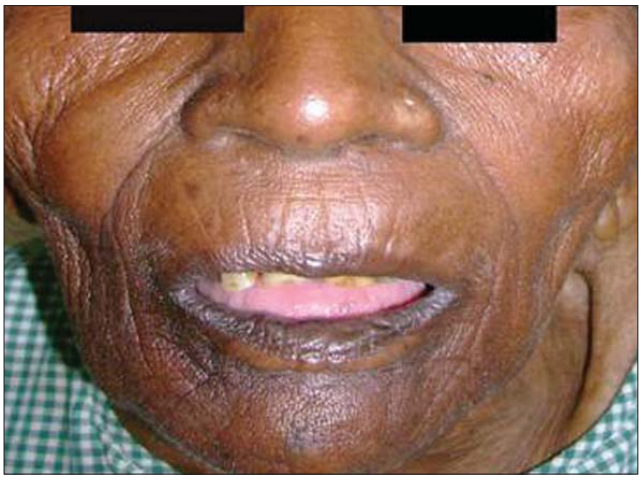

Figure 1: O.P.G. revealing bone loss from the alveolar ridge on the right side

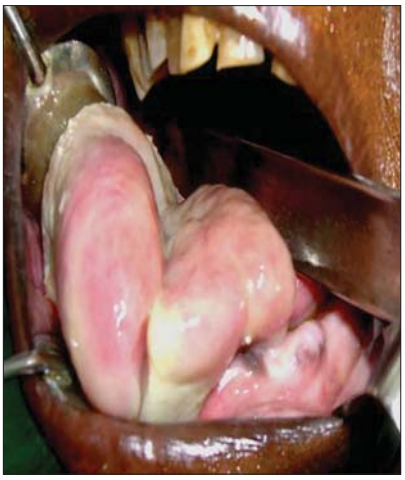

Figure 2: Excised specimen

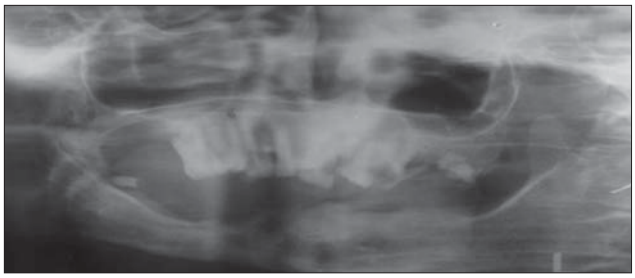

Figure 3: Post operative healing after 7 days

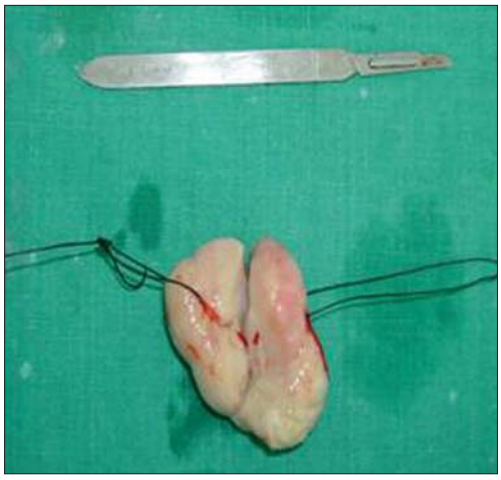

Figure 4: Photomicrograph

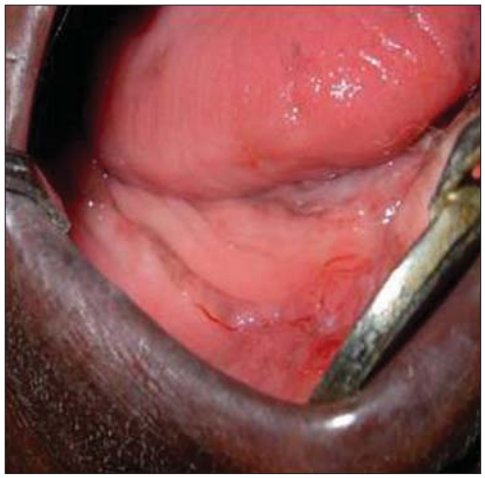

Figure 5: Intra oral growth

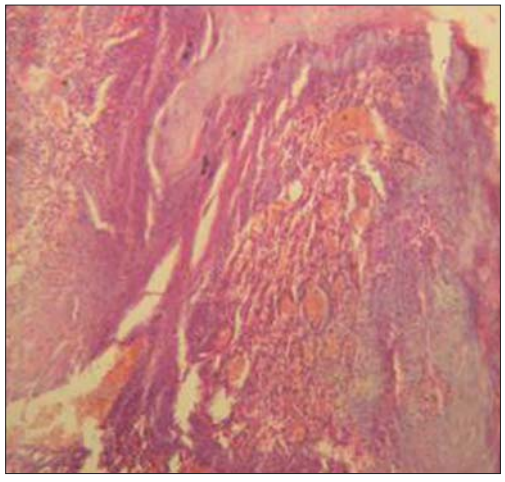

Figure 6: Extra oral view

Further irritation of already inflamed gingiva results in micro-ulceration through which underlying connective tissue is exposed to low virulent microorganism in the oral cavity. This evokes an exaggerated vascular and hyperplastic response in the connective tissue resulting in the formation of PG.[15]

PG clinically appears as smooth exophytic localized and well-circumscribed solitary growth with a sessile or pedunculated base, which is usually hemorrhagic and compressible The lesion can be smooth and divided into multiple lobules, sometimes may assume dumbbell-shaped mass. Surface may be ulcerated and friable and may be covered by yellow, fibrinous membrane and its color ranges from pink to red to purple depending upon the age of the lesion. Young PGs are highly vascular

in appearance and bleeds easily because they are composed predominately of hyperplastic granulation tissue in which capillaries are very prominent. Whereas older lesions tend to become more collagenized and pink.[6]

Rarely PG may cause significant bone loss, as reported by Goodman-Topper and Bimstein.[16]

PGa is a reactive lesions that develops rapidly, and ulcerates creating false positive picture of malignancy arising from connective tissue.[17] Differential diagnosis of PG can be parulis, peripheral giant cell granuloma, peripheral ossifying fibroma, hemangioma, peripheral fibroma, leiomyoma, hemangioendothelioma, hemangiopericytoma, bacillary angiomatosis, Kaposi's sarcoma, metastatic tumor, pregnancy tumor, and post-extraction granuloma.[18]

Definitive diagnosis of PG can be made from histopathological examination of the biopsied specimen. PGa histopathologically shows highly vascular proliferation that resembles like granulomatous tissue. The surface is usually ulcerated and replaced by fibrinopurulent membrane. A mixed population of inflammatory cell infiltrate comprising of neutrophils, plasma cells, and lymphocyte is evident. There are two histological types of PGs which differ in their histological features. The first type is characterized by proliferating blood vessels that are organized in lobular aggregates without specific changes superficially. This type was called as capillary lobular hemangioma (CLH) type. Second type non-LCH consists of highly vascular proliferation that resembles granulation tissue.[6]

As PG is a benign lesion, complete surgical excision along with removal of causative irritants is a treatment of choice. However, some other treatment protocols such as cryosurgery, laser surgery, and electrodessication have also been proposed. After complete excision, chances of recurrences are up to 16%.[19] Recurrence is believed to result from incomplete removal of the lesion, failure to remove etiological factors, or re-injury to the area.[20] Some recurrence manifest as multiple deep satellite nodules that surrounds the site of original lesions [Warner–Wilson Jones syndrome].[19] Powell *et al*. reported the use of Nd:YAG laser for the excision of a lesion because of lesser risk of bleeding as compared to other surgical techniques.[21] Moon *et al.* emphasized that the use of sodium tetradecyl sulfate (STS) multiple sclerotherapy successfully cleared the lesion in most of the patients without any major complication.[22] Ichimiya *et al.* reported a different approach using an injection of absolute ethanol in a patient with recurrence due to inadequate cryosurgery and concluded that this therapy is less invasive compared to surgical excision and appeared to be an alternate therapy for PG.[23] Parisi *et al*. used series of intralesional corticosteroid injection into the lesion for PG, particularly for a highly recurrent lesion.[24]

Conclusion

Although pyogenic granuloma is non-neoplastic growth in the oral cavity, proper diagnosis, prevention, management, and treatment of the lesion are very important. Pyogenic granuloma arises in response to various stimuli, such as local irritation, traumatic injury, sex hormones, so removal of causative irritant is a major line of treatment. Excisional surgery is the treatment of choice for pyogenic granuloma. But certain newer and alternative treatment modalities such as laser therapy, cryosurgery, injection of ethanol or corticosteroid or sodium tetradecyl sulfate have been reported. In spite of these treatments, the recurrence of the lesion is not so infrequent, so in some cases re-excision may be necessary.

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