ORIGINAL RESEARCH PAPER

ENT

KEY WORDS:

ORAL PYOGENIC GRANULOMA: A CLINICOPATHOLOGICAL ANALYSIS OF THIRTY-FIVE CASES

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INTRODUCTION:

Pyogenic granuloma (PG) is a benign non-neoplastic mucocutaneous lesion, with the term "pyogenic" being used erroneously since this condition doesn't produce purulent secretion[1] and it is not related to infection[2]. PG is also known as pregnancy granuloma or pregnancy tumor when occurring in pregnant women, or as vascular epulis, benign vascular tumor and hemangiomatous granuloma[3].

The term PG was introduced by Hartzell (1904). Hence it was also called as Crocker and Hartzell's disease[4].

Jaferzadeh, et al[2] (2006) defined PG as an inflammatory overgrowth of the oral mucosa caused by minor trauma or irritation. According to Neville, et al[5] (1998), these injuries might be caused in mouth by gingival inflammation due poor hygiene, trauma or local infection. Lin and Janniger[6] (2004) reported an association between dermatosis and the occurrence of PG.

It is believed that this lesion is formed as a result of an exaggerated localized connective tissue reaction to minor injury or underlying irritation. The irritating factors can be calculus, poor oral hygiene, non-specific infection, over hanging restorations, cheek biting, etc. As a result, the underlying fibrovascular connective tissue becomes hyperplastic, and there is proliferation of granulation tissue which leads to the formation of PG[7]. Factors such as inducible nitric oxide synthase, vascular endothelial growth factor, or connective tissue growth factor are known to be involved in angiogenesis and rapid growth of Pg[8].

Angeopolous[9] histologically described it as "hemangiomatous granuloma" due to the presence of numerous blood vessels and the inflammatory nature of the lesion. Cawson et al,[10] it as "granuloma telangiectacticum" due to the presence of numerous blood vessels seen in histological sections. They describe two form of PGs, The LCH and the non-LCH.

MATERIAL AND METHOD:

A retrospective review for thirty-five cases reported as PG was performed, which underwent biopsy during a period from September 2017 to October 2019 at the Department of Otorhinolaryngology, Hi-Tech Medical College and Hospital, Bhubaneswar. Data for the following parameters were recorded: age, gender, etiology, site and clinical presentation, variation in histology, treatment mode and recurrence.

RESULTS:

A total of thirty-five cases of PGs were evaluated.

Females showed a distinct predominance, the male: female ratio being 1:4.

Age of the patient ranged from 4 to 55 years. Maximum cases were found in second (31%) and third decades (40%) [Table 1].

Localized reactive hyperplastic lesions of gingiva are relatively common in biopsy services of oral pathology. Most commonly gingiva was involved (71%) followed by the lip (17%) and ventral surface of tongue (12%). Facial surface of gingiva involvement was more than lingual and palatal surface [Table 2].

The duration of lesions reported from 3 to 48 weeks and the maximum diameter of the lesion ranged from .1 to 2.5 cm and most of them were sessile (80%) and pedunculated (20%).lesions manifested as inflammatory overgrowths which were smooth non tender and non-fluctuant (86%), nodular swelling with normal mucosal color (77%) and ulceration (3%) [Table 3].

History of trauma or chronic irritation was present in 83% cases, hormonal factors (pregnancy tumor) in 11% cases [Table 4].

Histopathologically, there was almost similar reporting in all cases, consisting of marked vascular proliferation among immature fibroblastic connective tissue, granulation tissue, and chronic inflammatory infiltrate [Fig-1].

Table 1: Age groups of oral pyogenic granuloma

Age range	Number of cases (%)
0-10	1 (3)
10-20	11 (31)
20-30	14 (40)
30-40	5 (14)
40-50	3 (9)
50-60	1 (3)

Table 2: Sites affected by oral pyogenic granulomas

Site	Number of cases
Gingiva	25 (71)
Lip	6 (17)
Tongue	4 (12)

Table 3: Distribution of size, base attachment surface, and colour of lesions

Clinical features	Characteristics	PG(n=35)
Size(cm)	>1	4
	<1	31
Base Attachment	Sessile	28
	Pedunculated	7
Surface	Smooth	30
	Ulcerated	1
	Polypoid	4
Colour	Pinkish White	27
	Red	8

Table 4: Etiology for oral pyogenic granuloma

Etiology	Number of Cases (%)
History of trauma or irritation	29 (83)
Hormonal/Pregnancy induced	4 (11)
Miscellaneous	2 (6)

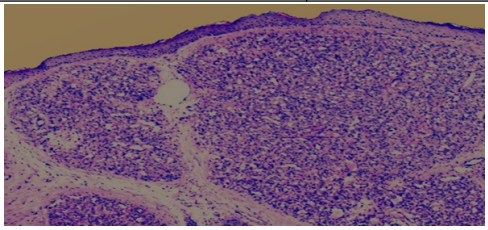

Figure 1: Microscopic aspect showing an oral mucosa consisting of continuous Para keratinized, stratified, pavement epithelium covered with a serofibrinous membrane. The underlying granulation tissue was rich in

blood vessel, fibroblast and macrophage.

DISCUSSION:

PG is a kind of inflammatory hyperplasia and also termed as granuloma pyogenicum. PG is a misnomer because the lesion does not contain pus[1] and is not a granuloma[2] also. Histologically, it is described by Angelopoulos as hemangiomatous granuloma due to increase in blood vessels[9] and also termed as granuloma telangietacticum according to Cawson *et al*[10]. It was also named a Crocker and Hartzell's disease[4]. There are 2 forms of PG - the lobular capillary hemangioma (LCH) and the non-LCH[10].

PG is caused by a known stimulant such as calculus or foreign material in the gingival crevice resulting in a proliferation of connective tissue[11]. Ainamol suggested that routine tooth brushing habit caused repeated trauma to gingival, resulting in these lesions[12]. Furthermore, release of variety of endogenous substances and angiogenic factors, trauma to deciduous teeth[13], aberrant tooth development[14], occlusal interferences[15], drugs such as cyclosporine[16] and selection of wrong healing cap for implants are some of the precipitating factors for Pg[17].

Oral PGs occur in all age groups, children to older adults, but frequently seen in females in the second decade due to increased levels of hormones[18].

It appears as an elevated sessile or pedunculated growth covered with red haemorrhagic and erythematous papules and show ulcerations and is covered by a fibrinous membrane[5,11]. The colour varies from red, purple pink, depending on the vascularity of the growth[19]. Gingiva is affected primarily, especially the marginal gingiva and common in maxillary than mandibular gingiva. Anterior areas are more frequently affected than posterior areas. All these lesions are more common on the facial than the lingual aspect[5]. Clinically, the lesion can be slow-growing, asymptomatic and painless, but it may also grow rapidly sometimes[11]. Radiographic findings are usually absent[20], however, some long-standing gingival PGs can cause localized alveolar bone resorption[9].

Differential diagnosis includes peripheral giant cell granuloma, peripheral ossifying fibroma, metastatic cancer, haemangioma, pregnancy tumour, hyperplastic gingival inflammation, Kaposi's sarcoma, bacillary angiomatosis, angiosarcoma, and non-Hodgkin's lymphoma. Peripheral giant cell granuloma is clinically similar to PG, but bone resorption in radiograph and appearance of the multinucleated giant cell are differentiating features. Also, fibroma can be distinguished by the consistency, texture, and the lighter colour. Metastatic tumours, even though clinically resembles PG, the microscopic appearance resemble as the tumour of origin. Haemangioma is a developmental disorder and is most commonly seen on the tongue. It can be multinodular, bluish red and can be diagnosed by a chairside procedure called diascopy. Kaposi's sarcoma and bacillary angiomatosis can be differentiated histopathologically and are also AIDS related. Pregnancy tumour occurs towards the end of pregnancy, and the tendency for this lesion to shrink after delivery indicates the definite role in aetiology of lesions. Also, pregnancy tumour is usually confined to the interdental papilla. PG can be distinguishable from angiosarcoma by its lobular growth pattern, well-formed vessels and cytologically bland endothelial cells. Clinical appearance of gingival non-Hodgkin's lymphoma varies but is usually found to be an asymptomatic gingival enlargement or mass resembling a PG.

Histopathologically, it can be classified as an LCH and non-LCH[10]. LCH has proliferating blood vessels in lobular aggregates, no specific changes such as edema and capillary dilatation. Non-LCH type consists of vascular core resembling granulation tissue with foci of fibrous tissue. The lobular area of LCH type has a greater number of blood vessels. Oral PGs are mainly LCH type. Natural course of the lesion can be in three phases of development as cellular phase, vascular phase and phase of involution.

Treatment includes surgical excision of the lesion with the removal of irritants recommended for small painless lesions. Excision of gingival lesions up to periosteum with thorough scaling and root planning of adjacent teeth to remove all visible sources of irritation. Various other treatment modalities include Nd: Yttrium-aluminium-garnet lasers, carbon dioxide lasers, flash lamp, pulse dye laser, cryosurgery, sodium tetradecyl sulphate sclerotherapy, and use of intralesional steroids have been proposed by clinicians. Treatment of oral PG during pregnancy would depend on preventive measures such as careful oral hygiene, removal of dental plaque, and use of soft toothbrush. In some cases, shrinkage of the lesion after pregnancy may make surgical treatment unnecessary.

Incomplete excisions, failure of removal of etiological factors contribute to the recurrence of these lesions. A recurrence rate of 16% and also a case of multiple deep satellite lesions surrounding the original excised lesion in a case of Warner Wilson James syndrome have been reported[21]. A need for regular follow-up is also emphasized because of higher recurrence rate, especially in the gingiva.

CONCLUSION:

PGs are benign tumor like lesions commonly encountered, but when presented late, especially when infected, they can pose diagnostic challenges by mimicking more sinister lesion, due to their remarkably large size[8]. Knowledge of their clinical features and demographics is essential for all health personnel who encounter this lesion. We presented a study carried out on patients with oral PGs to evaluate its clinico pathological features and to determine the prevalence of the same in relation to age, sex, site, and various clinical variation. We call attention to the uncommon mucocutaneous labial location of PGs and to the fact that surgical excision is the safest method for diagnosis and treatment of PGs of lip, even when involving mucosa and skin.

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