

## **PYOGENIC GRANULOMA**

### **Definition**

Pyogenic granuloma (PG) is a common reactive neoformation of the oral cavity, which is composed of granulation tissue and develops in response to local irritation or trauma. Various different names have been given to this entity, reflecting, in part, mistaken concepts about its aetiopathogenesis:

- Botryomycosis hominis
- Botryomycoma
- Telangiectatic granuloma
- Benign pedunculated granuloma
- Pseudobotryomycosis
- Fibroangioma
- Croker and Hartzell disease
- Septic granuloma
- Haemangiomatous granuloma
- Lobular capillary haemangioma
- Eruption capillary haemangioma

The most widely used term is PG, although it does not adequately describe the lesion's characteristics. The term "pyogenic" implies pus production related to an infectious aetiology; however, no pus-producing microorganisms are associated with PG.

Moreover, the lesion is not a true "granuloma" (i.e. a specific type of persistent chronic inflammation).

A lesion clinically and microscopically identical to PG may develop from the gingivae of pregnant women. Different names have been used to describe this entity, including pregnancy tumour, epulis, or granuloma, and granuloma gravidarum. Nonetheless, identical cases occur in patients with hormonal changes associated with puberty, menopause, or use of anti-contraceptive medication. Thus, the correct name for this entity is hormonal tumour (HT). Between 0.5% and 5% of pregnant females develop pathological growths in the gums, which are diagnosed as HTs.

### **Epidemiology**

Pyogenic granuloma is a relatively common oral lesion, possibly affecting 1% of the general population with a predilection for the female sex. PGs may occur at any age with a predilection for young adults, most of the patients being in the third or fourth decade of life.

## **Clinical presentation**

The predominant location for PGs is the gingiva, the labial maxillary gingiva being the most frequently affected site, especially anteriorly. Any other oral location may be affected, including the lips, buccal mucosa, and tongue.

PGs present clinically as exophytic, not well-delimited, widely based or pedunculated lesions. The surface is smooth or rough and deep red in colour and the consistency is softer than the rest of the mucosa. Ulceration is a frequent finding, sometimes covered by a fibrinous pseudomembrane, which imparts a whitish appearance. This white necrotic material may resemble pus, but, in the absence of a bacterial infection, no actual pus is produced. Depending on the duration and degree of fibrosis, the lesion may be firmer and paler. The size of PG is usually between 1-3 cm, although much larger lesions are not uncommon.

The majority of PGs are asymptomatic; however, lesions with an intense inflammatory component can be rather painful. Bleeding following mild trauma, or even spontaneously, is frequent. When PGs occur in the gingivae, they are also complicated with specific periodontal alterations (bleeding, periodontal pocket formation, gingival retraction, and tooth mobility).

## **Aetiopathogenesis**

PG is not an infectious but a reactive lesion, due to local irritating factors. Therefore, PG is included in the large group of inflammatory hyperplasias (IHs): neoformations due to increase in the number of constituent cells and not genuine tumours in the sense that their excessive growth is not autonomous but depends on a continuous stimulus of traumatic nature.

The traumatic agents responsible for gingival PG are gingival and periodontal irritants, including plaque accumulation, supra- and infra-gingival calculus, overhanging margins of crowns and fillings, implantation of foreign material etc. Other traumatic agents, such as biting, are responsible for PG developing in other parts of the mouth. Healing extraction sites may also give rise to a PG, sometimes termed epulis granulomatosa. Independent of their exact nature, the various chronic traumatic insults induce inflammation, followed by repair with production of excessive granulation tissue. Some investigators have related PG to prolonged treatment such as with cortisone, oral contraceptives, and diabetic medications, or even with allogeneic bone marrow transplantation.

HT is associated with hormonal changes, most often associated with pregnancy, but also occurring with puberty, menopause, or contraceptive treatment. The increase of oestrogens and progesterone produces an increase in vascularisation, capillary

proliferation, and vascular permeability. These changes lead to a greater susceptibility of the gums against local irritants, such as abundant plaque and calculus, therefore rendering the patients prone to inflammatory and reactive processes, including HT.

### **Diagnosis**

A definitive diagnosis of PG is reached through evaluation of clinical and histopathological characteristics. Microscopically, PGs appear covered by a stratified squamous epithelium, which is atrophic or ulcerated in almost 100% of cases. Ulceration is common. The underlying connective tissue is oedematous and occupied by exuberant granulation tissue featuring a large proliferation of small capillaries. A mixed inflammatory infiltrate, mainly composed of polymorphonuclear leukocytes, and to a lesser extent, lymphocytes, is evident. These pathological characteristics, along with the clinical features, make these lesions very similar to exophytic capillary haemangiomas. For this reason, on occasions, it is convenient to perform an aspiration puncture with a fine needle before extirpation to confirm the clinical impression of a vascular lesion and to avoid problems of haemorrhage.

The degree of fibrosis is minimal, although older lesions become progressively more fibrosed. In untreated, chronically irritated lesions, there is a progressive maturation of the lesion with gradual replacement by collagenous, fibrous connective tissue. The final stages of a PG may be identical to a fibrous hyperplasia.

PG in a gingival location should be differentiated from other reactive gingival lesions, such as peripheral giant cell granuloma (PGCG) and peripheral ossifying fibroma (POF). Although clinical differences from typical PG do exist (e.g. PGCG is usually associated with bluish-purple hue and may cause a “cupping” resorption of the underlying bone, while POF is pinker and often non-ulcerated), the final diagnosis relies on histopathological examination of the excised specimen.

### **Treatment**

Surgical excision of PGs is the treatment of choice. Excision should also include removal of the base of the lesion, with extension down to periosteum, and curettage. Extraction of associated teeth is rarely necessary. In small incipient lesions, extirpation of the lesion and electric-coagulation of the base is recommended. Nowadays, the use of different types of LASER extirpation is available and particularly useful for control of bleeding in PGs and other haemorrhagic lesions.

Elimination of the responsible chronic irritating factor is always of paramount importance. Hence, the suggested protocol of action for PG includes initial elimination of the traumatic factors with subsequent surgical excision. If the lesion is small, reddish

and free of fibrosis, the elimination of the traumatic factors may be sufficient for its own self extinction. Even if total resolution is not achieved, PG will likely lose its inflammatory and granulation tissue component, thus being smaller, less haemorrhagic and less prone to surgical complications.

Because of the risk of complications and a higher risk of recurrence for HT removed during pregnancy, surgical excision should be deferred with the exception of cases featuring excessive haemorrhage and ulceration, or marked functional and aesthetic problems. If surgery is necessary, it is preferably done after the second trimester and, due to the high content of vascular tissue, LASER treatment is advisable.

All obtained specimens should be evaluated microscopically to confirm the diagnosis.

### **Prognosis and complication**

Elimination of the causative traumatic factors followed by complete surgical excision of the lesion constitutes the basis for definitive treatment and prevention of recurrences.

However, according to some authors, recurrences occur in almost 20% of cases.

During surgical removal, special attention should be paid to complete removal of the base of the lesion to avoid possible recurrences.

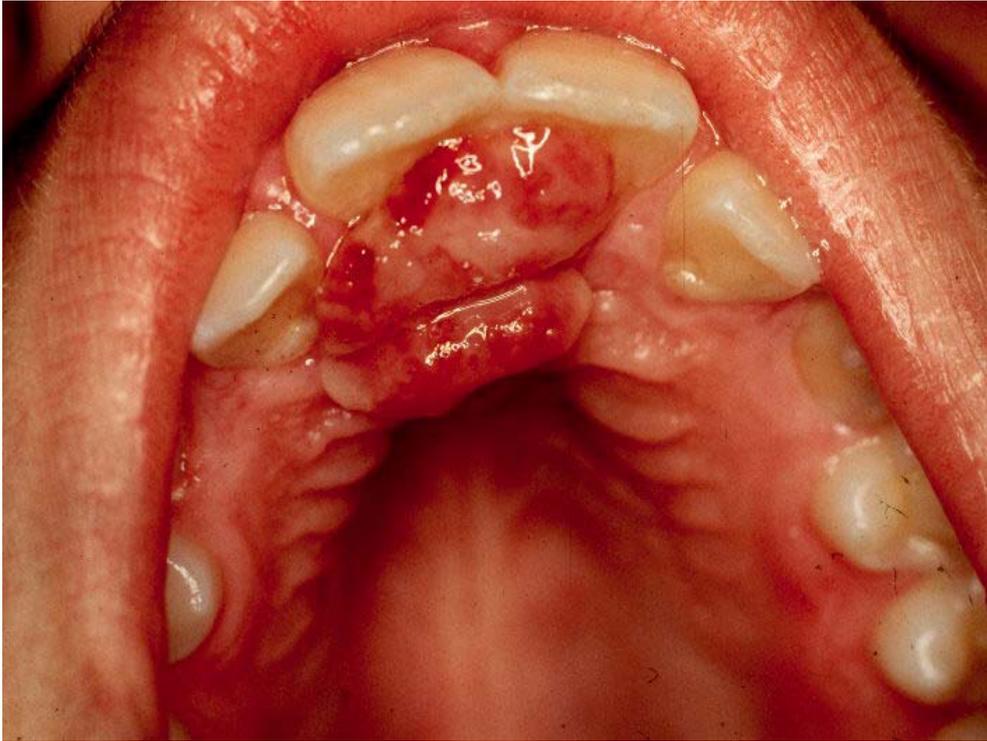
Complications of treatment include haemorrhage, which can be prevented through previous reduction of the amount of inflamed granulation tissue by means of elimination of the irritating factors. Use of LASER allows control of haemorrhage through induction of coagulation. On the other hand, extirpation of anterior large lesions can produce a wide range of gingival defects, which pose important aesthetic problems. In these cases, a more laborious and meticulous surgical approach will contribute to a cosmetically-acceptable result.

There is a general consensus that HT regresses after childbirth, although it rarely disappears completely. In our experience, the HTs persisted one month after childbirth, although smaller in size. Surgical excision and periodontal management were carried out without subsequent recurrences.

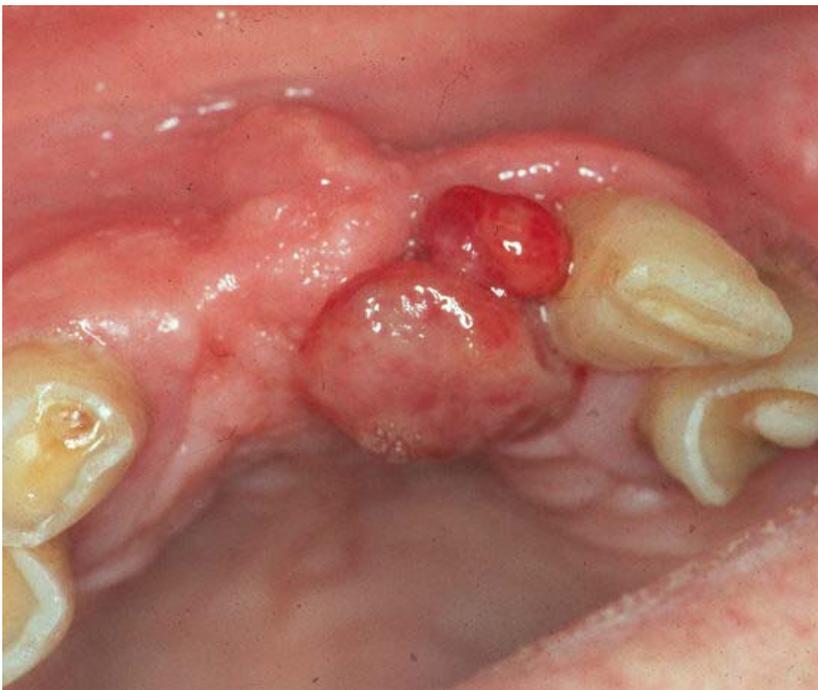
### **Prevention**

Prevention of recurrent or newly-developed lesions of PG entails complete removal of the causative traumatic insult. For gingival lesions, implementation of meticulous oral hygiene is of paramount significance. In cases that dental restorations and appliances, or tooth and periodontal abnormalities are implicated, these factors should be corrected or eliminated.

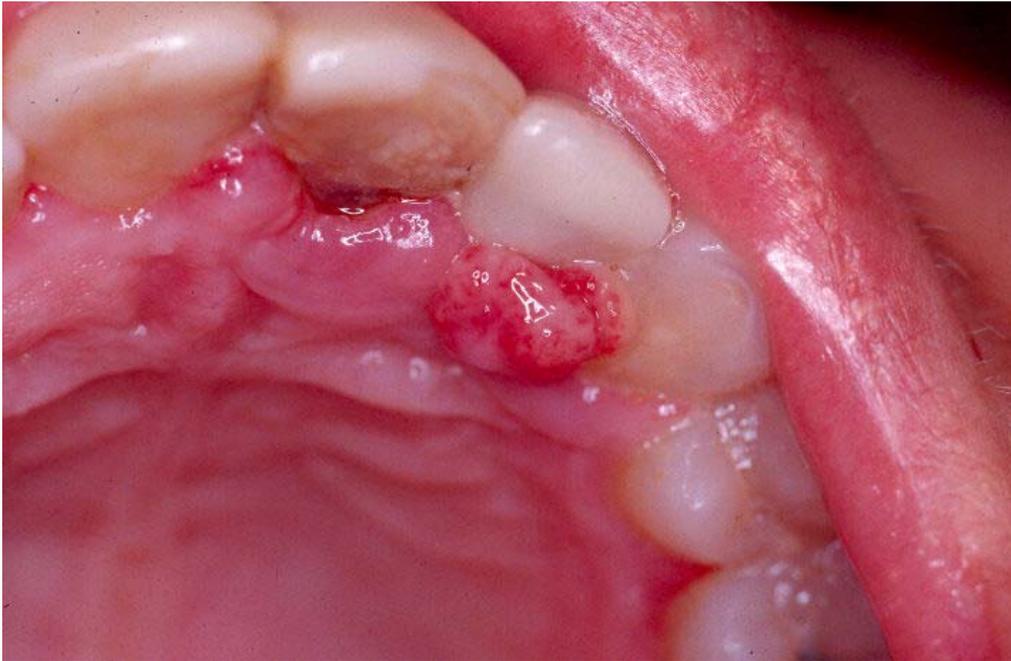
The adoption of preventive measures during pregnancy, such as better oral hygiene, will reduce the risk of pregnancy-associated HT.



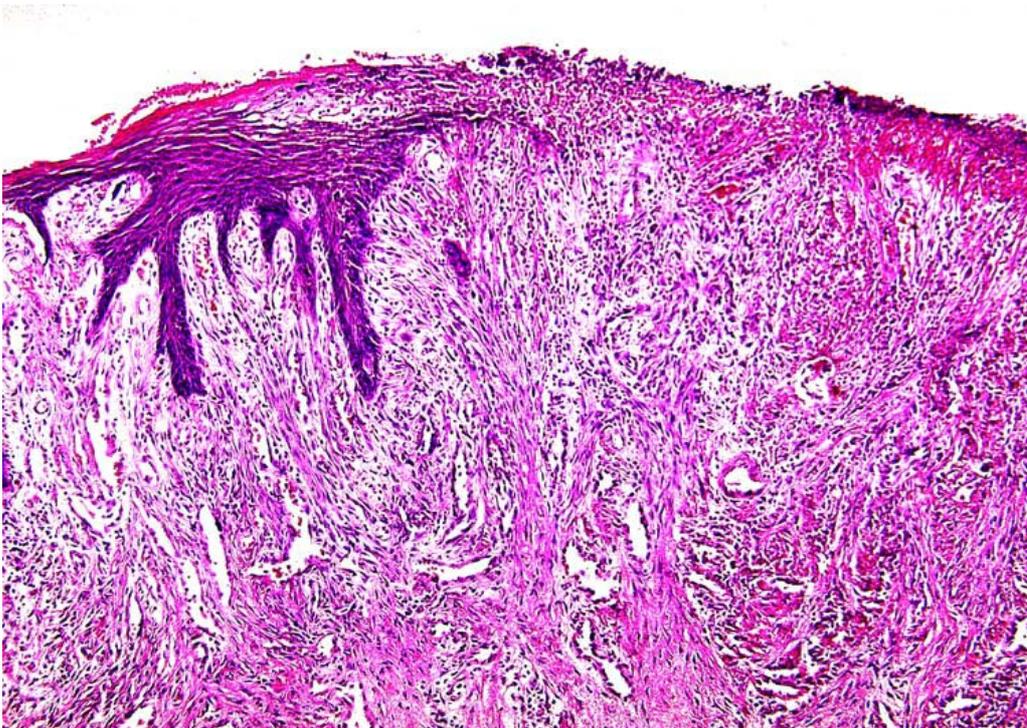
**Figure 1.** Pyogenic granuloma (clinical)



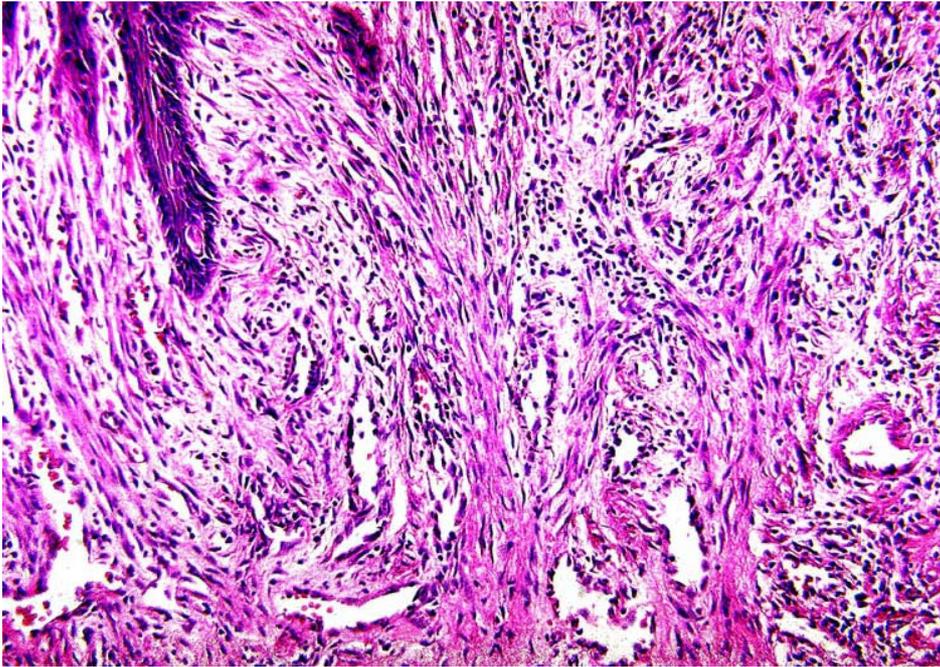
**Figure 2.** Pyogenic granuloma (clinical)



**Figure 3.** Hormonal tumor (clinical)



**Figure 4.** Pyogenic granuloma (histopathological, H & E 100x)



**Figure 5.** Pyogenic granuloma (histopathological, H & E 200x)

### **Further reading**

- 1 Patrice SJ, Wiss K, Mulliken JB. Pyogenic granuloma (lobular capillary hemangioma) a clinic pathological study of 178 cases. *Pediatr Dermatol* 1991; 8:267-76.
- 2 Blanco Carrión A, Suárez Cunqueiro M, Blanco Carrión J, Alvarez Velasco N, Gándara Rey JM. Hiperplasias inflamatorias de la cavidad oral. Estudio clínico e histológico de 100 casos (II). Características específicas de cada lesión. *Av Odontoestomatol* 1999; 15:563-76.
- 3 Eversole LR, Robin S. Reactive lesions of the gingiva. *J Oral Pathol* 1972; 1:30-38.
- 4 Priddy RW. Inflammatory hyperplasias of the oral mucosa. *J Can Dent Assoc* 1992; 58:311-5, 319-21.
- 5 Angelopoulos AP. Pyogenic granuloma of the oral cavity: statistical analysis of its clinical features. *Oral Surg* 1971; 29:840-47.