Infected Granulation Tissue Transforming into Pyogenic Granuloma and Peripheral Ossifying Fibroma: A Case Report

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ABSTRACT

Objective: Granulation tissue represents a subtle presentation of Reactive Hyperplastic Lesions (RHLs). With persistent chronic irritation, granulation tissue may progress into matured RHLs (pyogenic granuloma, peripheral ossifying fibroma and peripheral giant cell granuloma) which are similar clinically but different histopathologically. While some believe they are in continuum, others take each as separate entity. Treatment is similar in all RHLs but not without recurrence if treatment is not properly done. We believe they exist in a continuum based on the case findings and therefore present a case of infected granulation tissue transforming through PG to POF. The objective of study was to report a case of infected granulation tissue transitioning through pyogenic granuloma into peripheral ossifying fibroma.

Keywords: Reactive Hyperplastic Lesions (RHLs), Pyogenic Granuloma (PG), Peripheral ossifying fibroma (POF).

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CLINICAL IMPLICATIONS

Reactive hyperplastic lesions may exist as a continuum rather than discrete lesions. They may clinically appear similar but only histopathological evaluation can help distinguish one from another. RHLs should be managed aggressively and good oral hygiene measures instituted with constant follow-up to avoid recurrence.

INTRODUCTION

The oral cavity constitutes an important part of the human body, serving the essential function of mastication asides speech. Because of this, it is almost always subjected to trauma from masticatory forces. This, together with other injurious agents such as calculus, fractured teeth, overhanging restorations and ill-fitting dentures can cause chronic irritation to the soft tissues/mucosae of the oral cavity especially gingivae leading to the formation of gingival epulis or swelling.[1] Truschnegg et al.[2] grouped this swelling histopathologically into seven which includes hyperplastic squamous epithelium, granulation tissue, peripheral odontogenic fibroma, pyogenic granuloma (PG), fibroma, giant cell lesion, peripheral ossifying fibroma (POF)[2] with the last four entities classified as Reactive Hyperplastic Lesions (RHLs) by Kfir et al.[3] Distinguishing one member of this group from another may be challenging due to their similar clinical and radiologic presentations, therefore, histopathologic investigation is essential in arriving at a correct diagnosis. While some researchers believe this group of lesions may progress unidirectionally from one type to another,[4,5] others are of the opinion that each lesion is a separate entity on its own.[6] Thus this article aims to dispel the belief that they exist as separate entities but rather to further support that the RHLs exist in a continuum by reporting our case.

CASE REPORT

A 14-year-old male student reported at the Periodontology clinic on account of a recurrent gingival growth on the lower right quadrant interdentally between 6 and 7, and could not account for the time the swelling re-occurred. The swelling was firm in consistency with areas of ulceration marked by the upper teeth (Figure 1). There was a history of a similar growth about 2 years previously (Figure 2), which was excised at the same unit with histopathological diagnosis of infected granulation tissue. The gingival growth was excised and the site was aggressively curetted after adequate patient preparation to prevent recurrence. The excised lesion was sent for histopathologic examination.

Microscopically, the lesion is composed of hyperplastic stratified squamous epithelium that is infiltrated by mixed inflammatory cells. The underlying stroma is composed of proliferating vascular channels with sparse presence of chronic inflammatory cells (Figure 3). In another area within the stroma are foci of calcific-like materials resembling cementum and bony trabeculae (Figure 4a and 4b). Based on this microscopy, a histopathologic diagnosis of peripheral ossifying fibroma coexisting with non-lobular capillary hemangioma was made. A 24-month post-surgical follow up of the patient showed no evidence of recurrence (Figure 5).

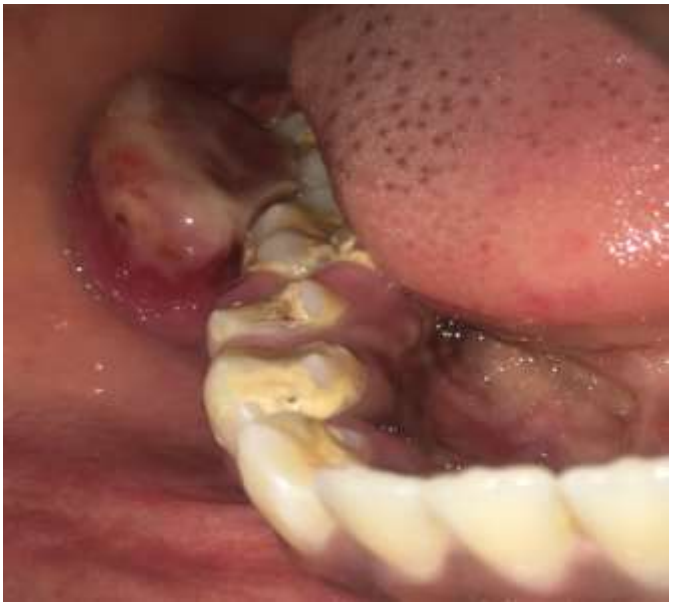

Figure 1: Clinical picture of recurrent lesion.

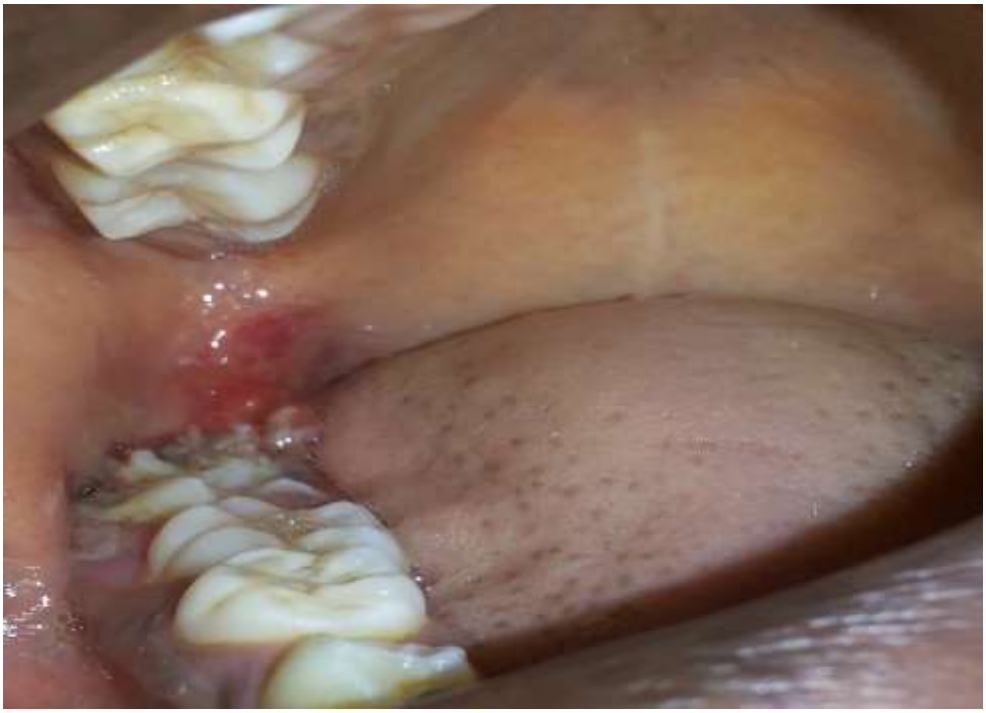

Figure 2: Clinical picture of Infected granulation tissue.

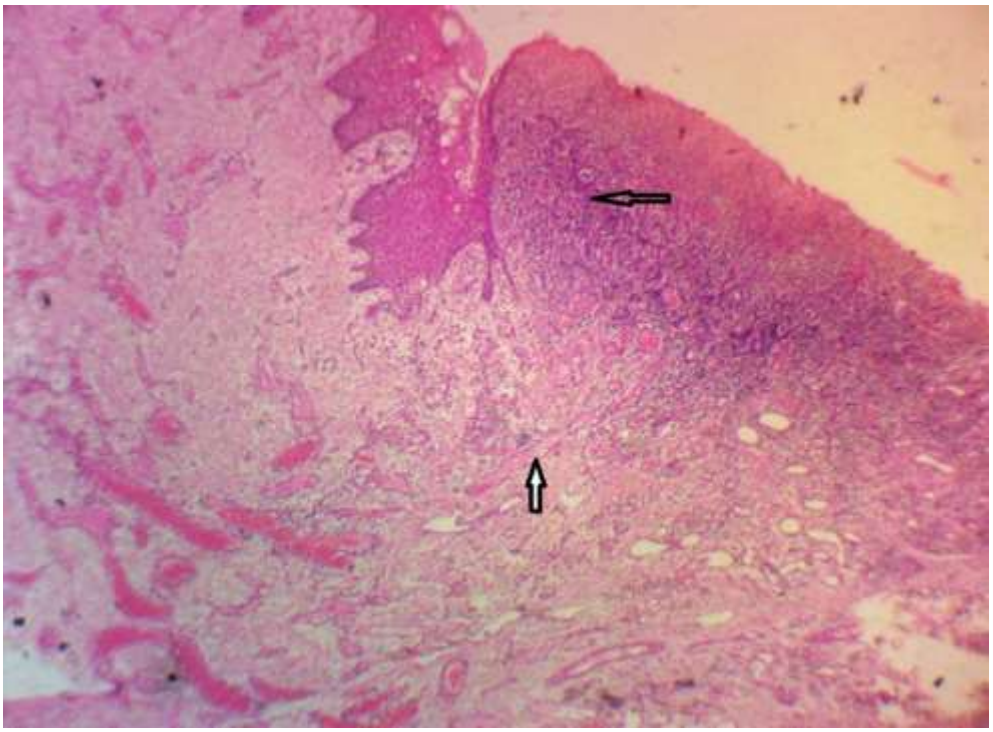

Figure 3: Photomicrograph of pyogenic granuloma X100 mag. shows hyperplastic epithelium infiltrated by mixed inflammatory cells with proliferating vascular channels in the stroma.
Note: black arrow indicates mixed inflammatory cells and white arrow indicates proliferating vascular channel.

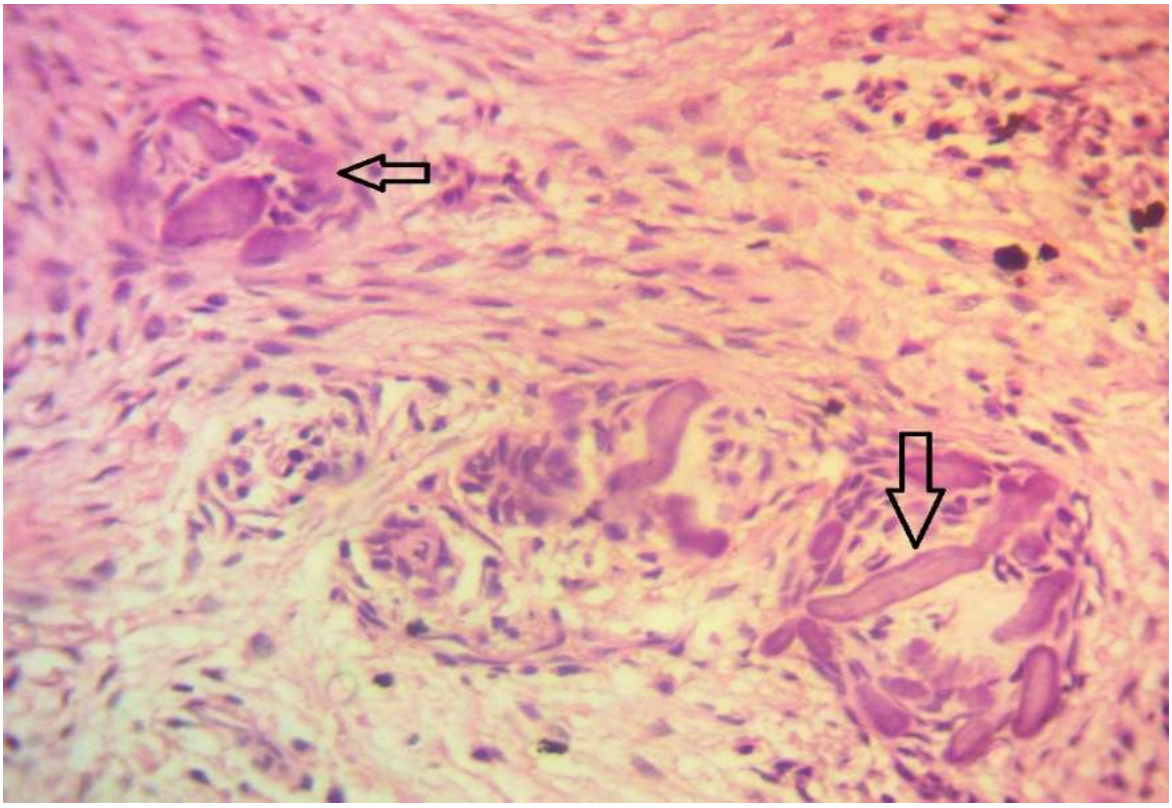

Figure 4a: Photomicrograph of POF X100 mag. Note the foci of calcific-like material as shown with the arrows

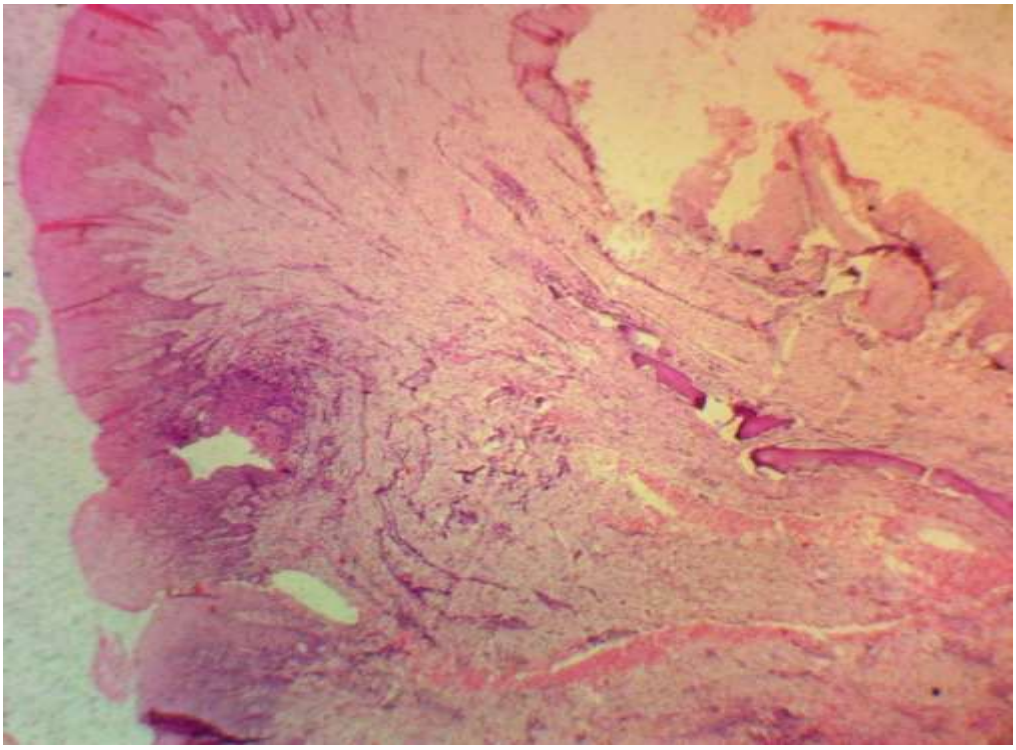

Figure 4b: photomicrograph of POF X40 mag. Note the bony trabeculae on the right

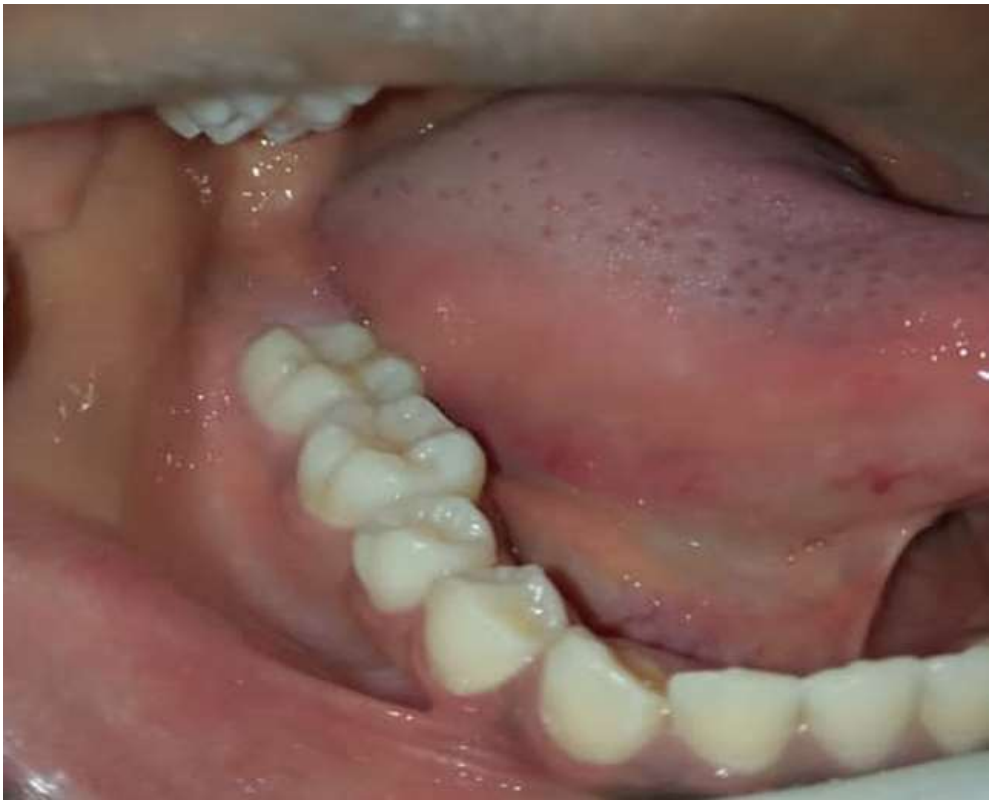

Figure 5: Clinical picture of same site 24months post-surgical excision.

DISCUSSION

Reactive hyperplastic lesions are a non-neoplastic group of lesions that result following exaggerated response of oral soft tissues to chronic irritation.[7] They include PG, POF, peripheral giant cell granuloma (PGCG) and fibrous hyperplasia.[3] They present as swellings which could affect any part of the oral mucosa but when found on the gingivae, they are called "epulides".[8] Apart from oral mucosa, PG could be found on the skin and mucous membrane.[9] Histopathologically, they consist of proliferating fibroblasts, endothelial vascular channels and chronic inflammatory cells, altogether termed granulation tissue; with or without calcific like materials, bone trabeculae and giant cells.

The earliest presentation of RHLs is granulation tissue which maybe unnoticeable except when associated with pain or swelling. Granulation tissue is formed in an attempt at repair of damaged tissues following chronic irritation. This may progress over time to form any of the aforementioned RHLs if exposure to stimuli persists. Most patients usually would present when the lesion has transformed into a painless, slow growing swelling for management. Our case showed the transition of infected granulation tissue to PG and POF.

PG is the next transition phase of granulation tissue. The term PG is a misnomer as this lesion is neither of infectious origin nor related to granuloma formation. There are two types of PG based on their histopathologic presentation which are lobular capillary heamangioma (LCH) and non-lobular (non-LCH).[10] LCHs are composed of vascular channels arranged in lobules separated by fibromyxoid stroma[11] while non-LCH presents with haphazard arrangement of vascular channels.[8, 12] Ours showed non-LCH pattern. Though our own case occurred in a male teenager, PG is commoner in females in the second decade of life. Surge in female sex hormones such as oestrogen and progesterone during puberty and pregnancy have been documented to have a role to play in the pathogenesis of PG. Oestrogen enhances formation of granulation tissue needed for PG formation through production of Granulocyte-Macrophage-Colony Stimulating Factor (GM-CSF) in keratinocytes, basic Fibroblast Growth Factor (bFGF) and Transforming Growth factor beta1 (TGF-beta1) in fibroblasts and Vascular Endothelial Growth Factor in macrophages (VEGF).[13] Progesterone on the other hand prevents acute inflammatory infiltration so as to allow it run a chronic course.[14] Clinically, PG presents as a sessile or pedunculated swelling that

bleeds readily.[7]It may also bleed less and become more firm or fibrotic as it matures. It usually would not cause resorption of bone except when large due to pressure effect. Management entails surgical curettage using scalpel, Nd-YAG or CO_2 laser, cryosurgery as well as initiating sclerosis of the lesion using sodium tetradecyl sulfate or intralesional steroids.[15, 16]

POF is a discrete, sessile or pedunculated, erythematous to pinkish swelling solely found on the gingivae.[17] It has predilection for second decade of life.[18] Some elements of calcification and bone may be found within this lesion, which display flecks of opacification when viewed under plain radiographs. It is believed this calcification and bone are formed from the pluripotential ability of periodontal ligament from which this lesion arises when chronically irritated.[19] Treatment involves surgical excision and deep curettage of the lesion down to bone to avoid recurrence.

Although no area of radio-opacity was seen radiographically in our case to suggest POF, microscopy however revealed calcific like materials and bone formation in an area, necessitating a diagnosis of POF while another area showed ulcerated epithelium infiltrated by chronic inflammatory cells, pyogenic fibrinoid membrane and proliferating vascular channels suggestive of prior existence of PG in the lesion. This clearly showed that this lesion was captured in transition (from PG to POF) and if left over time may form full-blown POF. Furthermore, immuno-histochemical study conducted by Elanagai et al support that RHLs exist as a spectrum following the quantification of osteopontin expression in them.[20] It was found that not only that the bone and calcific-like materials found in all POF expressed osteopontin; some stroma cells and extracellular matrix in PG also expressed osteopontin showing that those stroma cells in PG end up to lay down bone seen in POF over time.[20]

CONCLUSION

The case report documents that transitioning of a granulation tissue to PG and later POF could result if RHLs are constantly subjected to chronic irritation over a time period. Treatment modalities, which include proper surgical excision, aggressive curettage of the surrounding tissues and optimal oral hygiene measures are invaluable in treating any of the RHLs and prevention of recurrence since they are same lesion in different phases of development. Constant review is also advised.

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Conflict of Interest-
None declared

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