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RESEARCH ARTICLE
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PSEUDOEPITHELIOMATOUS HYPERPLASIA: A CASE REPORT

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Abstract
Pseudoepitheliomatous hyperplasia (PEH) is a benign epithelial proliferation that often mimics squamous cell carcinoma (SCC), posing a diagnostic challenge for clinicians. This case report highlights the importance of biopsy in distinguishing PEH from malignancy and presents the successful surgical management of a PEH lesion using a buccal fat pad flap. The case underscores the role of compliance in wound healing and demonstrates the clinical utility of the buccal fat pad in oral reconstruction, particularly in medically compromised patients.

Keywords: Pseudoepitheliomatous hyperplasia, squamous cell carcinoma, buccal fat pad, oral ulcer, biopsy, oral reconstruction

Introduction

Pseudoepitheliomatous hyperplasia (PEH) is a reactive epithelial condition that arises in response to chronic trauma, infection, inflammation, or neoplastic stimuli. While PEH is benign, it often exhibits features that closely resemble squamous cell carcinoma (SCC), leading to potential misdiagnosis unless confirmed by histopathology[1–3]. Clinical appearances such as ulceration, crusting, and irregular nodularity may further confuse the diagnosis.

This case report presents a patient with a palatal lesion initially suggestive of malignancy, which was ultimately diagnosed as PEH and managed with excision and buccal fat pad reconstruction. The case emphasizes the value of histopathological evaluation and the advantages of buccal fat pad flap (BFP) reconstruction in compromised patients.

Case Report

A 67-year-old male presented to the Department of Oral and Maxillofacial Surgery with a complaint of a persistent, painful ulcer on the left side of the palate, present for one month (Figure 1). The lesion appeared pale yellow, measured approximately 2 × 2 cm, and was tender on palpation. The patient had a history of type II diabetes mellitus, well-controlled with oral hypoglycemics for the past decade.

Routine blood investigations were unremarkable. An incisional biopsy was performed under local anesthesia (Figure 2), and the patient was advised to discontinue use of his upper denture for 30 days. Histopathological analysis revealed fibrous hyperplasia, and the lesion appeared to regress.

However, 20 days later, the patient returned with an increased lesion size and slough formation (Figure 3). On questioning, it was revealed that he had resumed using his denture prematurely. Given the lesion's recurrence and progression, an excisional biopsy was planned, followed by defect coverage using a buccal fat pad flap. The procedure was performed under local anesthesia, and post-operative healing was satisfactory. Within 15 days, the lesion had significantly reduced in size, with no signs of infection or recurrence (Figure 4). The patient was scheduled for regular follow-up visits, with no further complications observed.

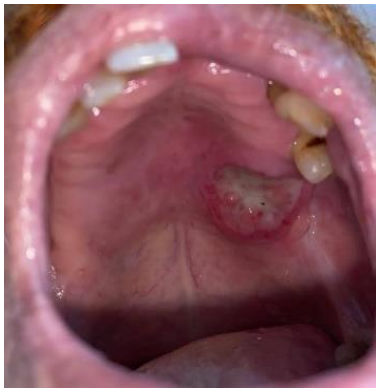

Figure 1: Lesion

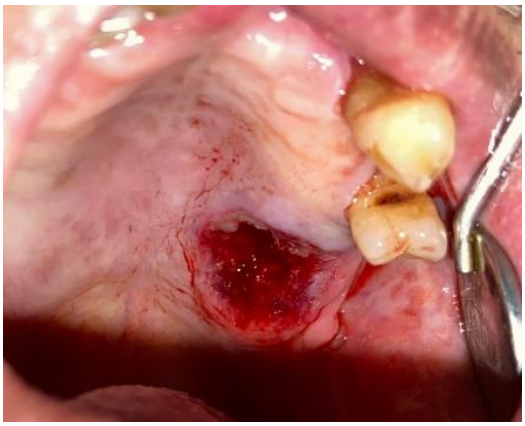

Figure 2: Incisional biopsy under local anesthesia

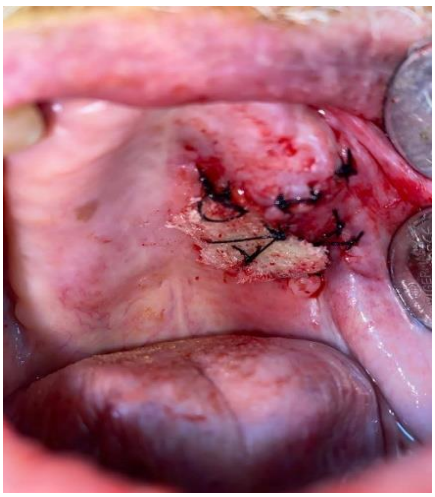

Figure 3: Increased lesion size and slough formation

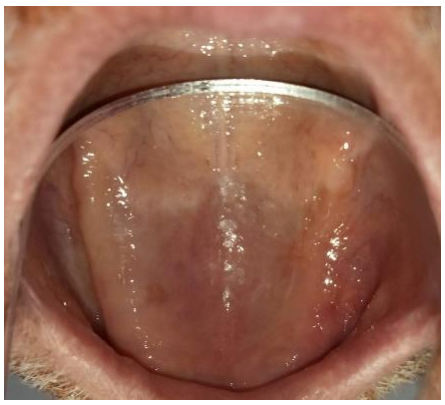

Figure 4: Follow-up with no sign of Infection.

Discussion

PEH is characterized by the proliferation of epidermal and adnexal epithelium in response to various irritants. It may occur as a primary lesion or as a secondary response to pre-existing tumors or chronic irritation[4]. Clinically, PEH often presents as a nodular or plaque-like lesion with scaling or ulceration, closely resembling SCC[5]. In some cases, the lesion may be pigmented, as seen in melanoma, further complicating the differential diagnosis[6].

The underlying pathogenesis is believed to involve cytokine release from inflammatory or tumor cells, which promotes epithelial proliferation[7]. PEH is histologically classified into three grades:

- Grade I: Acanthosis with elongated rete ridges and an intact basement membrane.
- Grade II: Irregular epithelial projections with loss of basement membrane integrity.
- Grade III: Deep epithelial extensions resembling well-differentiated SCC, often with granulomatous changes[4].

Distinguishing PEH from SCC is critical. SCC typically demonstrates elevated levels of p53 and matrix metalloproteinase-1 (MMP-1), with reduced E-cadherin expression[8]. Cytological hallmarks of SCC include nuclear enlargement, hyperchromasia, irregular contours, coarse chromatin, and prominent nucleoli[9]. Additionally, features such as feeder vessels and intrinsic vascularity on clinical examination may suggest invasive malignancy[10].

In our case, the lesion initially responded to conservative management but recurred due to non-compliance. A definitive excision and reconstruction using a buccal fat pad flap was performed. The BFP is a well-established reconstructive option for small-to-medium intraoral defects, particularly in the posterior maxilla[11]. It offers excellent vascularity, minimal donor site morbidity, and ease of access—all particularly beneficial in patients with systemic conditions like diabetes mellitus[12–15].

Compared to free vascularized grafts, which may require general anesthesia and have a higher failure rate in poorly vascularized tissues, the BFP provides a safe, quick, and effective method for achieving soft tissue closure[13]. Furthermore, the buccal fat pad's anatomical proximity and consistent volume (~10 mL) throughout life make it ideal for reconstructive procedures in the oral cavity[14].

Conclusion

PEH remains a diagnostic dilemma due to its close resemblance to SCC, both clinically and histologically. Biopsy and careful histopathological examination are essential for accurate diagnosis. This case emphasizes the importance of patient compliance and illustrates how the buccal fat pad flap offers a reliable and efficient reconstructive option for intraoral defects, particularly in medically compromised patients.

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