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Case Report

Pseudoepitheliomatous Hyperplasia: A Diagnostic Dilemma

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Abstract

Pseudoepitheliomatous hyperplasia (PEH), a neglected entity by oral pathologist, possesses utmost importance in the field of research. Of all the investigative challenges, PEH, a reactive epithelial proliferation is seen secondary to lesions with infectious, inflammatory, reactive, and degenerative origin. Small-sized samples, incomplete excision, improper orientation, and dense inflammatory changes render diagnostic confront to the oral pathologist in the exclusion of frankly invasive malignant lesions such as squamous cell carcinoma (SCC) from lesions exhibiting PEH. In this case report, we presented a case which was misdiagnosed as PEH, and due to this negligence, patient underwent extensive surgery of orofacial structures. This case was diagnosed as SCC on the basis of history, findings, and clinical examination, but due to the fear of the patient and negligence, the patient suffered a lifetime loss and compromised quality of life. Emphasis should be made on the amalgamated efforts of the clinician and pathologist to rule out this type of mystify lesion which will help in rendering the appropriate diagnosis and treatment planning.

Key words: Benign proliferation, Pseudoepitheliomatous hyperplasia, Squamous cell carcinoma

INTRODUCTION

Pseudoepitheliomatous hyperplasia (PEH) is taken into account to be a "benign proliferation of the epidermis into irregular squamous strands extending down into the dermis, respectively,[2] with no cytological atypia and mitotic figures."[3] Dr. Unna (1896) delivered to light the primary case of PEH as "Epidermal proliferation overlying a lesion of lupus." This lesion is additionally referred as pseudocarcinomatous hyperplasia[3] (as they mimic epithelial cell carcinoma squamous cell carcinoma [SCC])[4] and invasive epidermal hyperplasia, invasive acanthosis, verrucous epidermal hyperplasia, and carcinomatous hyperplasia.[5] Clinically, these wounds appear as skin ulcers/wounds, verrucous/multinodular growth, cauliflower growths, and dome-shaped swelling with smooth/warty surfaces.[5] This reactive proliferation of the epithelium occurs secondary to persistent inflammation resulting from the chronic traumatic wound, ulcer, bacterial/fungal infection, degenerative changes, retained foreign material, dermatitis, traumatic implantation of epithelium, and malignancy.[2,5] Lesions flaunting PEH are discerned by the gold standard of biopsy from invasive nasty lesions. PEH could be a benign lesion requiring only local conservative excision, whereas nasty lesion needs radical surgery. As this entity is neglected among pathologist, this article is written in view to enlighten the irreversible loss of orofacial structures due to its misdiagnosis.[1-3]

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CASE REPORT

A 35-year-old male patient reported to our department of oral medicine and radiology with the chief complaint of pain and swelling in left lower back tooth region for 3–4 days. The patient gave a history of similar type of swelling on the same region which was noticed 4–5 years ago. At that time, the patient visited to the dentist who gave

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him some medications for 5 days (patient does not know the name of the medications), and swelling was subsided at that time. Now, since 1 year, the patient is noticing the similar type of swelling which was painless and present on the left side of tooth region. The patient did not visit to any other doctor for 1 year. The patient started experiencing pain in that swelling since 3–4 days. Pain is continuous and dull aching. Pain is not radiating to any other associated surrounding structures. The patient did not take any over-the-counter medication for pain relief. The patient gave no relevant medical history. The patient has a habit of smoking bidi 2–3 packets per day since 5 years. The patient also has a habit of chewing tobacco 2–3 packets per day since 10 years.

On extraoral examination, there is no swelling present and has a symmetrical face [Figure 1]. On intraoral examination, while inspection, there is ulceroproliferative growth present on interdental and attached buccal gingival of 36, 37, and 38, which is obliterating the buccal vestibule region [Figure 2]. Blanching on bilateral buccal mucosa is appreciable. Homogenous white patch present on the right side of buccal mucosa adjacent to 46 and 47 [Figure 3]. While palpation, there is firm, indurated, sessile growth present on the buccal attached gingiva of the 36, 37, and 38 extending the gingivobuccal sulcus (GBS). There is buccal vestibule obliteration present on the left side. A growth is having a round border which bleeds to touch due to ulceration superimposing the swelling. There was a presence of fibrous bands palpable on the bilateral buccal mucosa. Mouth opening of the patient was normal. An intraoral periapical radiograph and orthopantomogram (OPG) were done which shows nothing significant changes in bone [Figures 4 and 5].

A diagnosis of SCC of GBS at 37 and 38 region was made. Other diagnoses made were OSMF Grade 1 and homogenous leukoplakia on right buccal mucosa. The patient was counseled to quit the habit of smoking and tobacco chewing. The patient was given nicorette 2 mg to be chewed when there is urge to use tobacco. The patient was advised intralesional injections dexamethasone 4 mg (dexona) and hyaluronic acid (Hynidase) twice weekly for the 1 month. Patient was prescribed 0.1% triamcinolone acetonide (kennacort) for topical application in all the surfaces of oral cavity for 3 times a day for 1 month except the white patch which was present on right buccal mucosa. The patient was also prescribed retino a cream to be applied admixed with honey on white patch present on buccal mucosa 3 times a day for 1 month. The patient was prescribed tablet SM Fibro once a day for 1 month. Patient was referred to the department of oral and maxillofacial surgery for incisional biopsy.

Patient reported to our department after 10 days with biopsy report which was came out to be SCC. The patient

Figure 1: Extra oral image

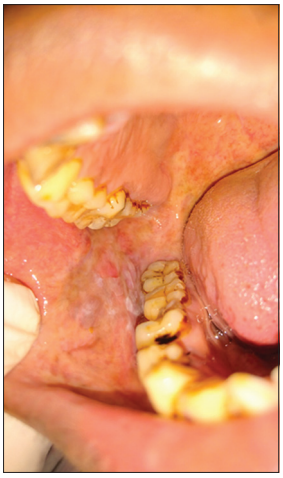

Figure 2: Intra oral image

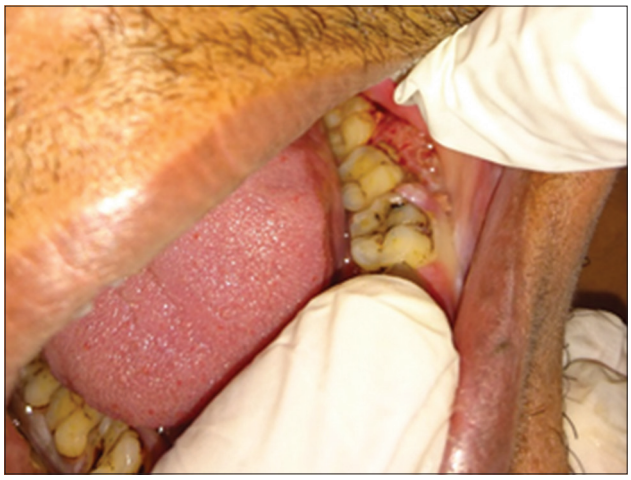

Figure 3: Pseudoepitheliomatous Hyperplasia

was not convinced and visited to other hospital and stopped all the medications prescribed by us and had frozen section procedure. Report came out to be PEH with no evidence of malignancy. The patient was treated with marginal mandibulectomy with reconstruction with

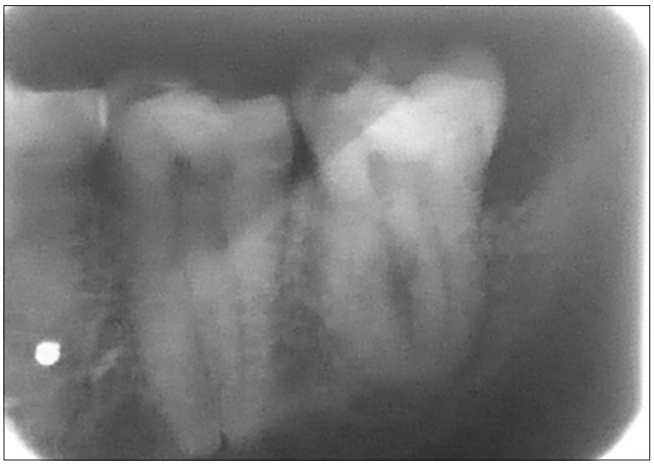

Figure 4: IOPA

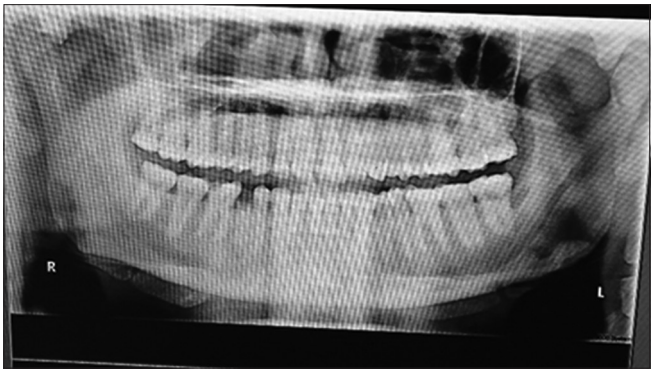

Figure 5: OPG

tongue with buccal fat pad flap on left side, and OPG was taken. Moreover, the specimen was sent for the biopsy after the procedure. The biopsy report came out to be well-differentiated SCC. Patient again came back to our department after 20 days with complaints of swelling in left alveolus region and reduced mouth opening. Now, the patient was referred to the cancer hospital for extensive oral surgery and radiotherapy. Patient remains asymptomatic for the next 2 years. Patient came to our department after 2 years with the complaints of pain in oral cavity and has pain while deglutition. On examination, there were no oral findings, so he was referred to ENT doctor for throat examination. Patient was followed up after the check-up and diagnostic tests that were advised by the ENT specialist. Patient was again diagnosed with malignancy in throat. He was treated for the same in cancer hospital, and now, he is not able to speak. Patient is still under follow-up.

DISCUSSION

PEH resembles SCC on histology, but these two entities are totally alike in terms of the patient management, and prognosis is concerned. These two diseases are customarily come across in biopsy specimens from the oral cavity and also from head-and-neck mucosa. If the sample tissue shows inflammation and has poor tissue orientation in the specimen, then it becomes too difficult to differentiate between these two entities.[6] PEH is primary, as in primary gingival PEH, or secondary, because of various reasons.[4] The differentiation between the PEH and nasty SCC becomes tougher when the deep rete pegs anastomose with each other or are cut tangentially. Furthermore, often patients would have already taken treatment for the oral cavity lesions and are on follow-up and repeat biopsy specimens are submitted where the tissue architecture may well be already distorted giving rise to difficulty in interpretation.[6]

Distinguishing PEH from SCC is of utmost importance for further management and prognosis of patients. In the abovementioned case, patient was diagnosed as SCC on the basis of clinical findings. Due to diagnostic dilemma, PEH resemblance to SCC and lack of the knowledge of the pathologists lead the patient to undergo extensive surgery and radiotherapy sessions which affected the patient physically, financially, and mentally. If it was being treated as SCC earlier, then patient would not have lost his speech. Always correlate the diagnosis with clinical findings as well as a history of the patient to save the patient from such disaster.[4-7]

CONCLUSION

PEH may be a benign epithelial proliferation identified microscopically in association with various heterogeneous lesions. The pathogenesis of PEH remains unclear; however, a scientific knowledge of PEH is important to rule out neoplasms. Clinicopathologic correlation remains a gold standard to succeed in the precise diagnosis. Tissues which are tiny in size, inappropriate orientation, and heavy inflammations in numerous lesions revealing PEH is very tough for pathologists to discriminate them from frank most aggressive lesions such as SCC. Adequate excision and sampling depth render in the exclusion of frankly malignant lesions and aid in appropriate treatment to the patient. Collaboration between clinician and pathologist is totally essential to deliver suitable treatment to the patient and avoid undesirable consequences. The diagnosis can occasionally be difficult as they mimic other lesions also, on clinic-pathological assessment. Thus, this text gives an insight regarding the assorted concepts of etiopathogenesis, histopathology, medical diagnosis, and malignant potential of PEH. A combined effort of a clinician and pathologist benefits every patient to rule out malignancy and render appropriate treatment because the only local conservative approach is important to get rid of PEH-associated lesions.

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