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Concatenation of Dysplasia to Carcinoma in Situ: Case Report

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Abstract: *Most oral and oropharyngeal lesions are innocuous and can be indisputably recognised and named based upon their clinical appearance alone. However, some lesions are difficult to identify and require a biopsy. Small percentage of these lesions may be premalignant or even malignant. If left untreated, lesions soon progress to dysplasia and then to carcinoma in situ. To stop this progress early diagnosis and intervention is of essential, which also reduces the morbidity and mortality. Complete excision and reconstruction is the choice of treatment for dysplastic lesions. Here we present a case report of 60 year old male with carcinoma in situ of soft palate, which was reconstructed with split thickness skin graft.*

Key Word: *Leukoplakia, Dysplasia, Carcinoma in situ, Reconstruction*

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I. Introduction

Risk of developing cancer increases with advancing age and same is true for premalignant lesions. Most lesions are detected in >40 years and those with similar risk factors for oral cancer, such as tobacco and/or heavy alcohol use. In oral mucosa we might encounter some epithelial lesions which may be at risk for transforming into an oral cancer, although it is difficult to predict which lesions will transform and how long it will take. Oral dysplasia term encompasses leukoplakia, erythroplakia, speckled leukoplakia and carcinoma in situ. Most oral lesions are innocuous and can be indisputably recognised and named based upon their clinical appearance and site alone. However, some lesions are difficult to identify and require a biopsy. Among these small percentage of these lesions may be premalignant or even malignant. Here we present a case report of 60 year old male with carcinoma in situ of soft palate. [1]

II. Case Report

A60 year old male reported to our OPD with the chief complaint of cheek bite and overgrowth in mouth since two months. Past dental history revealed that patient visited private dental practitioner 14 months back with complain of pain in left retro molar region. There conservative management was done and patient was symptom free for few days. Again after few months he developed pain and swelling in the cheeks, so he consulted the doctor once again but this time he was referred to us. A detail history was taken and thorough examination was done. Patient was alcoholic and occasional betel nut chewer. Face was bilateral symmetrical. Intra orally there was red and white patches on the right buccal mucosa. [Fig : 1] On left side there was presence of presence of rough overgrowth present on left half of soft palate approximately 4cm × 3 cm and pterygomandibular raphe which was of same colour as of oral mucosa with thick white plaque. [Fig: 2]Overgrowth on soft palate didn't cross the midline, uvula was spared. Patient was advised incisional biopsy which revealed moderate dysplasia of soft palate right side and speckled leukoplakia of buccal mucosa left side. Patient was advised for contrast CT scan and complete excision of the same but he denied at that time. Again patient reported to our OPD after 8 months with the same chief complaint along with big mass in palate. Intraoral examination revealed that the palatal overgrowth had spread widely. This time patient also complained of difficulty in swallowing. So once again incisional biopsy was done, but this time report came out to be severe dysplasia of soft palate [Fig: 3]. Contrast CT scan was advised, which showed focal mucosal thickening of soft palate and left para pharyngeal area. No evidence of lymph node enlargement was there. Patient's preoperative picture three day prior to surgery [Fig: 4]. Excision of the entire dysplastic tissue and reconstruction of the raw area was planned under general anaesthesia. Under nasal intubation dysplastic tissue with safe margin was excised with electrocautery. Initially dysplastic tissue seemed to be adhered closely to the underlying soft palate mucosa but eventually it separated from it with finger dissection. [Fig: 5] For reconstruction initially buccal fat from right side was procured, but it was not of sufficient length to cover the entire raw area. [Fig: 6] So the same was reconstructed with split thickness skin graft from right thigh.[Fig: 7] Oral intake of fluid was discontinued for one week for stabilization of graft, so patient was shifted to Ryle's tube feeding. Patient was followed every

day for viability and uptake of graft. Excised mass was sent for histopathological examination, which revealed carcinoma in situ. Post operatively patient was followed with the gap of every 5 days, then 10 days, quarterly [Fig: 8] and now it's on monthly basis [Fig:9]. Patient has no complaint of unintelligible speech and poor swallowing.

III. Discussion

The pharynx include base of tongue, soft palate, and uvula. Pharynx is further divided into nasopharynx, oropharynx and hypopharynx. Soft palate is part of oropharynx. Among all sites of oral cavity percentage of leukoplakia occurring in soft palate is 10.8% and if left untreated chances of showing dysplasia or carcinoma is 18.8%. Many oral cancers are preceded by potentially malignant or pre invasive state, which are detectable as distinct dysplastic lesions. Among clinical leukoplakia lesion, it has been usually seen that only about 50% of lesions show evidence of dysplasia, the remainder shows nonspecific hyperplasia and hyperkeratosis. It has also seen that up to 50% of squamous cell carcinomas arise from pre-existing oral lesions. [2, 3, 4]

Dysplasia is a continuum of progressive cellular disorganization graded as mild, moderate, severe, or carcinoma in situ. In carcinoma in situ the atypical cells are in all layers of the epithelium. In squamous cell carcinoma these abnormal cells are no longer confined just to the epithelium but have breached the basement membrane and enter into deeper tissues. Severe epithelial dysplasia has an overall malignant transformation rate of about 16% Moderate dysplasia's have a malignant transformation potential of 3–15%, whereas mild epithelial dysplasia shows a very low risk of 5%. At present, the degree of dysplasia is the best guide to potential progression of oral lesions. With regards to the clinical lesion it is apparent that only about 50% of biopsied leukoplakia's show dysplasia. [5,6] There are 90% chances that erythroplakia and speckled leukoplakia may transform into Carcinoma in situ or frank invasive squamous cell carcinoma. [7, 8]

The risk of malignant transformation varies directly with the duration of a lesion. Study done by Silverman et al inferred that patients developed invasive squamous cell carcinoma with pre-existing oral dysplasia and found that mild, moderate, and severe dysplasia had a progressively shorter interval time to frank malignant transformation. [9, 10] Some additional reports suggested that leukoplakia's may become malignant if left un-treated for 36 months, regardless of histological grade. **[11, 12]** In our present case patient had a long untreated duration, hence the lesion progressed to carcinoma in situ within a year.

Carcinoma in situ is the most severe form of epithelial dysplasia, characterised by full thickness cytological and architectural changes. Here abnormal cells have not spread beyond where they first formed. The words "in situ" translate to "in its original place."

Causative factors for oropharyngeal cancers include tobacco use, drinking alcohol, drinking and smoking together, Betel quid and gutka. Other causative factor is Human papillomavirus (HPV) infection. Oral cavity and oropharyngeal cancers that are associated with HPV tend to have a better outcome compared to those that are HPV negative. [13, 14]

Many researches are under process to learn about the DNA changes that cause the cells in the oral cavity and oropharynx to become cancer. In addition to biopsy, detection of oral dysplasia may be done by biologic predictors like P53 tumor suppressor gene mutations. It has been associated with an increased risk of recurrence after complete local excision. In normal oral mucosa P53 mutations are not typically present, the incidence of mutations ranges from 36% in hyperplasia, 85% in dysplasia, to 94% for invasive squamous cell carcinoma. So in future identification of P53 may have a role in the genetic profiling and risk stratification of oral dysplasia. [15]

Floor of the mouth, tongue, and lips were high-risk sites for oral carcinoma in situ. [1] Usually in the oral cavity such changes are relatively rare, and often, even in the presence of the most severe atypia, there is still an intact keratinised surface layer. There are two schools of thoughts according to one it's a premalignancy, but others regard it as evidence of actual malignant change but where invasion has not occurred. In our case report there was a gradual change from moderate to severe dysplasia, then to carcinoma in situ over a period of year time.

There are several methods available to the oral surgeon for resection of oral and mucosal dysplasia, among which the classical method of surgical excision is with a scalpel. Advantage of this traditional method is that it provides reliable tissue sample for post surgical histo pathological examination. Other methods are use of the carbon dioxide (CO_2) laser. Liquid nitrogen cryotherapy has limited usefulness when it comes to oral mucosal dysplasia, either as primary or adjunctive treatment. Also recurrence rate is high with this modality. [16]

If the lesion is of small dimension < 1cm, then there is no need for reconstruction of the raw area as it will heal via secondary intention, not much affecting the function. But in large areas of mucosal denudation healing via secondary intention can lead to unfavourable cicatricial changes with restriction of oral function, so reconstruction is of utmost importance. Modalities available for reconstruction of oral region are mucosal

grafting, split thickness skin grafting, buccal fat of pad etc. In our present case we reconstructed the raw soft palate area with split thickness skin graft which gave a very good result.

IV. Conclusion

Most oral carcinoma cases display considerable change before reaching such state. Oral dysplastic lesions with microscopic characteristic features have a high risk potential for transformation to malignancy. To have a check on this, appropriate diagnostic procedures for every suspicious dysplastic lesions in oral cavity should be considered. Early intervention in such dysplastic lesions to may be life-saving. Regardless of surgical method used, removal of all affected mucosa will reduce the risk of further premalignant or malignant degeneration. Also it is extremely important that patients with oral dysplasia must be followed by a specialist, trained to manage these types of lesions.

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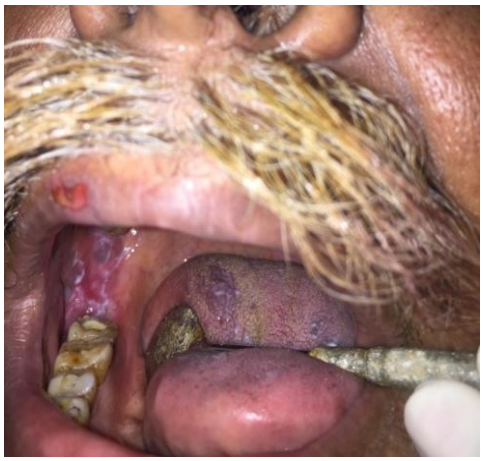

Fig 1: Pre operative red and white patches on the right buccal mucosa and retromolar area

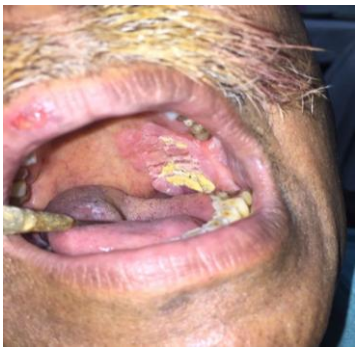

Fig 2: Pre operative overgrowth with white plaque on left half of soft palate and pterygomandibular raphe

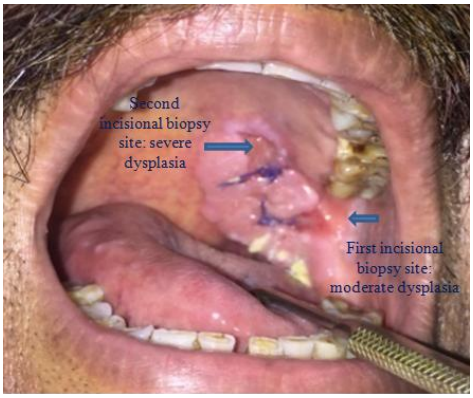

Fig 3: Showing biopsy sites

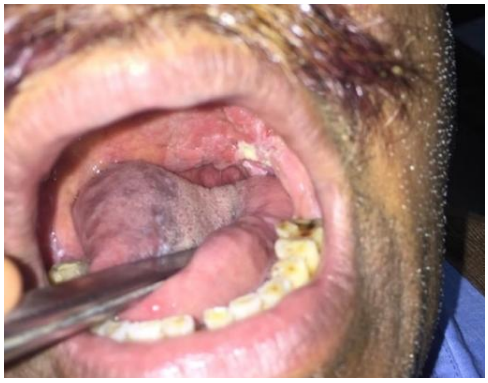

Fig 4: preoperative picture three day prior to surgery

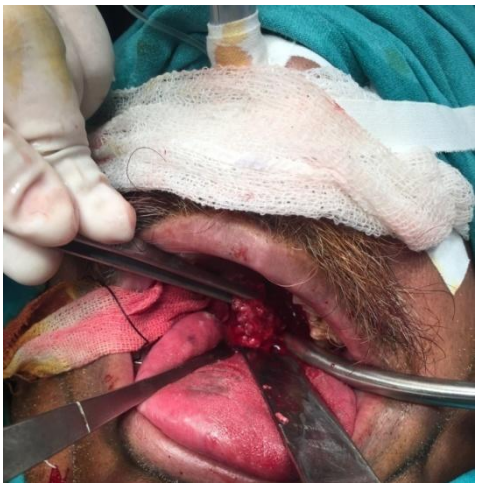

Fig 5: Excision of dysplastic mass

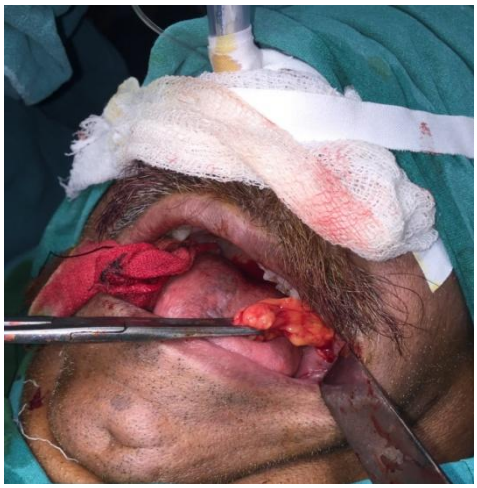

Fig 6: Procurement of buccal fat of pad

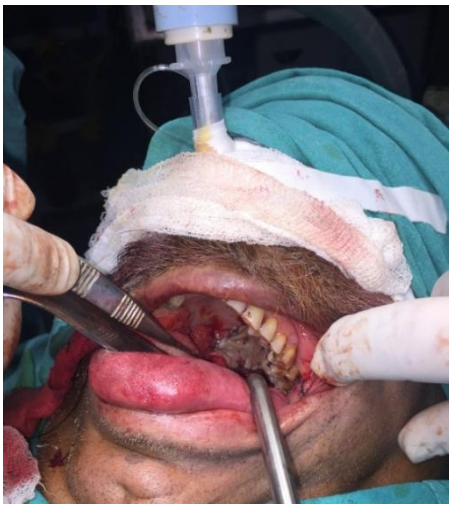

Fig 7: reconstruction of raw area with split thickness skin graft

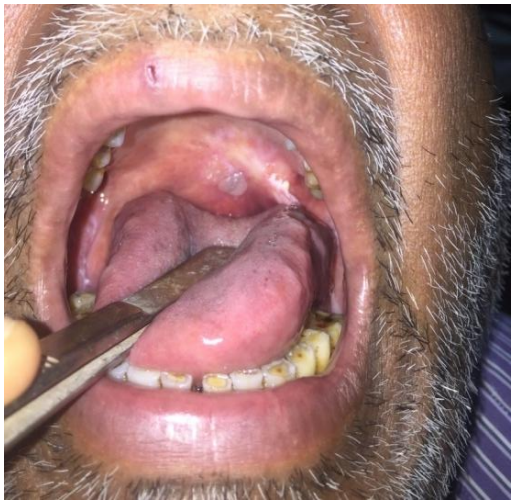

Fig 8: postoperative one month

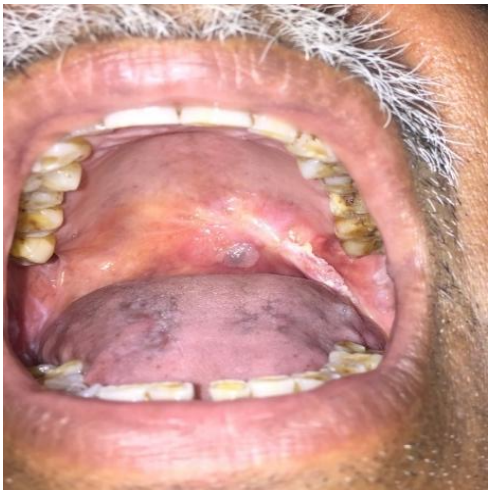

Fig 9: postoperative two month

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