Journal of Advanced Clinical & Research Insights (2019), 6, 60–62

CASE REPORT

Case report on oral leukoplakia with superadded fungal infection

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Keywords:
Candidal leukoplakia, oral leukoplakia, oral squamous cell carcinoma, premalignant disorder

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Received: 01 Feb 2019
Accepted: 26 March 2019

doi: 10.15713/ins.jcri.261

Abstract

Leukoplakia of the oral cavity is a precancerous lesion has a malignant potential and life threatening if not diagnosed early. The predisposing factor of *Candida* in leukoplakia has been a matter of argument of late. The fungus *Candida albicans* intrusion was proposed to be a noteworthy hazardous component for the threatening change of oral leukoplakia, and furthermore, it was found to be related with certain clinical attributes, for example, tissue injury, size of the lesion, site in the oral cavity, dysplastic changes, and tobacco use. Females as compared to males had a greater risk of malignant changes which was demonstrated in few researches. Various treatment modalities are incorporated to manage this condition which includes antioxidant therapy, supplements of carotenoids, and antifungal agents.

Introduction

Oral leukoplakia (OL) is a potentially malignant disorder (PMD) of the oral mucosa. It has been defined as "a predominantly white lesion of the oral mucosa that cannot be characterized as any other definable lesion."[1] It is also defined as "A white plaque of questionable risk having excluded (other) known diseases or disorders that carry no increased risk for cancer," which is well-known PMD of the oral mucosa. It was noted that 15.8–48.0% of oral squamous cell carcinoma (OSCC) patients were associated with OL in few studies.[2]

Perhaps, due to the uncommonness of associated investigations in developing countries, a solid conclusion on the worldwide malignant transformation of this condition is right now unavailable.[3] Therefore, assessing of the causative factors, which have the high potential to turn OL to malignant form, is still necessary.

Case Report

A 34-year-old male patient reported to our department with a chief complaint of whitish patch in the mouth for 4 weeks. Lesion was noted while brushing, and the patient experienced burning sensation on consuming hot and spicy food. On elucidating the habit history, the patient had the habit of chewing tobacco with betel quid for 20 years, 4–5 times/day. There was no significant medical history. On extraoral examination, no significant abnormalities were detected [Figure 1]. On intraoral examination, a well-defined plaque-type patch seen on bilateral buccal mucosa measuring about 3 × 4 cm in size, extending from commissural area bilaterally until the retromolar trigone anteroposteriorly, superior-inferiorly 1 cm above and below the mucosa. Borders are well-defined with surrounding erythematous mucosa [Figures 2 and 3]. Lesion gives a "crack mud" appearance. On palpation, lesion was non-scrapable non-tender, with no signs of indurations. These are the clinical pictures of the case. Incisional biopsy was performed to rule out malignancy [Figure 4].

Discussion

Cigarette smoking, alcohol consumption, and betal/tobacco chewing habits have been positively related with oral lesions such as oral submucous fibrosis (OSF), leukoplakia, and oral lichen planus, which have been proven with the potential malignant transformation.[4] It was noted that there was a high occurrence rate of OL and oral cancer among the youngsters who were previously diagnosed with OSF.

It was reported by the authors Roed-Petersen and Daftary in the year 1972 that *Candida* infection played a crucial etiological

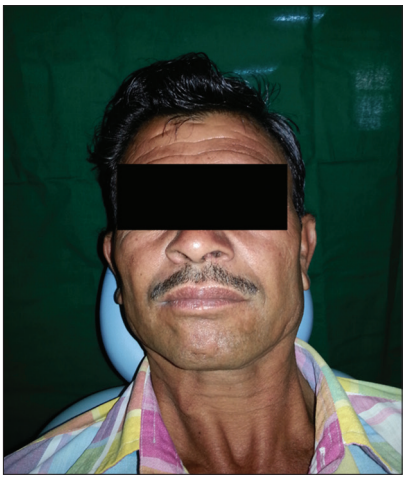

Figure 1: Straight profile image of the subject

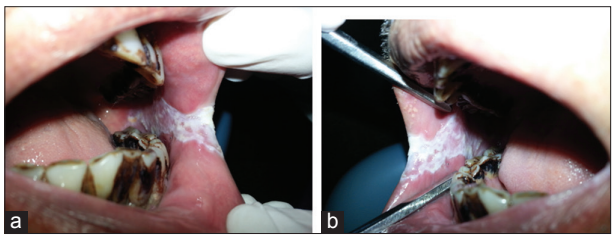

Figure 2: (a-b) Right and left buccal mucosa showing white plaque-type patch extending from commissural area bilaterally until the retromolar region

role in subjects diagnosed with OL. However, assessing the percentile value of *Candida* infection, it was found to be 13.5%, of the total OL group. As it was also noted in the literature as to *Candida* playing a major role, the clinical types and histological dysplasias have been assessed as well.[1]

The taking of a biopsy must be considered before attempting to eliminate the possible etiology, particularly when subjects are symptomatic.[5]

These *Candida*-associated leukoplakic lesions are found to be chronic in nature, and on clinical examinations, inspectory findings revealed discrete elevations, large whitish, dense, opaque plaques, on palpation it was hard to rough in consistency.[6] Moreover, if the lesions are at the commissures of the lips and the dorsal surface of the tongue, then there should be room for discussion about the diagnosis of Candidiasis versus *Candida*-associated leukoplakia. Following the antifungal treatment, if the lesions regress within the span of 4 weeks, then there is no rationale to whoop such lesions as OLs any longer. Nevertheless, in case of tenacity, the diagnosis of *Candida*-associated leukoplakia remains legitimate.[7]

Bánóczy stated the existence of that *Candida albicans* infection and its major role in malignant transformation into cancer and also OL was found to have higher probability of developing into cancer (25.9%).[8]

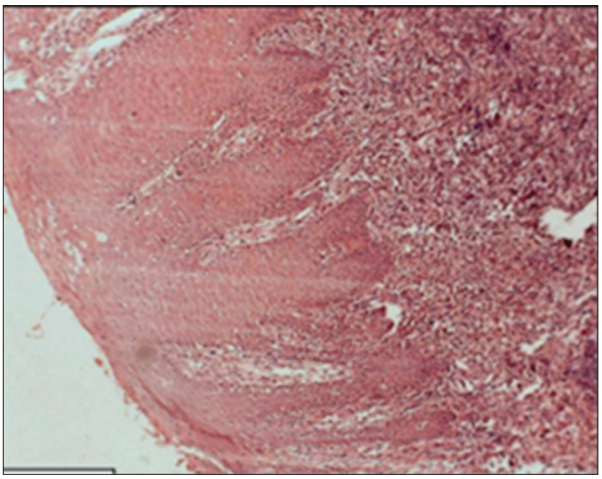

Figure 3: Photomicrograph of the biopsy site (right buccal mucosa) × 40

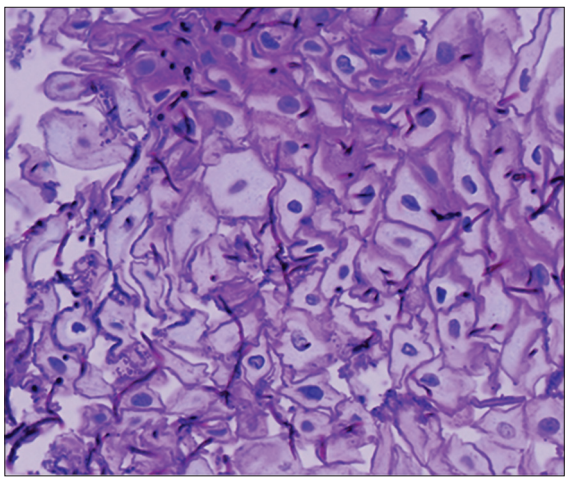

Figure 4: Photomicrograph showing fungal hyphae × 10

Non-homogeneous leukoplakias showed increased nitrosation potentials of candidal organisms as compared to the homogeneous form.[9]

The current classification of OL based on the size was subdivided into three groups: <2 cm, 2–4 cm, and >4 cm, and this has become a topic of discussion. OL is classified according to its, location/Site, size clinical presentation and histopatholical connotation (LSCP classification).[7]

The adjectives "premalignant," "precancerous," and "potentially malignant" designate the increased likelihood of malignant transformation. Currently, there seems no strong justification to change the WHO preference for the use of term "potentially malignant," for OL. In addition, using the term "potentially malignant" applies to the discussion on the different treatment modalities and the malignant transformation rate.[7]

General risk factors to be considered for conversion into malignancy in OL

Warnakulasuriya *et al.* indexed the following as the increased risk for malignant transformation from a premalignant disease.[1]

1. Gender – female
2. Duration – Chronic leukoplakias
3. Idiopathic OL – seen in non-smokers
4. Site – seen on the tongue and/or floor of the mouth
5. Size – measuring >200 mm^2
6. Type – Non-homogeneous
7. Histopathologically – the presence of *C. albicans* and epithelial dysplasia.

Basic therapeutic guidelines are noted below

1. To eliminate all causative factors.
2. If there are mild dysplastic features, treatment of surgical excision/laser surgery of the lesions is to be considered. Timely observation and follow-up are necessary.
3. Laser therapy and surgical excision are the preferred treatments for the presence of moderate-to-severe dysplasia/proliferative verrucous leukoplakia.
4. Surgical excision is best for red lesions and mixed red and white lesions (erythroplakia or leukoerythroplakia).
5. Follow-up for all lesions is a must and should be carried out.[10]

There has been improvement and disappearance of lesions on using Lozenges of Polyene-Nystatin in a significant number of cases. Patients with dysplasia in OL have shown resolution of the lesion within 11 days of systemic treatment with fluconazole antifungal agent, and *Candida*-associated leukoplakia has shown good results with topical antifungal agents including imidazoles.[11-13]

Hence, by evidence in literature, *Candida* can be considered as one of the etiological factors in OL lesions. *Candida*l lesions in immunocompromised persons would need the use of highly potent antifungal drugs such as amphotericin B.[14]

Conclusion

The early identification of OL is mandatory. Along with it, diagnosing any associated lesion is a must. Since the malignant probability of leukoplakia is high, observing and diagnosing the lesion clinically alone without biopsy must be discouraged. A biopsy must be performed to conclusive diagnosis and to do rapid treatment planning appropriately.

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How to cite this article: Lature ML, Burde K. Case report on oral leukoplakia with superadded fungal infection. J Adv Clin Res Insights 2019;6:60-62.