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Research Article

SURGICAL MANAGEMENT OF ORAL LEUKOPLAKIA WITH FREE GINGIVAL GRAFT: A CASE REPORT

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A R T I C L E I N F O

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Excision of the lesion followed by a Free Gingival Graft (FGG) was planned

A B S T R A C T

Background: Oral Leukoplakia is a potentially malignant disorder of oral mucosa. The etiology is considered multifactorial but smoking is the most frequent factor. A 49-year-old male patient was referred to the department of periodontology for the surgical excision of the leukoplakic lesion on the left lower buccal gingival region. Patient was treated with topical and systemic steroids for the same since last 2 years. On eliciting personal history, the patient had a habit of pan chewing since last 15 years and had quit the habit from 3 years. Intraorally the lesion revealed an irregular whitish plaque on the left lower buccal gingiva, firm in consistency measuring approximately 2.5 cm × 1cm. The lesion had well-defined boundaries. The surface over the lesion appeared to be rough and wrinkled. The lesions were non scrapable and nontender. No bleeding on probing from the site was noticed. Based on patient's history and clinical features a clinical diagnosis of homogeneous type of oral leukoplakia was made. Excision of the lesion followed by a Free Gingival Graft (FGG) was planned

Treatment: Excision of the leukoplakic lesion was done and the recipient bedfor FGG was prepared. A horizontal incision along with a vertical incision in the region of 35 and 38 were given to separate the lesion from connective tissue. An FGG from the 24 to 27 region of the palate was harvested and adapted onto recipient bed & the donor site was protected by Hawley's retainer. The graft was secured using resorbable sutures and periodontal dressing. The patient was followed up for a week, 2 weeks and further follow up of the patient is awaited. After two weeks, the color and appearance of the FGG was similar to the adjacent gingiva.

Conclusion: Conventional surgical approach of managing leukoplakia with free gingival graft was successful with uneventful healing.

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INTRODUCTION

Oral leukoplakia (OL) is the most frequent potentially malignant disorder of oral mucosa.[1] It was first defined by World Health Organization in 1978as a white patch or plaque which cannot otherwise be characterized clinically or pathologically as any other disease.[2] In 2012 van der Waal proposed a new definition which includes the histological confirmation "A predominantly white lesion or plaque of questionable behavior having excluded, clinically and histopathologically, any other definable white disease or disorder".[3]The estimated prevalence rate of oral leukoplakia in 2003 varied between 1.7 to 2.7% in general population.[4] OL is often found among men, and its prevalence increases with age advancement. It has been estimated that it mainly affects men over 40 years.[5]Banoczy and Rigoquote the prevalence of leukoplakia among smokers as 3.73 and mong nonsmokers as 0.26.[6] In the study by Dombi *et al*., these percentages were 6.03 and 0.22 respectively.[7]

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The etiology of oral leukoplakia is multifactorial. The most commonly associated risk factor is the use of tobacco in either smoked or smokeless form. Additionally, the use of areca (betel) nut, snuff andother forms of smokeless tobacco poses a significant risk for the development of leukoplakia. The role of chronic candidiasis in the development of leukoplakia has also been reported. In some cultures, (smoke from the burning end of a cigarette or similar device retained within the mouth) can produce a wide range of oral mucosal lesions including leukoplakia. In these populations, such leukoplakia has a 19-fold increase in the risk of malignant transformation compared with those cultures where tobacco is used in other forms.[8]OL is classified in two main types: homogeneous type which appears as a flat white lesion and non-homogeneous type which includes speckled, nodular and verrucous leukoplakia.[9] The homogeneous leukoplakia is a uniform, thin white area altering or not with normal mucosa. The speckled type is a white and red lesion, with a predominantly white surface. Verrucous leukoplakia has an elevated, proliferative or corrugated surface appearance. The nodular type has small

polypoid outgrowths, rounded predominantly white excrescences.[10]

Some other known diseases and disorders that may have a leukoplakic appearance are smokers' lesion,alveolar ridge keratosis, candidiasis-hyperplastic type, frictional keratosis, hairy leukoplakia, restoration associated lesion.[11]

It has been reported that between 16% and 62% of oral squamous carcinomas are associated with oral leukoplakia. Malignant transformation of oral leukoplakia in annual average is 1% in different populations and geographic areas with the higher risk reported by 43%.Despite enormous progress in molecular biology at present there is no certain marker to predict malignant transformation of oral leukoplakia in a particular patient. Howeverepithelial dysplasia is still currently considered "the gold standard" for determining the risk of malignant transformation.[12] According to Silverman,[13] 36% of dysplastic lesions and16% of non-dysplastic lesions progresses to carcinoma. However, it is known that epithelial dysplasia is correlated with clinically heterogeneous lesions that are considered to have the greatest risk.This present case report discusses a case of oral leukoplakia and its management with freegingival graft.

Case Report

A 49-year-old male patient was referred to the department of periodontologyfor the surgical excision of the leukoplakic lesion on the left lower buccal gingival region.Patient was treated with topical and systemic steroids for the same since last 2 years. On eliciting personal history, the patient had a habit of pan chewing since last 15years and had quit the habit from 3 years.Intraorally the lesionrevealed an irregular whitish plaque on the left lower buccal gingiva, firm in consistency measuring approximately 2.5 cm × 1cm.The lesion had well-defined boundaries. The surface over the lesion appeared to be rough and wrinkled.The lesions were non scrapable and nontender. No bleeding on probing from the site was noticed. Based on patient's history and clinical features a clinical diagnosis of homogeneous type of oral leukoplakia was made.

Since nonsurgical therapy failed to yield satisfactory results, surgical excision of the lesion was planned. As the area of lesion was huge (35 to 38), simultaneous free gingival graft to cover the excised area was planned.

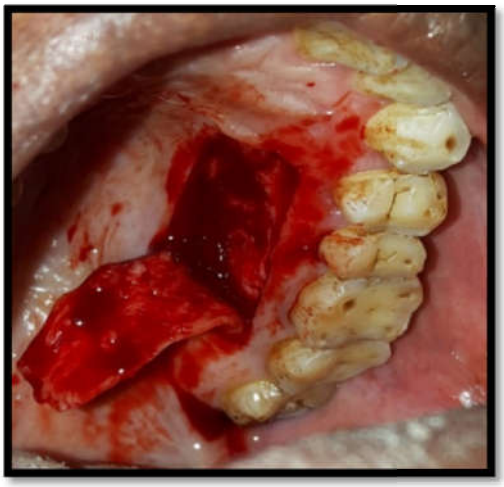
Initially,a tin foil was used as a template over the site of lesion to approximate the size of free gingival graft required from the palate.

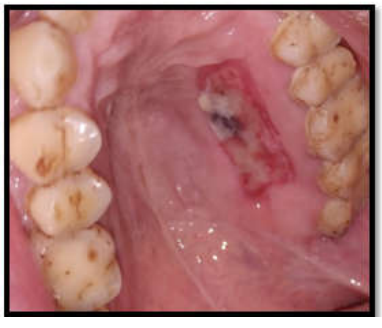
After adequate anesthesia, a partial thickness horizontal incision was given from the region of 35 to 38 with scalpel blade no 15 and a vertical incision in the region of 35 and 38 to separate the lesion from the underlying connective tissue which removed the leukoplakic lesion byholdingwith tissue forceps. The excised tissue was sent for histopathological examination.

The histopathological report revealed hyperorthokeratotic stratified squamous epithelium which exhibited acanthosis. The surface was irregular and also showed the presence of chevrons. Atrophy of the epithelium is seen in places. The

underlying connective tissue shows a mild inflammatory infiltrate composed of mainly lymphocytes suggestive ofLEUKOPLAKIA. There is no evidence of epithelial dysplasia or malignancy.

A partial thickness gingival graft was harvested from the palatal regionbetween24and 27, and was adapted to recipient bed. The template was placed over the donor site around which theincision was made. The blade was inserted at one edge to elevate the tissue by holding it with tissue forceps. After separating the graft loose tissue tags, fatty tissues and the blood clot present at the recipient site was removed to establish sufficient vascularity. The graft was secured using resorbable sling sutures and was protected with periodontal dressing, the donor site was protected by Hawley's retainer. The patient was followed up for a week,2 weeks and further follow up of the patient is awaited. Two weeks after surgery, the color and the appearance of the area with the free gingival graft was similar to that of the adjacent gingiva with uneventful healing at both the sites.





1-week Post-operative photographs

2 weeks Post-operative photographs

DISCUSSION

The main objective of oral leukoplakia's managementis to detect and to prevent malignant transformation. At first,risk activities such as smoking cessation is recommended. Following the histopathological evaluation,the degree of dysplasia will guide the choice of the treatment. Oral leukoplakia presenting low malignant risk (no dysplasia or simple dysplasia) may be either completely removed or not.[14] In the presence of moderate or severe epithelial dysplasia, surgical treatment is recommended.[3] The surgical treatment can be conventional surgery, laser ablation, electro cauterization and cryosurgery etc.[15]

However, surgical excision of OL does not lower the risk of subsequent malignant transformation but it brings the opportunity for a complete histopathological examination of the lesion.[15]

According to Monteiro *et al* results suggested that Er: YAG laser could be more effective in terms of clinical success when compared with the results obtained with the use of the traditional scalpel to treat OL. Although, there are very few reports of patients treated with Er: YAG laser in the treatment of OL.[16]

Another alternative therapy to treat oral leukoplakia is cryosurgery. According to Lodi G *et al*cryosurgeryis not considered to be the first line therapy for oral leukoplakia because of the risk of post-operative scarring and tissue contraction which limits the use of thismethod.[15]

Electro coagulation can be used alone or as an adjuvant to scalpel surgery. According toElitsa G *et al* electro coagulation produces thermal damage in the underlying and surrounding tissue, which causes postoperative pain, oedema and also leads to considerable tissue scarring.[17]

Another possible choice of management is to "wait and see" to keep oral leukoplakia under clinical and histological surveillance with frequent visits and biopsies without other treatment. This follow-up can observe an early malignant transformation and subsequent specific treatment.[15]

However, all the techniques which can be used in treating oral leukoplakia has its own, merits and demerits. In this present case the conventional scalpel technique is used for the excision of leukoplakic lesion and its subsequent management with free gingival graft due to the potential risk of malignant transformation of OL. When a tissue cell is exposed to a carcinogen, it probably tries to adapt to it.[19] Following changes could be appreciated in the oral epithelium, which includes a sequela of hyperplasia, features of cellular degeneration, a well-characterized feature of adaptation (atrophy).The cells gradually reach a stage of irrevocable cell damage, manifesting as either apoptosis or malignant transformation.[20]

The necessity for treatment was recommended by Mehana *et al* in a systematic review that surgery may reduce malignant transformation of OL with dysplasia, though it does not eliminate the risk completely.[19]Considering the patients probability of gingival recession, larger area of lesion and higher success rate the former technique was adopted.

CONCLUSION

Patient who visited our college with achief complaint of white lesion on his left lower back teeth region over the gingiva was managed by complete excision of white lesion followed by placing a free gingival graft from the palate. Patient was followed up for the first and second week. Patient was satisfied with the treatment outcome. Thus, we can conclude that conventional surgical approach of managing leukoplakia with free gingival graft can be one of the treatment options, which was successful with uneventful healing in this present case report.

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