Case Report

Management of leukoplakia of palatal gingiva using free gingival graft

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Abstract:

Oral leukoplakia is a frequently encountered oral potentially malignant disorder that, if left untreated, could progress to oral squamous cell carcinoma. Though the tongue and buccal mucosa are the most common sites to be affected, the involvement of palatal gingiva is relatively rare. It is usually associated with high recurrence rates and a higher tendency to undergo malignant transformation. Surgical excision of the lesion is considered the gold-standard treatment strategy. This case report describes a case of oral leukoplakia involving the palatal gingiva in a 71-year-old male having no deleterious oral habits. The lesion was treated using surgical excision, and the defect was repaired using a free gingival graft from the opposite palatal gingiva. Although no recurrence was noted till 2 years following surgery, the patient was advised to undergo regular monitoring.

Key words:

Free gingival graft, gingiva, leukoplakia, palate

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INTRODUCTION

Oral leukoplakia is one of the most commonly encountered oral potentially malignant disorders in our daily clinical practice. The World Health Organization Collaborating Centre in 2020 defined oral leukoplakia as "a predominantly white plaque of questionable risk, having excluded (other) known diseases or disorders that carry no increased risk for cancer."[1] They are generally asymptomatic and require a biopsy to rule out the presence of oral squamous cell carcinoma. In a recent systematic review, the prevalence of oral leukoplakia was estimated to be 1.39%, and the overall pooled estimated global prevalence was reported as 3.41%.[2] In India, the prevalence of leukoplakia is estimated to be around 5%–6%.[3] The prevalence is higher in males than in females and also higher in persons over 60 years of age. It is six times more common in smokers than in nonsmokers. Oral leukoplakia can occur at any site and can be unifocal or multifocal. The most common sites include the tongue (36%–50%), buccal mucosa (18%–26%), floor of mouth (8%–22%), gingiva (6%–22%), and hard palatal mucosa (7%).[4]

Although the exact etiology for the development of oral leukoplakia remains an enigma, there are various risk factors associated with increased occurrences in some individuals. These risk factors include smoking, tobacco chewing, betel quid chewing, alcohol consumption, immunosuppression (e.g., HIV-affected individuals), presence of human papilloma virus, family history of cancer, and certain syndromes (e.g., dyskeratosis congenita and Fanconi anemia). The absence of these risk factors indicates an underlying genetic predisposition of the individual, and the risk of malignant transformation is higher in these individuals than in those having risk factors. Proliferative verrucous leukoplakia (PVL) is a rare form of oral leukoplakia first identified by Hansen *et al*. in 1985. Its etiology is unknown, as it lacks the usual risk factors associated with oral leukoplakia, and it exhibits a strong tendency for malignant transformation.

The proportion of malignant transformation is estimated to be around 3.5%–9.8%, with the rate varying between 0.13% and 40.8%.[2] The 5-year survival rate drops sharply to 50%–66% once it transforms into oral squamous cell carcinoma, thereby endangering the patient's life and physical and mental health.[5] Therefore, prompt diagnosis, early proper treatment, and close patient monitoring for any recurrence are essential before the individual becomes a social burden. Though many medical interventions (such as retinoids, beta-carotene, and bleomycin) have

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been tried, the treatment of oral leukoplakia includes mainly surgical excision of the lesion using a scalpel, cryosurgery, laser surgery, or vaporization.[6]

This case report describes a case of leukoplakia in the palatal gingiva in a nonsmoker geriatric patient, which, after excisional biopsy, was diagnosed as verrucous hyperplasia with mild dysplasia. It was managed by surgical excision of the lesion followed by free gingival grafting of the surgical defect.

CASE REPORT

A 71-year-old male patient reported to the department of periodontics with a chief complaint of noticing a painless white lesion on the upper right gum, which had appeared rough on touching with the tongue for the past 6 months. There was no history of the patient having any deleterious oral habits, including smoking or tobacco chewing, in recent times or the past. The patient gave no history of taking any medications for any systemic diseases. There was no history of such lesions appearing in the same or other parts of the oral cavity, nor was he treated for such lesions.

On intraoral examination, a white, nonscrapable patch with brownish pigmentation was noted on the palatal gingiva adjacent to the upper right second premolar and first molar region, extending approximately 17 mm × 8 mm in greatest dimensions [Figure 1]. The surface appears corrugated and feels rough on palpation. No ulceration was noted. A composite and an amalgam restoration were present on the upper right first and second molars, respectively. The rest of the oral cavity appears normal. None of the cervical lymph nodes were palpable.

The intraoral periapical radiograph of the region appeared normal with no bone pathology. All hematological investigations were within normal limits. According to the clinical and radiographic examination, the case was provisionally diagnosed as leukoplakia of the palatal gingiva. An excisional biopsy of the lesion for histopathological evaluation was planned.

A written informed consent was taken before proceeding with the surgery. Before starting, the patient was asked to rinse his mouth with 0.2% chlorhexidine gluconate for 1 min. After swabbing the region with povidone-iodine, the area was anesthetized by greater palatine nerve block and infiltration around the 15–17 region using 2% lignocaine hydrochloride with 1:80,000 adrenaline (Xicaine, ICPA). A semilunar-shaped incision was made around the lesion with at least 3 mm of normal tissue all around, using a No. 15c B.P. blade. A crevicular incision was also given around teeth 15, 16, and 17 using a No. 12D B.P. blade. The lesion was excised by raising a full-thickness mucoperiosteal flap except at the edges, where it was a partial thickness (to act as the recipient bed for the free gingival graft). The interdental gingiva was removed using curettes to remove any remaining lesions. The area of the defect was then measured, and accordingly, a similarly shaped free gingival graft was harvested from the left side of the palate and sutured with 4-0 polyglycolic acid suture (Truglyde). An absorbable sterile collagen sponge was applied to the donor site and held in place by sutures [Figure 2]. The excised tissue was sent for histopathological examination. The patient was given 10 mg of ketorolac tromethamine immediately following surgery. Antibiotics (Amoxicillin 500 mg + Clavulanic Acid 125 mg, TDPC, Metronidazole 400 mg, TDPC) and analgesics (Ketorolac tromethamine 10 mg) were prescribed for the next 5 days. The patient was advised not to brush the region for 2 weeks. Furthermore, a 0.2% chlorhexidine gluconate rinse for 1 min was advised for the next 14 days. The patient was recalled after 14 days for suture removal.

Sections stained with hematoxylin and eosin [Figure 3] showed the presence of hyperplastic, hyper-orthokeratotic stratified squamous epithelium with nondescript underlying connective tissue. In some areas, wavy keratinization, along with plugging is noted in the superficial surface of the epithelium. Minimal dysplastic changes are observed in the epithelium. The connective tissue shows focal areas of chronic inflammatory cells and multiple dilated blood vessels. The overall histopathological features are corroborative of verrucous hyperplasia of oral epithelium with mild dysplasia. Although the histopathological features indicate that the lesion is the initial stage of PVL, considering the clinical manifestation and follow-up data, it is difficult to achieve the same. Hence, the case was treated as verrucoid leukoplakia.

The patient was advised not to indulge in any deleterious oral habits such as smoking, tobacco chewing, or alcohol consumption in the future. The patient was followed up for 2 years, every 3 months apart, with no signs of recurrence to date [Figure 4]. The patient was informed to follow-up every 6 months for at least 5 years.

DISCUSSION

Oral leukoplakia affecting the palatal gingiva is a relatively rare entity that should be dealt with caution from the very beginning, as it has been reported that 4%–20% of gingival leukoplakias tend to undergo malignant transformation.[4,7] This tendency to undergo malignant transformation seems to increase with the age of the patient and also if the lesion is nonhomogeneous. In addition, the absence of any deleterious oral habits, like smoking, in a patient presenting with oral leukoplakia (like in the above case) increases the probability

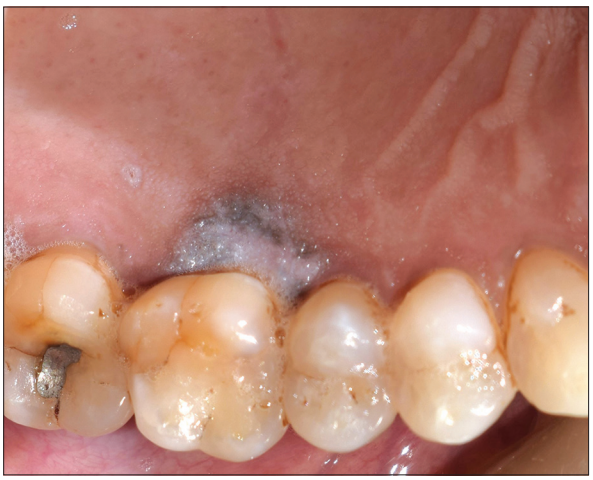

Figure 1: Intra-oral view of gingival leukoplakia affecting the palatal gingiva

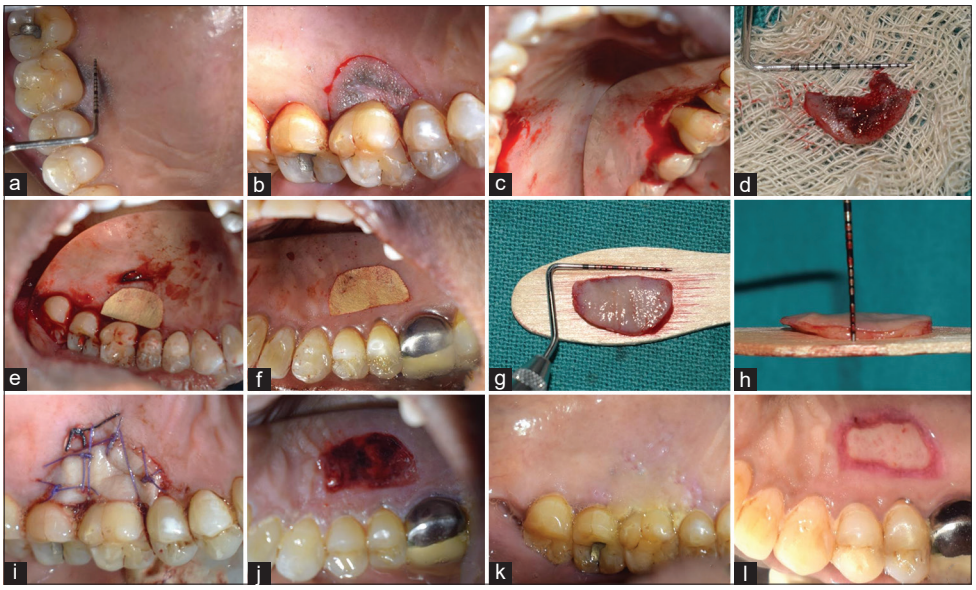

Figure 2: Intraoral view showing (a) Preoperative measurement of the tissue; (b) Incision design; (c) Recipient bed after excision; (d) Excised tissue; (e) Measurement of recipient site using template; (f) Template placed on donor site; (g) Length of the harvested graft; (h) Thickness of the harvested graft; (i) Immediate postoperative view of recipient site; (j) Immediate postoperative view of donor site; (k) Recipient site after 14 days; (l) Donor site after 14 days

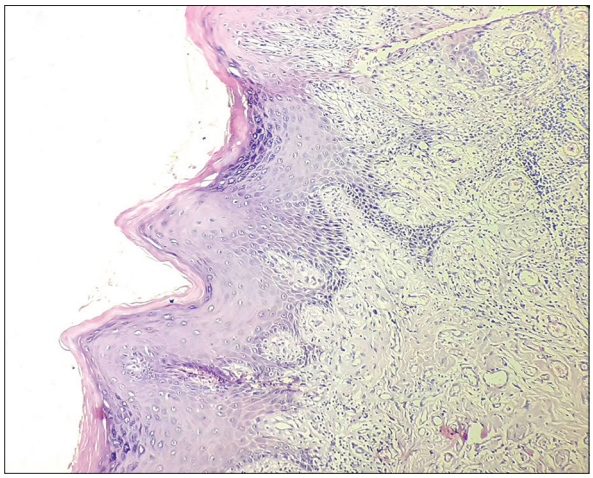

Figure 3: Haematoxylin and eosin stained section showing hyperplastic, hyperkeratotic oral epithelium with mild dysplasia

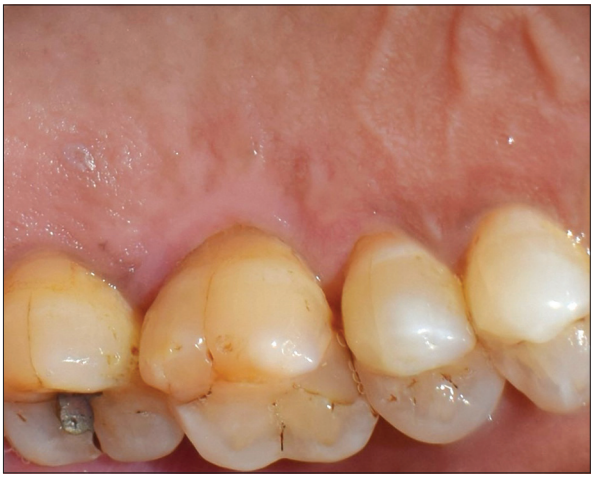

Figure 4: Follow-up after 2 years

of malignant transformation, as there seems to be a genetic predisposition. The absence of smoking and the presence of verrucous hyperplasia histopathologically might favor the lesion being an initial stage of PVL. However, since the patient is male and the lesion is restricted to a single site with <3 cm spread and has not recurred after surgery, it does not fulfill the diagnostic criteria of PVL, as stated by Cerero-Lapiedra *et al*. in 2010. Apart from this, studies have shown that gingival leukoplakia has a high recurrence rate of 42%–54.5%, making it even more worrisome. Moreover, atypical verrucous hyperplasia, which is characterized by broad-based papillary or verruciform outgrowth with or without dysplasia, is also mostly noted in gingival leukoplakia.[8] Thus, oral leukoplakia of the palatal gingiva should be diagnosed and treated as early as possible.

Several methods have been described in the literature for the treatment of oral leukoplakia. This includes surgical removal of the lesion using a scalpel, laser, photodynamic therapy, and cryosurgery; application of topical or systemic medicines (such as retinoids, Vitamin A, beta-carotene, and bleomycin); cessation of oral habits; and also, some prefer no intervention but continuous strict surveillance. A study by Lodi *et al*. showed that none of the medicinal treatments reduces the risk of the development of oral cancer more than a placebo.[9] Thus, surgical excision of the lesion remains the gold standard treatment of oral leukoplakia. Since the patient presented with leukoplakia in the palatal gingiva showing nonhomogeneous texture, along with the fact that the patient is 71 years old with no history of deleterious oral habits, surgical excision of the lesion is justified.

The surgical excision of oral leukoplakia using a scalpel involves total excision of the lesion along with a safety margin of normal tissue to minimize recurrences. A study by Kuribayashi *et al*. found that the optimal safety resection margin of at least 3 mm width is required to be excised to reduce the

recurrence rate to 10% or less. They also found that almost all the lesions that were resected with a safety margin of <2 mm in width had recurred.[10] Thus, in our case, the lesion was excised, having a safety margin of at least 3 mm all around. Apart from the width, the depth of the excision is also important to prevent recurrences. Since the gingival tissue thickness around teeth is usually <3 mm, the lesion was excised using a full-thickness palatal flap.

Another reason for the high recurrence rate of gingival leukoplakia is possibly due to the fact that complete removal of the altered cells is practically impossible, especially the sulcular epithelium surrounding the tooth. It is also common for the dysplastic/altered cells to involve the interdental gingiva and extend buccally or lingually/palatally. Thus, the removal of the interdental gingival tissue is justified as it prevents disease recurrence by removing any residual dysplastic cells.

A free gingival graft from the left palate was used to cover the exposed bone so that it aids in faster healing, less patient discomfort, and also prevents any recession around the palatal surfaces of the teeth. Free gingival grafts were initially reported by Björn *et al*. as a method to augment the attached gingiva. Later, it also found its application in recession coverage surgeries, especially in cases having narrow, shallow recessions. The thickness of the graft is of utmost importance, and it usually ranges between 1 and 2 mm. If it is too thin, it will undergo necrosis, and if it is too thick, then the peripheral layer of the graft will not receive nutrients from the plasmatic circulation via diffusion, and it will not survive. The area of the recipient bed also plays a major role in ensuring the plasmatic circulation around the graft. The survival ability of the free gingival graft in the above case is ensured by the connective tissue bed present around the edges of the graft. The donor site is left to heal by secondary intention.

Despite surgical excision, leukoplakia of the gingiva shows a high recurrence rate, and also, such lesions tend to undergo malignant transformation over time. The most widely accepted reason is the concept of field cancerization. According to this concept, genomic instability seems to be present in the entire epithelium of a patient showing dysplastic changes. So, even after surgical excision, a small random mutation at any site of the aberrant keratinocytes may lead to recurrence and cancer.[11] The risk of malignant transformation also increases with the presence and severity of epithelial dysplasia. Thus, mere surgical excision of the lesion is not sufficient. Such patients should be kept under regular surveillance and observed for the development of leukoplakia in the same site or other parts of the oral cavity. Therefore, although there was no sign of recurrence for 2 years, the patient was advised to undergo checkups every 6 months. Since, to date, the efficacy of using chemotherapeutic measures for preventing oral leukoplakia has not been proven, further medications were not prescribed.

CONCLUSION

Oral leukoplakia of the palatal gingiva is a relatively rare sighting, especially in patients having no deleterious oral habits. However, its presence should be dealt with immediately, as it shows a high recurrence rate and a higher tendency to undergo malignant transformation. Surgical excision of the lesion should be the mainstay of treatment, having a safety margin of at least 3 mm to prevent recurrences. If the defect created after excision is small, it can be managed by free gingival grafting. Even after surgical removal, periodic follow-up is necessary for early detection in case of recurrence.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given his consent for his images and other clinical information to be reported in the journal. The patient understands that his name and initials will not be published and due efforts will be made to conceal his identity, but anonymity cannot be guaranteed.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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