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NON SCRAPABLE WHITE LESION PRESENTING AS AN IDIOPATHIC LEUKOPLAKIA ON THE LEFT LATERAL BORDER OF TONGUE IN AN ELDERLY MALE INDIVIDUAL WITH DIABETES MELLITUS COMORBIDITY – A CASE REPORT

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ABSTRACT

In this case report, we highlight the importance of thorough diagnosis in a diabetic patient of the geriatric population.

KEYWORDS: *Leukoplakia, Diabetes, Tongue, Oral, Pathology*

Article History

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INTRODUCTION

Premalignant lesions and premalignant conditions are formed from oral premalignancy which is an intermediate stage. Potentially Malignant Disorders constitute premalignant lesions and conditions as per the World Health Organisation (WHO).

CASE REPORT

In the outpatient department, a geriatric male individual presented with a white patch in relation to the tongue on the left side laterally for the last 60 days. Extra oral examination was within normal limits. An approximate measurement of about 2.5cm x 2.5 cm plaque, white in colour, homogenous in nature was seen in relation to the tongue on the left lateral side during intra oral examination. [Fig-1]The surface was rough in texture and slightly raised in appearance with a cracked mud feel during palpation. The main clinical characteristics were of non scrapability and non tenderness. Diabetic medication was taken by the patient. Leukoplakia of unknown cause was considered a provisional diagnosis. Haematological investigations were within normal limits. Negativity was seen in toluidine blue staining. The performed biopsy was incisional in nature. The age of the patient played an important role in differential diagnosis which was inclusive of keratosis which might be frictional or traumatic in nature. Xerostomia, diabetes mellitus, medication and geriatric nature provoked the differential diagnosis of oral hyperplastic candidiasis. Differential diagnosis also included

chemical injury resulting in white coloured plaque. Local application of any drug or medication also leads to white plaque formation. Histopathological features included stratified squamous epithelium of hyperplasticity along with infiltration cell infiltrate intra epithelially which was in accordance with leukoplakia. Clinical examination and patient history lead to the conclusion of leukoplakia of unknown etiology otherwise known as idiopathic leukoplakia. Complete surgical excision of the lesion on par with histopathology report was in accordance with leukoplakia. The rate of recurrence was of nil capacity even after one year of patient follow-up.

DISCUSSION

Tobacco induced leukoplakia usually differs from that of one with idiopathic in nature[2,3,4]. The rate of recurrence is directly proportional to malignancy. OSCC was witnessed in a seventy six year old patient of feminine gender in whom the lesion presented as idiopathic leukoplakia then transfiguring into verrucous proliferative leukoplakia within a span of one and a half years[5]. The rate of malignant transformation globally accounts to 1% whereas 0.3% in India, finally ending up at 0.13% to 36.4%[6,7]. Carcinogenesis mainly results in the Indian male population[8]. Etiology targets smoked tobacco, smokeless tobacco, areca nut, betel nut, snuff, chronic candidiasis and nitrosamine production. The main characteristics of malignant transformation are aneuploidy and loss of hetetozygosity in case of involvement of ventrolateral surface of the tongue. Management of oral leukoplakia depends on the elimination of all risk factors, surgical excision and laser. The current lesion was named idiopathic as the patient was devoid of tobacco associated habits[8]. Leukoplakia might occur in single or multiple appearances. Gender predilection and usage of habits play an important role in etiology. Clinically, might occur as homogenous or non homogenous types. Homogenous types might present as shallow cracks or smooth, wrinkled or corrugated appearance. Verrucous, papillary and exophytic appearances contribute to non homogenous type. In case of erythroleukoplakia, lesions present as white patches or nodules on erythematous base. Irregular blunt or sharp projections are seen in exophytic lesions. Homogenous type is usually asymptomatic whereas non homogenous types of mixed white and red types are usually accompanied by pain and discomfort. Malignancy might present in case of the presence of redness or palpable induration.The hyperplastic parakeratotic epithelium was the histopathologically pathognomonic feature in this case. Histopathological features of epithelial dysplasia include hyperkeratosis in the prickle cell layer, decreased cellular cohesion, enlargement of nucleoli, nuclear hyperchromatism, cellular pleomorphism, presence of mitotic figures, e and loss of polarity of basal cells. An increased rate of aggressiveness has been noted in those with a malignant nature.Patient history, habits, systemic condition, medication and histopathology help in arriving at an accurate diagnosis. Risk factors for malignant transformation include females, chronic duration of the lesion, non smokers, tongue, floor of mouth, more than 200 mm diameter, non homogenous, candidiasis and epithelial dysplasia. Smoking increases the risk of leukoplakia in diabetics with a positive correlation of A1C concentration. In certain diabetic patients, the coexistence of oral leukoplakia with candidiasis and lichen planus was witnessed. Vasculopathy, dysregulation in the immune system, and neuropathy are characteristic triads of diabetes mellitus. The quality of life of diabetic patients is at risk of periodontitis, diseases and disorders of the salivary gland, abnormal oral mucosa and burning mouth syndrome. Diabetes mellitus predisposes to oral leukoplakia. Decreased salivary secretion and low salivary pH in conjunction with oral mucosal atrophy result in a defect in the protective barrier. Elevated blood glucose levels directly stimulate free radicals and decrease antioxidant potential. Gingival tissues exhibit microangiopathy leading to hypoxia and decreased blood supply, which might in alliance with defective cellular immune response lead to oral leukoplakia. Leukoplakia might be inflicted by smoking and alcohol. Recently, diabetes has been a third major inciting factor for oral leukoplakia. Oral lesions are commonly seen in diabetic individuals. Female predilection is witnessed in the link between diabetes and oral

leukoplakia. Increased LDL-C/HDL-C ratio is observed in diabetic patients due to deranged metabolism of lipids. Immunosuppressive drugs taken by diabetics can influence oral leukoplakia. Important factors are onset, period, habits, clinical picture, trauma and recurrence. Cell mediated immunity is affected by betel quid. This leads to impairment of T cells and cellular immune response. A diet which is rich in fat and low in fibre predisposes to oral leukoplakia. Increased intake of saturated fats also leads to increased incidence of oral cancer. Smoking, alcohol and diabetes play an important role in oral pre cancerous and cancerous lesions. Mortality in relation to oral cancerous lesions might be increased by type 2 diabetes mellitus.

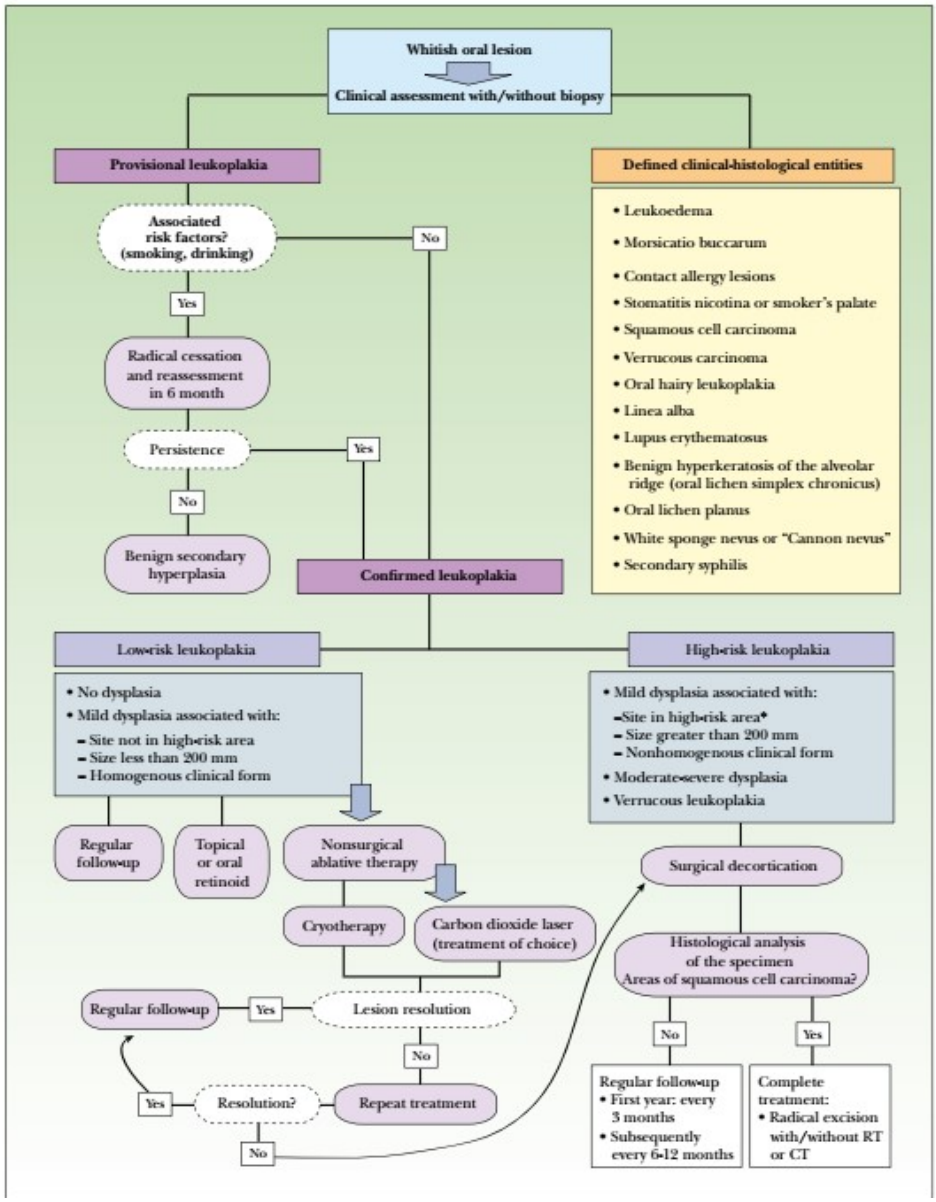

Figure 1: Flow Chart about Oral Leukoplakia

DIFFERENTIAL DIAGNOSIS IS AS FOLLOWS

- Cannon Nevus
- SLE

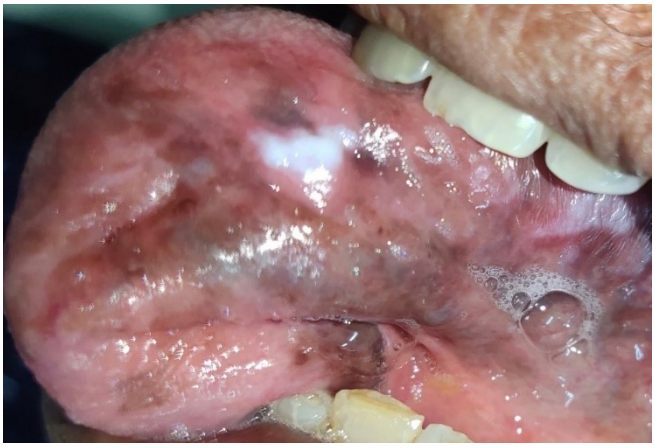

Figure 2

CONCLUSIONS

More scarcity is evident in the scientific literature on the means of idiopathic leukoplakia. Future research should account for etiology, genetics, anatomical location and malignancy for a better perspective.

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