Review Article

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ORAL LEUKOPLAKIA: AN OVERVIEW WITH EVIDENCE BASED MANAGEMENT

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Abstract

Oral Leukoplakia is a Potentially Malignant disorder of Oral Cavity commonly seen in routine dental practice. It is strongly associated with substance abuse like Tobacco, Alcohol, Arecanut etc. Since decades, many treatment modalities have been suggested and studied but no complete cure is available till date, thereby becoming a challenging condition. This review article may be helpful for the practicing Oral Medicine Specialist, Dental surgeons, ENT surgeons and Oral surgeons.

Key words: Arecanut, Alcohol, Evidence based Management, Oral Leukoplakia, Oral Precancerous lesion, Potentially Malignant isorder, Tobacco

Introduction

Oral Leukoplakia (OL) is the most common Oral Potentially Malignant disorder of the oral mucosa. The word Leukoplakia is derived from, *Leukos* meaning *white* and *plakia* meaning *patch*. OL is strongly associated with tobacco, alcohol, arecanut etc. Usually they are asymptomatic but the non-homogenous type is symptomatic in nature. The risk of malignant transformation in Oral Leukoplakia varies from 0.3% to 25%.[1,2,3]

As this condition is very common in Indian subcontinent, the present review is focused to encompass all the aspects of the disease i.e. definition, classification, epidemiology, etiology, pathogenesis, clinico-histologic presentation and various evidence based treatment modalities for this condition.

Definition:

According to WHO (1967)[1] : It is a raised white patch of Oral Mucosa measuring 5mm or more, which cannot be scraped off and which cannot be attributed to any other diagnosable diseases.

According to WHO (1978)[4] : It is a white patch or plaque in the Oral Cavity, which cannot be scrapped off or stripped off easily and more over which cannot be characterized clinically or pathologically as any other disease.

According to Axell T, Holmstrup P, Kramer IRH, Pindborg JJ, Shear M. (1983)[5] : It is a white patch or plaque in the oral cavity, which cannot be scrapped off or stripped off easily and more over which cannot be characterized clinically or pathologically as any other disease and it is not associated with any physical or chemical agents except the use of tobacco.

According to Axell T, Pindborg JJ, Smith CJ, Vander Waal (1994)[6] : It is a predominantly white lesion of the oral mucosa that cannot be characterized as any other definable lesion; some oral Leukoplakia will transform into cancer.

According to WHO (1997)[1,7] : It is a predominantly white lesion of the oral mucosa that cannot be characterized as any other definable lesion

According to WHO (2005)[1] : It is a white plaque of questionable risk having excluded (other) known diseases or disorders that carry no increased risk for cancer.

According to Warnakulasuriya et al (2007)[1]: Leukoplakia should be used to recognize white plagues of questionable risk having excluded (other) known diseases or disorders that carry no increased risk for cancer.

According to Chandramani More (2017): It is defined as a non scrapable, well delineated, raised, white patch on the oral mucosa, caused by known carcinogens; which undergoes morphological alteration and has high risk of malignant transformation.

Epidemiology:

The prevalence rate of Oral Leukoplakia is 2.6%. Most of the Oral Leukoplakia are seen in patients with age of 50 and infrequently encountered below the age of 30. Oral Leukoplakia is most commonly present in men. Gupta *et al,* 1980 showed the annual incidence of 1.1 to 2.4 percent among men and 0.2 to 0.03 percent among women.[3]

Etiology: [3,8-11]

The exact etiology of Oral Leukoplakia is Unknown but the large number of factors have been implicated for their occurrence.

Tobacco: Various forms of tobacco habits such as smoking tobacco- bidi, cigarette etc. and Smokeless tobacco chewing are main etiologic factor of Oral Leukoplakia. The lesion more frequently occurs in tobacco smokers than non-smokers and the lesion will regress once the habits are discontinued or reduced.

Alcohol: Synergistic effect of alcohol and tobacco both, increases risk of oral leukoplakia.

Microorganism infection:

Candida albicans- Chronic candidal infection are often associated

with oral leukoplakia. This type of lesion will have epithelial dysplasia when compared with non Candidal lesions.
Herpes Virus- Herpes virus type I and Human Papilloma virus have some role in development of oral leukoplakia.
Hormonal Imbalance: imbalance of both male and female sex hormones may induce keratogenic changes in oral epithelium leading to oral leukoplakia.
Dietary Deficiency: Vitamin A causes metaplasia and hyperkeratinisation of the epithelium which leads to Oral Leukoplakia. Vitamin B complex may also cause leukoplakia changes in the mucosa.
Trauma / Chronic Irritation: Chronic irritation to the mucosa by ill-fitting dentures, sharp cusp edges of teeth and eating hot and spicy food leads to hyper keratinization of mucosa.
Galvanism: Galvanic reactions occur in the oral cavity when there is difference in the electrical potential between two dissimilar metallic restorations.
UV Radiation: UV radiation leads to hyperkeratosis in the oral mucosa.

Classification:

1. According to Dr. Chandramani More, (2017)
Type 1 -Thin or Thick
Type 2- Localized or Diffused
Type 3- Proliferative or Verrucous type
Type 4- Candidal
He has excluded the mixed type of lesion (red and white) induced by Tobacco and Areca nut; with a justification that the word 'Leukoplakia' signifies the white patch and lesions other than white patch shall not be categorized under the types of Oral Leukoplakia.
2. According to Burket's, (2015)[3]
Homogenous Oral Leukoplakia -
White often well demarcated plaque with an identical reaction pattern throughout entire lesion, surface texture vary from smooth and thin to a leathery appearance with surface fissures referred as "cracked mud".
Non-homogenous Leukoplakia (Erythro-Leukoplakia)- White patch or plaque with intermingled with red elements. If the surface texture is homogenous but contains verrucous, papillary or exophytic components this type is also consider as non-homogenous type.

3. According to Shafer's, (2012)[12]
Homogenous
Smooth
Furrowed (fissured)
Ulcerated
2. Non-homogenous
A. Nodulo-speckled

4. Staging of Oral Leukoplakia according to Shafer's, (2009)[8]
According to size, clinical aspect and pathological features-
1. Size- it is denoted by L
L1- size of Leukoplakia < 2 cm
L2- size of Leukoplakia 2-4 cm
L3- size of Leukoplakia > 4cm
Lx- size not specified
2. Clinical aspect- it is denoted by C
C1- homogenous
C2- non homogenous
Cx- not specified
3. Pathological features:
P1-no dysplasia
P2-mild dysplasia
P3-moderate dysplasia
P4-severe dysplasia
Px-not specified

4. Site of Leukoplakia
S1-all sites excluding floor of mouth, tongue
S2-floor of the mouth and tongue
S3-not specified
5. According to WHO, (2005)[8]
Squamous hyperplasia – benign lesion.
Mild dysplasia – better prognosis.
Moderate dysplasia.
Severe dysplasia.
Carcinoma in-situ – poor prognosis.

6. According to Modified classification of Vander waal et al, (2005)[8]
L- Size
L1- size of Leukoplakia < 2 cm
L2- size of Leukoplakia 2-4 cm
L3- size of Leukoplakia > 4cm
Lx- size not specified
P- Pathology
P0- no epithelial dysplasia
P1- distinct epithelial Dysplasia
Px-dysplasia not specified in the pathology report
Oral Leukoplakia staging system:
Stage I- L1 P0
Stage II- L2 P0
Stage III- L3 P0, L3 P1
Stage IV- L3 P1

7. According to Burket's, (2002)[13]
Homogenous Leukoplakia
Speckled Leukoplakia (nodular Leukoplakia)
Verrucous Leukoplakia (verruciform Leukoplakia)
Proliferative verrucous Leukoplakia

8. According Axell et al, (1984)[14]
Homogenous Leukoplakia (simplex): a uniform whitish lesion with a smooth or corrugated surface.
Erythro-Leukoplakia (erosive Leukoplakia): a whitish lesion that includes red areas.
Nodular Leukoplakia: a lesion with slightly raised, rounded, red and whitish excrescences that may be described as granules or nodules.
Verrucous Leukoplakia: an exophytic lesion with irregular sharp or blunt projections.

9. According to Banoczy et al, (1982)[14]
Type I or Leukoplakia simplex: keratinized mucosa with flat or very slightly elevated white lesions. It corresponds to homogenous type of Leukoplakia.
Type II or Leukoplakia verrucosa: verrucous proliferation raised above the mucosal surface.
Type III or Leukoplakia erosive: white lesion with erythematous area or erosion, and fissures. In an international seminar on oral Leukoplakia. Lesion is slightly raised, rounded, red and or whitish excrescence that may be described as granular or nodules.

10. According to WHO, (1980)[8]
1. Homogeneous Leukoplakia
Lesions those are uniformly white.
2. Non homogeneous Leukoplakia
Lesion in which part of the lesion is white and appears reddened. The lesion is well demarcated raised white areas, with red areas.

11. According to Mehta et al, (1971)[14]
Homogenous Leukoplakia: characterized by a raised plaque formation consisting of a plaque or groups of plaques varying in size with irregular edges. The lesions are predominantly white but can be greyish white or yellow

Ulcerated Leukoplakia: characterized by a red area at times with yellowish area of fibrin, giving the appearance of ulceration.
Nodular (speckled) Leukoplakia: characterized by white patches on an erythematous base.

Clinical Presentation[1,3,8,12-14]
Sex and Age distribution- Males are more frequently affected than Females. It occurs at any age group, more commonly seen in 35-45 years of age.
Site- It can occur anywhere in oral mucosa. But most common site is buccal mucosa and commissures. Tongue, edentulous alveolar ridge mucosa, palatal mucosa, lips are also involved in descending order.
Symptoms- Oral Leukoplakia is an asymptomatic lesion, sometimes burning sensation and thickness of oral mucosa is the common symptom.
Clinical features-
Homogenous type- white, non scrapable often well demarcated plaque with an identical pattern throughout entire lesion, surface texture may vary from smooth and thin to a leathery thick appearance with surface fissures referred as *cracked mud* or *ebbing tide appearance.*
Non homogenous type- white patch or plaque intermingled with red elements. If the surface texture is homogenous but contains verrucous, papillary or exophytic components, this type is also consider as non-homogenous type.
Speckled: mixed, white and red, but retaining predominantly white character.
Nodular: small polypoid outgrowths, rounded red or white excrescences.
Verrucous: wrinkled or corrugated surface appearance.
Proliferative verrucous: presents with multiple, simultaneous Leukoplakia.

Diagnostic Procedures for oral leukoplakia:
Conventional oral examination (COE) - A conventional oral examination (COE), using normal (incandescent) light, has long been the standard method for oral Pre cancer and cancer screening.[15,16,17]
Toluidine blue staining - Toluidine blue (TB) is an acidophilic metachromatic dye with a high affinity for acidic tissue components. It stains tissues rich in nucleic acids and acidic tissue components like sulphates, carbohydrate and phosphate radicals and is useful adjuvant in screening of Oral Leukoplakia.[18]
Lugol's iodine - Lugol's iodine staining is the iodine reacts with glycogen in the cytoplasm. Petruzzi et al. (2010) observed that when Iodine is applied on the suspicious lesions, normal mucosa stains brown or mahogany due to its high glycogen content, while dysplastic and cancer lesions do not stain, and appear pale compared with the surrounding tissue.[19]
Chemiluminiscence- refers to the emission of light from a chemical reaction which is of varying degrees of intensity with colours that span the visual spectrum. The relevant technology (ViziLite system), involves the use of an oral rinse with a 1% acetic acid solution for 1 min followed by the examination of the oral mucosa under diffuse chemiluminescent blue / white light (wavelength of 490-510 nm). In this screening method, normal mucosa appears blue, whereas abnormal mucosa appears more aceto white with brighter, sharper and more distinct margins.[16,20]
VELscope – The VELscope is a hand held device that can enhance the visibility of oral mucosal abnormalities by activating tissue Autofluorescence.[16,21]
Exfoliative Cytology - Exfoliative Cytology is the microscopic examination of shed cells from an epithelium surface. It is recommended as a diagnostic aid for studying the malignant potential especially of oral leukoplakia lesions.[16]

Biopsy - Biopsy is surgical removal of tissue from a living organism for the purpose of microscopic or bacterial examination or chemical analysis for obtaining the diagnosis. Useful to confirm the dysplastic changes in Oral leukoplakia, especially the speckled leukoplakia and erythro-leukoplakia.[22,23]
p 53 marker – is also used as biomarker for detecting dysplastic and malignant changes in Oral Leukoplakia. [16,17]

Histological Feature:
Leukoplakia can divided as benign hyperkeratosis, mild dysplasia, moderate dysplasia, severe dysplasia or invasive carcinomas microscopically. *Mild dysplasia* histologically shows hyperplasia of cells of the basal layers, cytological atypia with mild pleomorphism of cells and nuclei, and minimal architectural changes. Moderate dysplasia histologically shows proliferation of atypical cells in epithelium with prominent cytological changes. Hyperchromatism and cellular pleomorphism seen. Loss of polarity of basal cells. In *severe dysplasia*, abnormal proliferation of cells is seen from basal layer to upper third of epithelium. There is marked pleomorphism of cells with changes in nuclear- cytoplasmic ratio. Architectural changes are severe, with loss of stratification. In *carcinoma-in-situ*, the cytological and architectural changes are clearly evident throughout whole epithelium.[8,12,14,24]

Differential Diagnosis:[1,25]

Disorder	Diagnostic feature
White sponge nevus	Noted in early life, family history, large areas involved, genital mucosa may be affected
Frictional keratosis	History of trauma, mostly along the occlusal plane, an etiological cause apparent, mostly reversible on removing the cause
Chemical injury	Known history, site of lesion corresponds to chemical injury, painful, resolves rapidly
Pseudo membranous Candidiasis	The membrane can be scraped off leaving an erythematous/raw surface
Leukoedema	Bilateral on buccal mucosa, could be made to disappear on stretching (retracting), racial
Lichen planus (plaque type)	Other forms of lichen planus (reticular) found in association
Lichenoid reaction	Drug history, e.g. Close to an amalgam restoration
Discoid lupus erythematosus	Circumscribed lesion with central erythema, white lines Radiating
Skin graft	Known history
Hairy Leukoplakia	Bilateral tongue keratosis specific histopathology with koilocytosis
Leukokeratosis Nicotina palate	Smoking history, greyish white palate

Management:
Elimination of etiologic factor –
Discontinuation of the substance abuse, through counselling & advocacy.
Non pharmacological management -
Increase intake of fresh green leafy vegetables and red fruits.
Regular eating of black dates and beetroot.
Maintain Good Oral Hygiene
Pharmacological Management
Carotenoids [26]

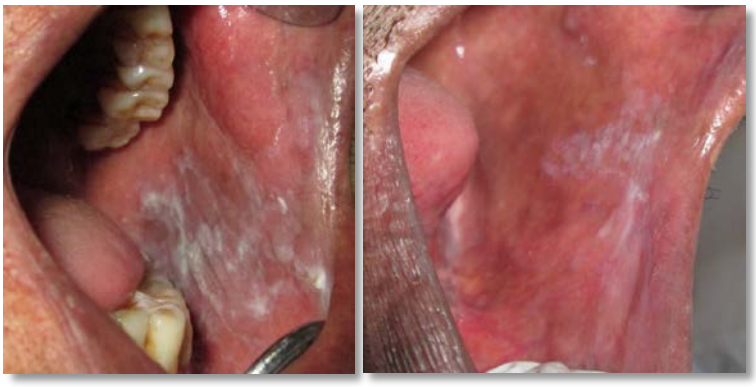

Figure- 1: Fig. 1A and 1B shows Extensive, Diffused white lesion on left buccal mucosa suggestive of Homogenous Leukoplakia

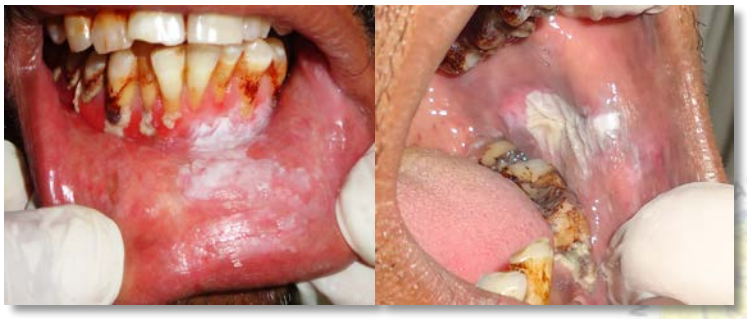

Figure- 2: Fig. 2A shows Localized white lesion on Lower labial mucosa and 2B shows Localized White lesion on left buccal mucosa suggestive of Homogenous Leukoplakia

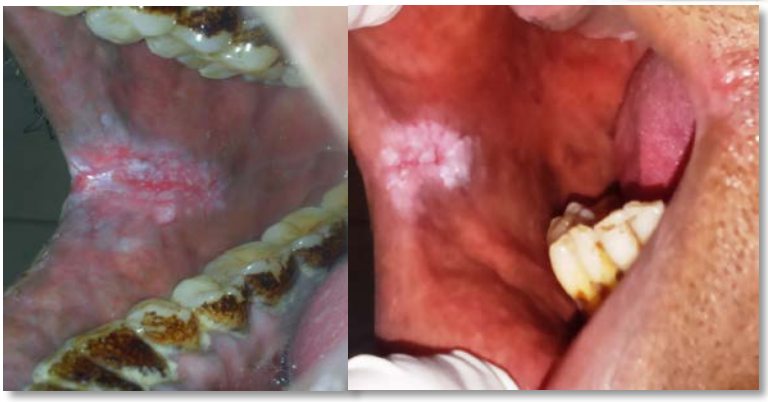

Figure- 3: Fig. 3A and 3B shows Localized Red and white lesion on right Commissure suggestive of Non homogenous Leukoplakia

Beta-Carotene

The beta-carotene is an antioxidant and is used to prevent oral Leukoplakia and possibly oral cancer.

Rich source of beta carotene are Carrot, Sweet potatoes, dark leafy greens, tomatoes, **cantaloupe melon, squash, sweet red peppers, dried apricots, pias and brocholi**. Beta carotene has protective effects against cancer because of its anti-oxidizing action. A ligation between beta-carotene and oxygen, which is an unstable reactive molecule, which diminishes the damaging effects of free radicals. The daily requirement - 20 to 90mg/day of beta-carotene in time periods from 3 to 12 months. [26]

Garewal et al (1999)[26] observed that β carotene, 60 mg/d, for 6 months produces sustained remissions in patients with oral Leukoplakia.[29]

Sankaranarayanan et al (1997)[26] observed that systemic beta-carotene 360 mg/ day for 12 month gives resolution in clinical symptoms in 55 % cases of Oral Leukoplakia. They also observed that malignant transformation is not reported.

Tome et al (1992)[26] observed that systemic β carotene, 90 mg/day, in 23 patients for 7 months resulted in clinical resolution of Oral Leukoplakia in 26% of the cases.

Garewal et al (1990)[26] observed that systemic β carotene, 30 mg/day, in 24 patient for 6 months resulted in clinical resolution of symptoms of oral Leukoplakia in 8% of the cases. They also observed that their 8% of the patients showed malignant transformation.

Lycopene

Lycopene is a carotenoid without pro vitamin A action. It is considered as one of the most efficient biological antioxidant. It is useful in reduction in the development of degenerative diseases caused by free radicals, such as Cancer. [26]

Lycopene is a bright red Carotene and Carotenoid pigment. It is a phytochemical found in tomatoes and other red fruits and vegetables, such as red carrots, red bell peppers, watermelons, and papayas (but not strawberries or cherries). The daily dose requirement is 3 to 10 mg/day, once a day for 3 months. [26]

Patel et al, (2014)[26] observed that Lycopene (3mg), Vitamin E (200 IU) and Selenium (100 mcg) in treatment of Oral Leukoplakia gives significant improvements both clinically and histologically symptoms.

Win Pa Aung et al, (2013)[26] observed that 10 mg Vitamin A along with 500 mg Lycopene twice a day improves symptoms of Oral Leukoplakia.

Singh et al (2004)[26] conducted a study to evaluate and compare Efficacy of Lycopene in treatment of Oral Leukoplakia. They performed study on 58 cases of Oral Leukoplakia given Lycopene 8mg/day, 4mg/day, and Placebo for three months. They observed that Lycopene reduces hyperkeratosis and reduce size of the lesion in 80% of the cases with 8mg/day Lycopene compared to 4mg/day.

Vitamins

Vitamin A:

Carotene is a yellow pigment found in vegetable foods. It gets converted into fat soluble Vitamin A. Vitamin A is a group of fat-soluble Retinoids, including retinol, retinal, and retinyl esters. It is involved in immune function, vision, reproduction, and cellular communication. Retinoic acid is obtained from carotene and animal products such as meat, milk, fish and eggs, which, while in the intestine, are converted, respectively, into retinal and retinol. The daily Requirement is 350 - 600mcg/day. 13-cis retinoic acid is the retinoid recommended for Oral Leukoplakia treatment. [26]

Epstein et al (1999)[26] observed that topical 0.05% Vitamin A (tretinoin) acid gel 4 times a day resulted partial response in 14 patient and complete response in 3 patient of oral Leukoplakia.

Stich et al (1988) [26] observed that carotene 180 mg/week + Vitamin A 100.000 IU / week for 6 month shows 27.5 % remission of oral Leukoplakia.
Hong et al (1986)[26] observed that oral Leukoplakia treated with 13-cis-retinoic acid 1 to 2 mg per kilogram of body weight per day for 3 month shows 67% of clinical resolution in symptoms of oral Leukoplakia.
Shah et al, (1983)[26] observed that Topical Vitamin A 1-5mg /day for 11 month gives clinical resolution of oral Leukoplakia.
This treatment was not widely accepted due to its side effect. Hyper vitaminosis, toxicity, teratogenic effects and alterations in various organic systems.

Fenretinide

Fenretinide (4-HPR) or N-4- hydroxyphenyl retinamide is a Vitamin A analogue that was synthesized in the United States during the late 1960s. A characteristic feature of 4-HPR is its ability to inhibit cell growth through the induction of apoptosis with mechanisms that may be both receptor-dependent and receptor-independent. Chemo-preventive efficacy of Fenretinide has been investigated in clinical trials targeted at different organs.[26]
Lippman (2006) conducted the study to evaluate and compare efficacy of 4-HRP in Oral Leukoplakia. They observed that systemic 4 HRP 200mg/day for 9 month resulted clinical resolution in Oral Leukoplakia.
Fausto chiesa et al (2005) conducted the study to evaluate and compare efficacy of 4-HRP in Oral Leukoplakia. They performed study on patient with Oral Leukoplakia and gave them 4-HRP 200 mg/day [100 mg twice a day] for 12 months. They observed resolution in oral leukoplakia symptoms.

L-Ascorbic Acid (Vitamin C)
L-ascorbic acid also called Vitamin C, a water soluble Vitamin, is found in citrus fruits such as Kiwi, Strawberries, Papaya, and Mango. The daily requirement 100–120 mg/per day for adults. It has been suggested that a daily intake of atleast 140 mg/day is required for smokers because they usually present a reduction of the L-ascorbic acid concentration in serum leukocytes.[26]
Ascorbic acid has Antioxidant properties and reacts with superoxide produced as a result of the cells normal metabolic processes; this inactivation of superoxide inhibits the formation of nitrosamines during protein digestion and helps avoid damage to DNA and cellular proteins. There are NO studies regarding the efficacy of Ascorbic acid in Oral Leukoplakia treatment. [26]

Beta-Tocoferol (Vitamin E)

Beta -Tocoferol is the commonest and most active form of Vitamin E. It is found in Plant oil, Margarine and Green leaves. The Daily requirement is 10 mg/day for adult men and 8 mg/day for adult women. Its absorption rate is reduced when consumption exceeds 30mg/day.[26]
Benner et al (1993)[27] conducted a study to evaluate the toxicity and efficacy of tocoferol in oral Leukoplakia. They performed study in 43 patients with oral Leukoplakia in use of 400 IU twice daily for 24 weeks. Follow-up was performed at 6, 12, and 24 weeks. They observed that 10 patients (23%) had complete clinical remission of lesion and 10 (23%) had a partial clinical response. Nine (21%) had histologic responses (complete reversal of dysplasia to normal epithelium).

Bleomycin:
Bleomycin with iontophoresis has been studied in the treatment of Leukoplakia and Papilloma of the head and neck region.
Wong et al (1989) observed that complete resolution of hyperkeratotic Leukoplakia with atypia using local injections of 5 mg of Bleomycin weekly for eight weeks.

Green tea:
Green tea and its major polyphenol constituents shown to have many health benefits including cancer prevention. Tea catechins and Tea catechin etabolites / catabolites are bio-available in the systemic circulation after oral intake of green tea or green tea catechins. Tea pigments are the oxidized product of 40% green tea polyphenols and are composed primarily of theaflavins and thearubigins. Applying the tea extracts directly to the lesions may help improve the local concentrations of the active constituents.[26]
Li N et al (1999) observed that 760 mg of mixed tea capsules along with mixed tea ointment topically shows resolution in symptoms of oral leukoplakia within 6 months verses placebo group (Topical glycerine).

Spirulina:
The Blue green microalgae Spirulina, used in daily diets by natives of Africa and America, have been found to be a rich natural source of proteins, carotenoids, and other micronutrients. Spirulina algae inhibits the process of oral carcinogenesis.[26]
Mathew, et al. (1995) conducted a study to evaluate and compare the chemo preventive activity of Spirulina fusiformis (1g/day for 12 months) in reversing oral Leukoplakia. They observed that complete regression was seen 57% subjects with homogeneous Leukoplakia.

Surgical Management :
Photodynamic Therapy
Photodynamic therapy (PDT) is a non-invasive method for the treatment of premalignant lesions and head and neck cancers. The principle of PDT is a non-thermal photochemical reaction, which requires the simultaneous presence of a photosensitizing drug (photo sensitizer), oxygen, and visible light. Mainly, the light source consists of a portable diode laser and the light is transmitted via laser fibres to or into the tumour.[26]
Illumination of the tumour by light at the activating wavelength results in the destruction of cells by a non-free radical oxidative process. These reactive oxygen species may damage crucial cell components, such as structural proteins, enzymes, DNA, and phospholipids. PDT is a cold photochemical reaction. The photosensitizing agents are of inherently low systemic toxicity. PDT damage heals mainly by regeneration rather than scarring. Due to the organ preserving principle of PDT, important structures are maintained with good functional and cosmetic outcome. Only for very superficial skin lesions or premalignant lesions of the oral mucosa, the 5-Aminolaevulinic acid can be applied topically. For all other indications intravenous application is mandatory. [26]

Carbon dioxide Laser:
Since 1970, studies have shown that the CO2 laser is an effective instrument for the treatment of premalignant lesions of the oral mucosa. Soft tissues can be superficially removed by evaporation with minimal thermal damage to adjacent tissue, which results in minimal tendency to scar and little postoperative pain and oedema. CO2 laser can be used for selective removal of epithelium. Therefore this modality is very suitable for the treatment of intraepithelial lesions like oral Leukoplakia.[26]
Broun's et al (2007)[27] conducted a study to evaluate the treatment results of co2 laser vaporisation in patients with oral Leukoplakia (OL). They observed that 14/35 patients, there was a recurrence between 1 and 43 months (mean 18.7 months), the annual recurrence rate being approximately 8%. In all together 5 of 35 patients, malignant transformation occurred in a mean period of 54 months.
Regular follow up-
1- 2 years of Regular follow up is mandatory to see the regression of lesion and symptoms if any. The follow up will help in knowing whether patient has discontinued habits or not, as leukoplakia

holds a high chance of converting into a malignant lesion. Patient will be asked or trained for self-examination regularly.

Conclusion:

Oral Leukoplakia is a potentially malignant disorder of oral cavity. The present review article has explored all the definitions, classification, clinical features and treatment options available in the existing literature and has suggested the treatment modalities based on evidence. The key objective in the management of oral leukoplakia is to prevent malignant transformation. The cessation of tobacco and arecanut related habit is the most important step. The health education, counselling the individual, and behavioural therapies are most essential methods of prevention at a primary level. The degree of dysplasia will guide the choice of treatment. Oral leukoplakia presenting with high malignant risk, may be removed, based on the location and size of the lesion. Thus, a complete knowledge from etiology of the lesion to diagnosis and delivery of suitable treatment will help in preventing the leukoplakia from becoming malignant.

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