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# Potentially Malignant Oral Disorders

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## Abstract

Most cancerous lesions are derived from potentially malignant oral disorders (PMOD). The World Health Organization (WHO) points out the following lesions as the main PMOD: leukoplakia, erythroplakia, actinic cheilitis, submucous fibrosis, and lichen planus. Leukoplakias are white plaques or spots that cannot be removed by scraping, and these lesions aren't characterized clinically or pathologically like any other diseases. Erythroplakias are red lesions of the oral mucosa that also cannot be characterized clinically or pathologically as another definable disease. Actinic cheilitis is an injury that affects the vermilion of the lower lip and has this anatomical location due to its etiological factor, which is the progressive and excessive exposure to ultraviolet rays of sunlight. Submucous fibrosis is a chronic disease of the mouth that presents as an inflammatory subepithelial reaction, followed by an alteration in the submucous fibroelastic tissue. Lichen planus is a dermatological disease characterized by white patches or striations, symmetrical and bilateral, and its treatment is basically done with topical corticosteroids.

**Keywords:** potentially malignant oral disorders, leukoplakia, erythroplasia, actinic cheilitis, lichen planus

## 1. Introduction

Head and neck cancer is a worldwide public health problem, and according to the International Agency for Research on Cancer (IARC) in 2018, 1,454,892 new cases of head and neck cancer worldwide have been estimated. When all the sites involving the head and neck region added, these tumors occupy the third place, behind only the lung tumors (2,093,876) and the breast (2,088,849) [1]. By analyzing the sexes separately, head and neck tumors are the fourth most common cause of cancer in men (796,946 cases), behind lung, prostate, and colorectal cancer. In women, they are also the fourth most common cause (657,966 cases), behind breast, colorectal, and lung cancer, and thyroid tumors are the most frequent in this population (436,344 cases). In Brazil, according to the National Cancer Institute (INCA), there is an estimated 11,200 new cases of cancer of the oral cavity in men and 3,500 in women for each year of the 2018–2019 biennium, placing this neoplasm in fifth place in the prevalence [2].

The incidence can change by region of the world. In developing countries, in men, lip and oral cavity cancer alone is the third in incidence, partly because of the high disease rate in India, which accounts for 36% of the population of countries with low human development index (IDH) [2].

Understanding the world statistics on cancer, more specifically on head and neck cancer, is essential in order to propose measures of prevention and early diagnosis, such as anti-smoking policies, HPV vaccination, and improvement of the oral health and diet of the population. Such measures would have a significant impact on the incidence and mortality of this disease [2].

Among the risk factors, potentially malignant oral disorders have a prominence, since they are generally the first indication of the disease [3]. The World Health Organization (WHO), in its latest publication, has defined PMSD as clinical presentations that carry a risk of developing oral cavity cancer, a clinically defined precursor lesion or clinically normal oral mucosa [4]. The WHO identifies as PMOD the following disorders: erythroplakia, erythroleukoplakia, leukoplakia, oral submucous fibrosis (OSF), congenital dyskeratosis, smokeless tobacco keratosis, palatine lesions associated with reverse smoking, chronic candidiasis, lichen planus, discoid lupus erythematosus, glossitis syphilitic, and actinic cheilitis. In this chapter, we will discuss the most common PMODs with the highest potential for malignant transformation, which are leukoplakias, erythroplasias, oral lichen planus, actinic cheilitis and oral submucous fibrosis [1, 4].

## 2. Leukoplakia

According to the WHO, leukoplakia is defined as a white, variable-risk plaque, excluding (other) known diseases or disorders that do not carry an increased risk of cancer; therefore, the nomenclature is restricted only to the clinical aspect and histological changes. Due to the fact that its clinical diagnosis is basically made by exclusion, this disorder makes a differential diagnosis with other well-known lesions and with very similar clinical characteristics such as pseudomembranous candidiasis, Lichen planus, leukoedema, and lupus erythematosus [5]. The specific causative factors of leukoplakia are still unknown; however, it is known that the smoking habit is closely linked to the progression of leukoplakia. In addition, other risk factors have been associated with the development of this disorder, the consumption of alcohol that would act synergistically with tobacco, trauma, as well as infestations of microorganisms such as human papillomavirus (HPV) [6].

Oral leukoplakia is the most common PMOD, presenting a prevalence of 1% and an annual malignant transformation risk of 2%. It is found in equal proportion between men and women, rarely occurs in the first two decades of life, and is more prevalent among individuals, and this is their main etiological factor. The anatomical sites in which about 70% of the leukoplakia are found are jugal mucosa, gingiva, and vermillion of the lip; however, lesions located on the tongue and floor of the mouth contribute to over 90% of the cases that present some level of dysplasia or even a carcinoma [6–8].

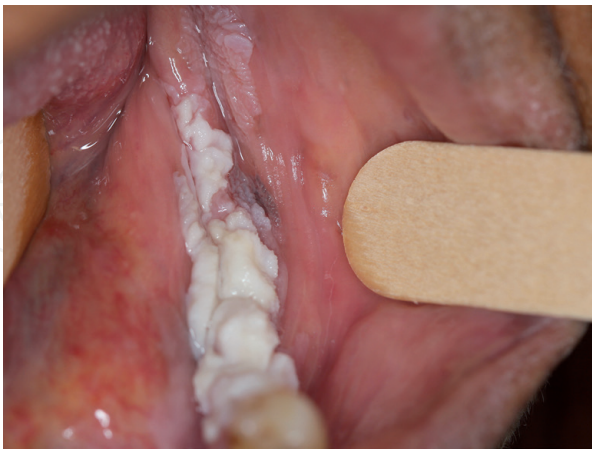
Clinically the leukoplakias are subdivided into homogeneous and nonhomogeneous. Homogeneous leukoplakias are characterized as uniformly flat and thin lesions that have a low percentage of malignant transformation, as well as spontaneous regression after elimination of risk factors, especially smoking habit (**Figure 1**) [3, 9]. Nonhomogeneous leukoplakias are described as white and red lesions (erythroleukoplakias), which may appear irregularly flat or nodular, and are subdivided into a variety of subtypes, such as erythematous or speckled, nodular and verrucous (**Figure 2**). Verrucous leukoplakia, one of the most misdiagnosed subtypes of non-homogeneous leukoplakia because of its challenging clinical appearance, although presenting as a uniform white lesion, its verrucous texture is the characteristic that differentiates it from homogeneous leukoplakia (**Figure 3**) [10]. Proliferative



**Figure 1.**  
*Homogeneous leukoplakia on the floor of the mouth. Source: author's file.*



**Figure 2.**  
*Nonhomogeneous leukoplakia on the back and lateral edge of the tongue. Source: author's file.*



**Figure 3.**  
*Exuberant verrucous leukoplakia in the alveolar ridge. Source: author's file.*

verrucous leukoplakia (PVL) is a very rare form and falls between nonhomogeneous leukoplakias and is a subtype of verrucous leukoplakia. It's a distinct, multifocal, progressive course associated with high rates of recurrence and malignant transformation [4]. PVL mainly affects middle-aged women with no harmful habits such as smoking and presents clinically as a diffuse and homogeneous white





**Figure 4.**  
*Proliferative verrucous leukoplakia affecting the hard palate and alveolar ridge. Source: author's file.*

plaque at the onset, which gradually becomes erythematous and exophytic with the progression of the disorder, affecting mainly the gingiva, alveolar mucosa, and palate (**Figure 4**) [4, 6, 11].

Regarding the histopathological characteristics of leukoplakia, a thick layer of keratin in the epithelium (hyperkeratosis) is present, with or without thickening of the thorny layer (acanthosis). It is still possible to observe in some leukoplakia the presence of hyperkeratosis with epithelial atrophy. Generally, leukoplastic lesions do not exhibit epithelial dysplasias, but the presence of them would be a worrying sign for a possible malignant transformation, a fact that is most observed among nonhomogeneous leukoplakias [4, 6].

The diagnosis of leukoplakia is basically by excluding other diseases or disorders that do not carry increased risk of malignant transformation. In order to perform an accurate diagnosis of leukoplakia, different levels of leukoplakia must be followed by certainties (factor C) that lead us from a primary clinical diagnosis to the definitive diagnosis based on the histopathological examination of the lesion [4, 6].

Thus, in the van der Waal factor, C1 evidence is obtained in a single visit, in the first contact between the dental surgeon and patient, applying only palpation and inspection as the primary means for diagnosis, in addition to anamnesis to collect data that may make up this provisional clinical diagnosis. In the certainty factor C2, evidence is obtained from negative results of elimination of etiological factors such as mechanical irritation, during a period of follow-up of 2–4 weeks or in the absence of any suspicious etiological factors (definitive clinical diagnosis). Factor C3 is similar to C2 but complemented by incisional biopsy (provisional histopathological diagnosis), and C4 is the evidence obtained from surgical excision of the lesion followed by histopathological examination of the resected specimen (definitive histopathological diagnosis) [11, 12]. Performing biopsy in the diagnosis of leukoplakia is important because only through this examination it is possible to determine whether to perform the histopathological diagnosis or not of epithelial dysplasias, thus guiding the treatment [12].

The treatment of leukoplastic lesions is dependent on the result found in the histopathological examination; in this way, the treatment plan is often individualized according to the histological findings, such as the degree of dysplasia found in the epithelium. The WHO in its latest manual for the classification of head and neck tumors (2017) defines dysplasias as architectural and cytological epithelial changes caused by an accumulation of genetic alterations associated with an increased risk of progression to squamous cell carcinoma. Therefore, in lesions that present mild dysplasia or do not present dysplasia, more conservative measures should be taken,

such as clinical follow-up of the lesion every 6 months throughout life, evaluation of the need for new biopsies, and end of smoking [3, 10]. In leukoplakias that present moderate or severe dysplasia/carcinoma in situ, it is recommended that the lesion be removed completely, if possible, or by CO2 laser therapy. However, even with the surgical treatment, there appears to be no reduction in the risk of developing a carcinoma or even relapse of the leukoplakial lesion [6, 7, 10].

Regarding the prognosis of leukoplastic lesions, recurrence rates after any type of treatment can range from almost 0 to 30%, which means regular follow-up of patients every 6 months [10].

### 3. Erythroplakia

Erythroplakia is defined as “a red spot that can’t be characterized clinically or pathologically like any other definable disease” [4]. When a mixture of red and white changes occurs, this lesion would be classified as a nonhomogeneous leukoplakia called erythroleukoplakia [10]. Erythroplakia is multifactorial, since no isolated etiological factor has been evident, but several intrinsic and extrinsic etiological factors have contributed to the origin of this disorder, such as smoking, alcohol consumption, candida infection, and even nutritional deficiencies such as iron and vitamin A deficiency [6].

Erythroplakia in comparison to leukoplastic lesions is rare and has a prevalence rate in South and Southeast Asia ranging from 0.02 to 0.83% but presents a high percentage of malignant transformation ranging from 14 to 50%, and about 90% of cases already present moderate or severe dysplasia/carcinoma in situ. Because of the high rates of malignant transformation and the presence of high-grade dysplasias, many specialists have already considered it a primordial clinical sign of squamous cell carcinoma. It is a prevalent disorder in middle-aged adults in the elderly, aged between 45 and 74 years, with no prevalence among the genders [6, 10, 13].

Clinically, erythroplakia presents as a well delimited, asymptomatic, reddish, smooth and shiny stain or plaque with a soft and velvety texture [3, 6]. If hardened areas are observed in the lesion, it is already indicative of the presence of a possible invasive carcinoma at the site. The preferred anatomical location is the floor of the mouth, but it can be observed anywhere in the oral cavity, such as the lip, hard palate, or oral mucosa [3]. The clinical presentation of a solitary lesion is consistently useful to clinically differentiate erythroplakia from erosive lichen planus, lupus erythematosus, and erythematous candidiasis, as these lesions always appear bilaterally and are more or less symmetrical (**Figure 5**) [10].



**Figure 5.**  
*Erythroplakia affecting palate and superior alveolar ridge. Source: author's file.*

Histopathologically, 90% of erythroplakia present as severe epithelial dysplasias/carcinomas in situ or squamous cell carcinomas. The epithelium will show no production of keratin and is regularly atrophic. This absence of keratin associated with epithelial atrophy allows the underlying microvasculature to be exposed, thereby elucidating the reddish coloration of the lesion. In relation to connective tissue, it regularly exposes chronic inflammation [9].

The diagnosis of erythroplakia, as well as leukoplakias, is made by exclusion. This disorder presents clinically very similar to other lesions commonly found in the oral cavity, such as vascular lesions, candidiasis, mucosites, and even Kaposi's sarcoma. Because it has so many options for differential diagnosis, as in leukoplakia, one can use the steps or factors of analysis guided by Isaac van der Waal (factors C1, C2, C3, and C4) [10]. In addition, lesions on the floor of the mouth and belly regions and lateral border of tongue should be biopsied, since in some anatomical locations, the highest rates of malignant transformation occur and the presence of high degree dysplasia. With the accomplishment of the biopsy, for diagnostic purposes, it will be possible to verify the presence or absence of dysplasias. According to Neville et al. [6], 90% of the erythroplakia already present severe epithelial dysplasias/carcinomas in situ [9].

As in leukoplakias, the treatment plan for erythroplakia is guided by the definitive diagnosis obtained only after the histopathological examination. In the absence of dysplasia or presence of mild dysplasias, the lesion is monitored every 6 months, and if there is any change, perform a biopsy to check if any dysplastic modification has occurred. In lesions presenting moderate to severe dysplasia/carcinoma in situ, complete removal of the lesion should be done with safety margin. As with leukoplakia, total excision of the lesion does not guarantee that there is no recurrence of erythroplakia; in addition to the fact that this disorder already has high levels of malignant transformation, its removal does not exclude the likelihood of future cancerous lesions on the site or in other oral locations. Something that should be very clear regarding the treatment of erythroplakia and other PMOD is that the patient who has one of these disorders will never be medically released, as these must be followed for life to assess whether or not there was any dysplastic or even the appearance of cancerous lesions in other oral sites [3, 6, 10].

#### 4. Oral lichen planus

Oral lichen planus is a chronic and systemic mucocutaneous disease often found in the oral cavity, but it can also affect other body parts such as the skin, nails, scalp, and vaginal mucosa. The British physician, Erasmus Wilson, in 1869, was the first to describe lichen planus, and he believed that the cause of this disorder would be fungal infections [6, 14]. Thus, the pathophysiology of OLP for years has been a mystery, but it is known that this disorder occurs due to T-cell-mediated autoimmune destruction of the basal cells of the epithelium. Recently it was considered a PMOD, after several discussions among scholars, due to the fact that the lesion shows a low degree of malignant transformation, around 0.5% [13, 14].

The etiological factors for this disorder are still unknown, but it is believed to be related to stress, anxiety, diabetes, autoimmune diseases, and genetic predisposition [15]. Stress and anxiety may not have total influence on the pathogenesis of lichen planus, but it has been observed that patients with this disorder are usually subjected to high levels of stress [6].



Oral lichen planus affects between 0.5 and 2% of the population, having a predilection for women between the ages of 30 and 60 years, being a rare disorder in children [6, 15]. The main intraoral sites of lichen planus are the jugal mucosa, tongue, and gingiva. An important feature of this lesion is bilaterality and symmetry [14].

Clinically, oral lichen planus is characterized by six distinct forms: reticular, erosive, bullous, plaque, papular, and atrophic, with reticular and erosive forms being the most prevalent. The reticular OLP is routinely present in the posterior jugal mucosa bilaterally. Other anatomical areas may be affected, such as the lateral border and back of the tongue, gingiva, palate, and vermilion lips [6, 15]. This type of OLP is much more common than erosive, but the latter is the most studied because it is symptomatic, which leads more patients to seek treatment specialists [6]. The reticular type is thus defined by its appearance of intertwined and asymptomatic white striations, the pathognomonic sign of the disorder being the Wickham striae (**Figure 6**). In the erosive type, erythematous and atrophic areas are observed, with varying degrees of central ulceration, and at the periphery of the atrophic regions, fine irradiated white streaks are usually observed (**Figure 7**) [6, 14, 15]. If the erosive state is aggravated, a separation between the epithelium and the underlying connective tissue may occur, resulting in a rare clinical presentation of oral bullous lichen planus [6].



**Figure 6.**  
*Lichen planus on jugal mucosa, showing Wickham striations. Source: author's file.*



**Figure 7.**  
*Erosive lichen planus on jugal mucosa. Source: author's file.*



Lichen planus has typical histopathological characteristics, but they are not specific for the lesion. Its epithelium has varying degrees of orthokeratosis and parakeratosis, and depending on whether the lesion is reticular or erosive, the thickness of the thorny layer may vary. Epithelial ridges may be absent, atrophic, or hyperplastic but usually exhibit sharp, serrate-like progressions. Another striking feature is the presence of hydropic degeneration, that is, the destruction of the basal cell layer of the epithelium and an intense infiltration of banded inflammatory cells predominantly composed of T lymphocytes. Some lesions of lichen planus may show some degree of dysplasia, being able to present aberrant mitoses and nuclear and cellular pleomorphisms, among other dysplastic alterations [6, 16].

The diagnosis of OLP is basically made by clinical findings, mainly in the reticular type, by the presence of the pathognomonic signal (Wickham striae). In addition to the clinical diagnosis, the histopathological examination may be requested for a definitive diagnosis. One thing that can make it difficult to diagnose OLP is the existence of candidiasis overlapping with lichen lesion, and for this, it is recommended that the treatment for candidiasis be carried out first and only subsequently the definitive diagnosis of OLP and the respective treatment plan of the same [6, 15].

As the reticular type does not present symptoms, there is no need for specific treatment, but as already mentioned, candidiasis can occur overlapping with lesions of lichen planus; in this way it is proposed that the antifungal treatment be performed based on topical nystatin, and mouthwash with nystatin or application of Miconazole gel is recommended. In erosive lichen planus, because it has painful symptomatology, treatment with topical corticosteroids initially, such as triamcinolone acetonide and beclomethasone, is suggested. The second line of treatment would be the use of retinoids, cyclosporine, and calcineurin inhibitors prescribed for about 2 weeks. In addition to drug treatment, photodynamic therapy is usually used to relieve symptoms [6, 13]. Lastly, patients with this disorder should be evaluated periodically for 3–6 months, especially in atypical cases with some degree of dysplasia [6].

## 5. Queilite actinic

Actinic cheilitis is a PMOD that frequently presents in vermillion of the lower lip, attributed to modifications in the keratinocytes of the labial mucosa. The expression cheilitis was first used in 1923, meaning inflammation on the lips, being multicausal, which include prolonged exposure to solar UV rays, allergic reactions, and systemic diseases. The term actinic refers to changes generated by radiant energy [6, 17, 18]. The etiopathogenesis of this disorder is multifactorial, but it is undeniable that the main etiological factor associated with actinic cheilitis lesions is prolonged exposure to the sun's rays, with UV radiation and with its wavelength of 200–400 nm acting as a carcinogenic factor, as it can cause cell damage, thereby generating mutations in the DNA and tumor suppressor genes, especially in the p53 gene. Lately, other risk factors have been associated with actinic cheilitis, such as smoking, immunosuppression, chronic lupus, and lichen planus [17, 18].

This disorder occurs in light-skinned individuals exposed for long periods to solar UV rays, which is more common in men and those performing outdoor work, such as rural and construction workers, or have a history of progressive exposure in the sun. The lesions mainly affect individuals in the age range

between 50 and 70 years. As previously mentioned, the primary anatomical site is the lower lip, and this is due to the fact that its epithelium is thinner, has a discrete layer of keratin, has fewer melanocytes, and receives direct radiation. Actinic cheilitis has a malignant transformation rate of 17%, with squamous cell carcinoma growing gradually, and metastasis occurs only in the late stages of the lesion [3, 6, 16, 19].

This disorder develops slowly, and the first noticeable clinical changes are atrophy of the border of vermillion of the lower lip, exhibiting a smooth surface with spots of whitish staining. As the lesion progresses, the margin between the vermillion area and the cutaneous portion of the lip is erased. In more advanced states, rough areas with the presence of ulceration can be observed, in addition to the association with leukoplasic lesions. In these late states, in many cases, clinical signs may already be found that indicate malignant transformation, such as recurrent ulcers which do not heal (**Figure 8**) [3, 6, 17].

Actinic cheilitis can histologically be characterized by the presence of an atrophic stratified epithelium, hyperkeratinization, atrophy, or thickening of the thorny layer and varying degrees of epithelial dysplasia. In the underlying connective tissue, it is possible to observe an infiltration of chronic inflammatory cells and also collagenous bundles exhibiting basophilic changes resulting from the change from an eosinophilic collagen to a basophilic granular material, called solar elastosis [3, 6, 17].

The diagnosis of actinic cheilitis is basically clinical; because it is a very characteristic lesion, patients usually report a nonelastic sensation of the lips, followed by dryness and increase of volume; besides, the accomplishment of the incisional biopsy is mandatory mainly for its high rate of malignant transformation and also to propose a suitable treatment plan for the lesion. In the absence of dysplasias or presence of mild dysplasia, the use of 5-fluorouracil (Efudix®), which can be applied twice a day for 2–4 weeks, is recommended. Cryotherapy, which consists in freezing a tissue area to potentiate cell destruction without damaging the healthy tissues around the lesion, and the use of laser therapy are possible therapeutic alternatives. In the occurrence of moderate or severe dysplasia/carcinomas in situ, a vermillion-ectomy is indicated, the affected vermillion mucosa is removed, and the vermillion reconstruction of the lip occurs from the internal labial mucosa [3, 6, 17, 19]. In addition, all patients with actinic cheilitis should be directed to use sunscreens and other forms of protection against UV rays; thus, in the same way as with other PMODs, individuals who have actinic cheilitis should be monitored routinely throughout life.



**Figure 8.**  
*Actinic cheilitis in the lower lip exhibiting epithelial atrophy and loss of sharpness of the demarcation line between the labial mucosa and the epidermis. Source: author's file.*

## 6. Oral submucous fibrosis

Oral submucous fibrosis is a chronic disorder of the mucosa that lines the upper digestive tract that surrounds the oral cavity, oropharynx, and routinely the upper third of the esophagus, and is often found in individuals living in Southwest Asian countries [6, 13]. Before the etiology of this disorder was considered multifactorial and complex, it is now recognized that its appearance is due to the chewing of areca nut; this sachet is composed of areca palm nut and hydrated lime, sometimes with sweeteners and condiments, wrapped in a betel leaf [6, 20]. Because this custom is common in Southwest Asia, the highest frequency of this disorder is in the population of this region, mainly in India. Its frequency is about 0.5% in the population, and estimates suggest that about 2.5 million people are affected. The predominant age group is between 20 and 40 years, since the rural villagers begin the habit of chewing areca nuts very early and for long periods of time, around 16 hours. Oral submucous fibrosis has a malignant transformation rate between 2.3 and 7.6% [3, 11, 19].

Initially oral submucous fibrosis presents as vesicles and ulcers, often on the hard palate and buccal mucosa. With the progression of the disorder, the patient may present with xerostomia, difficulty in moving the tongue, and decreased elasticity of the oral mucosa, lips, and floor of the mouth, and some patients may report oral burning sensation. On palpation examination, dense fibrous bands may be felt, and a change in the color of the buccal mucosa with whitish and opaque tones. In the final stage, generalized fibrosis of the oral cavity and progressing trismus are observed [14].

Histologically, oral submucous fibrosis is described as a submucous deposition of dense collagenous connective tissue that shows little presence of blood vessels but with large numbers of chronic inflammatory cells. In lesions in soft states, the present epithelial alterations are subepithelial vesicles, whereas in lesions in more advanced states, it is possible to observe epithelial atrophy and hyperkeratosis. In addition, about 10–15% of FSO lesions present some level of dysplasia and even present carcinoma in situ [6].

The diagnosis is based on clinical findings and confirmed by biopsy and subsequent histopathological examination. There is no specific treatment for oral submucous fibrosis, but it is imperative that the diagnosed individual ceases the harmful habits. However, it is important to note that the lesion will not regress only with the removal of the habit, but it will prevent a possible malignant transformation. In patients who have mild or moderate cases of the disorder, treatment with corticosteroids applied to the lesion is recommended in order to reduce the symptoms. For later stages, surgical therapy is the most recommended, aiming to relieve trismus by releasing fibrous tissue through conventional techniques of tissue reconstruction, covering local/regional advancement flaps and microvascular flaps. Other treatment options include iron and multivitamin supplements and intralesional injections of lycopene alone or in combination with steroids, and some studies have shown the efficacy of interferon use for improved mouth opening [6, 11, 14, 19, 20]. Patients with oral submucous fibrosis are at least 19 times more likely to develop squamous cell carcinoma than individuals who do not have squamous cell carcinoma that even after the treatment for the symptomatology of the disease, the patients should be monitored routinely [6].

## 7. Conclusion

Potentially malignant oral disorders are the first indications of micro- and macroscopic alterations of possible malignant transformations, so knowledge about

these lesions is of great importance for specific care and prevention against any type of carcinoma. The transformation of normal mucosa to dysplastic mucosa occurs through a complex set of interactions between the individual's organism and environmental factors. Risk factors involving PMOD such as sun exposure, smoking habits, alcohol ingestion, and infection by microorganisms are issues that need to be addressed in order to better treat, prevent, and reduce malignant transformation rates. Thus, it is suggested that clinicians design educational plans aimed at the prevention of PMOD, as well as possible malignant transformations, in this way, enabling their patients against exposure to the causal factors of the disorders. In summary, the diagnosis and treatment plan for potentially malignant oral disorders are fundamental, since lesions that have high degrees of dysplasia should be treated with surgical procedures and those with no or slight degrees of dysplasia should undergo conservative treatments, such as drug therapy or phototherapy.

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## Conflict of interests

The authors declare that there is no conflict of interest.

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