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The role of the dentist in the diagnosis and early detection of neoplastic lesions based on the example of Proliferative verrucous leukoplakia, classified as an OPMD

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Abstract: Proliferative verrucous leukoplakia (PVL) was first described by Hansen, Olson, and Silverman in 1985 as a form of hyperkeratosis, remained resistant to treatment and was further distinguished by the aggressive nature of the lesions and a high rate of malignant transformation (MT). The OPMDs classification developed in 2020 recognized PVL as a distinct form of multifocal oral leukoplakia, characterized by a progressive clinical course and with changing clinical and histopathological features. According to WHO, the risk of malignant transformation of PVL among OPMDs is approximately 50%. This frequent MT of PVL involves transformation into VC or OSCC, which, according to various sources in the literature, occurs in between 43.87% and 65.8% of cases, and according to some in up to 70% of cases. Some reports even suggest a possible malignant transformation rate of around 100%.
PVL usually occurs in multiple areas of the oral cavity simultaneously and is characterized by slow but continuous progression. The aetiology of PVL and the factors influencing its progression to cancer are unknown. Close monitoring of patients is recommended and, if necessary, biopsies should be performed to assess changes in the appearance, colour, and size of observed lesions, as well as the appearance of subsequent lesions. Early diagnosis, surgical removal, and patient observation are crucial in the treatment of PVL.

Keywords: oral potentially malignant disorders (OPMDs), proliferative verrucous leukoplakia (PVL), malignant transformation (MT), squamous cell carcinoma (OSCC), verrucous carcinoma (VC).

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Introduction

Head and neck cancers (HNCs) are the seventh most common cancer globally. [1] They are diagnosed more often in men than in women, usually around the age of 60, with the incidence of the disease increasing markedly after the age of 45. [2] Almost half of all HNCs are oral cancers, [3] the most frequent form of which is oral squamous cell carcinoma [4] (OSCC), which is the 11th most widespread cancer worldwide, with a 5-year survival rate of 50% to 60%. [5] According to data presented by the Global Cancer Observatory (GCO), in 2022 the global incidence of OSCC exceeded 389,000 cases. [6] It is estimated that in more than half of all cases, the development of OSCC is preceded by oral potentially malignant disorders [5] (OPMDs), [7] which include, among others, proliferative verrucous leukoplakia (PVL). [8, 9]

In their practice, dentists encounter lesions classified as OPMDs. The most common of these is leukoplakia, [8] while PVL is characterized by a high rate of recurrence and malignant transformation [5] it also includes the presence of multiple leukoplakias with variable clinical features. [10]

One important issue that should be considered in connection with PVL is possibility of diagnostic error. [8] In view of this fact, it is worth discussing this topic, reminding dentists of this problem and raising awareness of the challenges related to OPMDs, and PVL in particular, that dentists may encounter in their daily work. This condition can progress unnoticed by the patient, and routine dental check-ups can often detect any disturbing changes. [11] Furthermore, due to the high risk of malignant transformation, early diagnosis is crucial. [12]

Definition and place among OPMDs

PVL was first described by Hansen, Olson, and Silverman in 1985 [13, 14, 15, 16] as a form of hyperkeratosis based on an analysis of 30 cases of patients affected by this condition. A follow-up of patients showed that initially simple hyperkeratosis progressed to multifocal lesions, with erythematous components often observed in the examined tissues. Over time, the lesions became exophytic and verrucous. In some cases, the development of verrucous carcinoma (VC) or OSCC is observed. Hyperkeratosis, described by Hansen *et al.* as PVL, remained resistant to treatment [14] and was further distinguished by the aggressive nature of the lesions [17] and a high rate of malignant transformation (MT). [13, 14]

In 2005, the World Health Organization (WHO) Collaborating Centre for Oral Cancer organized a workshop in the UK. [18] It was during this meeting that a working group of experts first introduced the term potentially malignant disorders (PMDs), [18] nowadays referred to as OPMDs, [8] to define lesions and conditions in the oral cavity with a risk of malignant transformation into cancer. [18] This does not mean that all such lesions will develop into cancer, only that they have an elevated risk of MT. [5] Among other things, the working group came up with a revised definition of oral leukoplakia (OL). Henceforth, WHO describes leukoplakia as any white plaque of questionable risk after first excluding (other) known diseases or disorders characterised by no increased risk of cancer. [5, 18] The working group also introduced the term erythroleukoplakia, which is described as a lesion with a mixed white and red surface, [8, 18, 19] and divided leukoplakias into homogeneous and non-homogeneous forms. [8, 18] PVL was (according to the 2005 WHO report) [8, 18, 19] classified as a subtype of non-homogeneous leukoplakia, characterized by the simultaneous occurrence of multiple multifocal leukoplakias. [8, 18] Warnakulasuriya emphasizes that PVL-type lesions fit perfectly into the terminology of PMDs, [18] currently termed OPMDs. [8]

The above-mentioned classification of OPMDs, developed in 2005, was published two years later, [9] and the nomenclature and classification were updated during subsequent workshops held in 2020. [20] Most of the above-described conditions retained their current status, [8] including leukoplakia, which is the most common condition among OPMDs. [8] PVL was recognized as a distinct form of multifocal oral leukoplakia, characterized by a progressive clinical course and with changing clinical and histopathological features. [8, 20]

PVL had also previously been described as a form of oral papillomatosis, although it does not always have a verrucous appearance. [21] In 2011, Jose M. Aguirre-Urizar proposed the term [22] proliferative multifocal leukoplakia [21, 22] for PVL, emphasizing the hyperplastic and multifocal nature of this condition. [22] Researchers based their theory on the belief that multifocality is a more important factor than the verrucous appearance of a lesion. [11] In a 2018 publication, Villa *et al.* described a study of 42 patients diagnosed with PVL, in whom clinical differentiation of the examined lesions had been confirmed. Villa *et al.* [13] proposed the name "proliferative leukoplakia" due to the fact that the condition does not always have a verrucous or nodular form. [13, 23]

Risk of malignant transformation

The risk of malignant transformation among OPMDs is estimated at 7.9%, [12, 18, 24] and PVL [8, 25, 26, 27] not only has the highest degree of malignant transformation, [5, 8, 9, 10, 14, 19, 26, 27] but also by the highest potential for recurrence [8, 9, 19, 26, 27] as a markedly aggressive form of the disease. [10, 23] According to WHO, the risk of malignant transformation of PVL among OPMDs is approximately 50%. [20] This frequent MT of PVL involves transformation into VC or OSCC, [16, 28] which, according to various sources in the literature, occurs in between 43.87% and 65.8% [27] of cases, and according to some in up to 70% of cases. [8, 28] Some reports even suggest a possible malignant transformation rate of around 100%. [10]

In their 2021 meta-analysis, González-Moles *et al.* showed that the majority of patients, i.e. 72.21%, developed OSCC, while 33.66% of patients with PVL later went on to be diagnosed with VC. [20]

In their retrospective clinicopathological analysis of PVL, non-dysplasia leukoplakia (LK), oral lichen planus (OLP)/oral lichenoid lesions (OLL), and VC, Alkan *et al.* (2022) demonstrated that in all the studied cases, malignancy developed on the basis of PVL, most frequently in the form of squamous cell carcinoma. In addition to a high rate of malignant transformation, the authors also noted the aggressive nature of the studied lesions. [10] In a systematic review conducted by Ramos-Garcia *et al.* in 2020, the rate of MT in cases of PVL was estimated at 43.87%. [9] On the other hand, based on a systematic review from 2021 Palaya *et al.* demonstrated that the risk of malignant transformation developing from PVL, most often in the form of OSCC, is approximately 46.5% [19, 21] and thus in almost half of all cases. [19] Previous publications had reported malignant transformation in over 70% of all cases of PVL. [17] MT rates of as high as 100% have even been claimed (Zakrzewska *et al.* 1996). [29, 30] A 2024 review by Vergier *et al.* focusing on the gingival form of PVL, reported that the malignant transformation rate of the disease was 47.75%, with a patient mortality rate of 5.84%. The mean follow-up time before malignant transformation was 3 years. [31] A 2025 review by Mohideen *et al.* demonstrated a 48.4% malignant transformation rate. The probability of progression of PVL lesions to malignancy was statistically higher in women than in men, i.e. 1.96 times higher. [21] Similar conclusions were reached by Palaya *et al.* in 2021, confirming that exposure to MT in women with PVL was 1.7 times higher compared to men. [19]

As Mohideen *et al.* reported in their 2025 publication, gingival lesions of PVL have a tendency to transform into OSCC in over 50% of cases. [21] The same researchers demonstrated that patients with PVL may develop OSCC more frequently than VC. [21] Similar conclusions were drawn by Gonzales and Moles based on a meta-analysis conducted in 2021, in which the majority of patients, i.e. 72.21%, went on to develop OSCC, while VC affected a smaller number, i.e. 33.66%. [20]

The risk of malignant transformation among OPMDs is also most often associated with the development of OSCC. [8, 12, 18, 24] Patients diagnosed with OSCC additionally face a greater risk of developing another cancer of this kind. [16] This was confirmed by Faustino *et al*. in their 2023 review, in which they emphasized that OSCC arising from PVL often results not only in the recurrence of this cancer, but also in the possibility of new, primary tumours developing. [32] In their 2021 systematic review, Proaño-Haro *et al.* showed a recurrence rate of 67.27% in patients treated for PVL. [23] In contrast, in 2025 Mohideen *et al.* noted a 55.56% recurrence rate in patients diagnosed with cancer arising from PVL. [21]

Clinical picture, diagnostic difficulties

In the initial clinical picture, flat [13, 33] single lesions or several white lesions [13] can be observed. [13, 33] Over time, as the lesion spreads or several adjacent lesions merge, lesions can appear in other locations. [8] As a consequence, over time, the initial lesion may not only enlarge, but new lesions may develop. [28] This type of disease is characterized not only by a tendency for new lesions to develop but also for them to grow and recur after treatment (proliferative). In addition, the surface of the lesion can transform from flat to rough and warty. [8, 10, 16] Additionally, the appearance of red spots, warty surface, erosions, paresthesia, or pain are features suggesting possible malignancy of the lesion. [10, 28] The initial clinical picture may correspond to lichen planus or leukoplakia. [26] PVL is characterized not only by the progressive spread of lesions, [5, 10] but also by subsequent foci appearing over time, [10] and thus by the multifocal nature of this disease. [5, 10, 11]

Over time, the above-described flat lesion gradually spreads, becoming diffuse and exophytic. [5] However, in its early stages it does not always have a verrucous appearance, [11, 33] which obviously complicates the diagnosis. [11] Diagnostic difficulties are likewise encountered when the lesions take on a lichenoid-like form. In 2020, Biancardi *et al.* described the case of three patients treated for lichenoid lesions in the oral cavity confirmed by a microscopic diagnosis of lichen planus. A final diagnosis of PVL was only made several years later. As a consequence, problems may arise when the clinical symptoms are similar to lichen planus, resulting in an incorrect diagnosis and, consequently, ineffective treatment. [34] PVL is characterized by its progression and irreversibility. [8]

Various clinical stages in the course of PVL can be distinguished. These include the initial occurrence of one or more white patches on the oral mucosa, which subsequently grow and spread to other sites. This in turn is followed by a change in the surface of some or all leukoplakia, assuming a more verrucous appearance. Finally, malignant transformation occurs, either in the form of VC or OSCC. [14, 35] The aggressive clinical course of PVL had already been pointed out by Hansen in 1985. [14, 17]

Not only may the physician examining the patient have problems diagnosing PVL, but so too can the pathologist. [8] Aguirre-Urizar *et al.* (2011) also noted the frequent underdiagnosis of PVL. [22] The condition may progress unnoticed by the patient, as a consequence of which frequent dental check-ups are necessary to detect alarming changes. [11]

PVL is characterized by the presence of multiple leukoplakia with variable clinical and histopathological features. [8] Microscopic findings in PVL may include hyperplasia, [10] hyperkeratosis, verrucous hyperplasia, verrucous carcinoma, or squamous cell carcinoma. [7, 10]

Occurrence and location

PVL usually occurs in multiple areas of the oral cavity simultaneously and is characterized by slow but continuous progression. [18, 36] It most often affects the gingiva, [8, 11, 37] the alveolar mucosa, [11, 31] as well as the tongue and buccal mucosa. [11] The most common location for PVL is within the gingiva, as was confirmed by a meta-analysis performed by Torrejon-Moya *et al.* from 2020, which showed that 50.9% of all PVL cases were identified in this area. [37] Similarly, in their 2021 review Ramos-García *et al.* confirmed the occurrence of PVL within the gingiva in 39.6% of all cases and within the buccal mucosa in another 21.6%. [9] The gingiva is also the most common location of MT, accounting for 39.9% of all incidences of the disease. [9] In 2025 Mohideen *et al.* showed that PVL changes, in the MT group most frequently affected the gums (in approximately 20% of all cases), followed by the gums and buccal mucosa (17.4%), the tongue (9%), and the buccal mucosa (8.4%). [21]

Prevalence, epidemiology, etiology, risk factors

PVL usually affects older people. [19] In their systematic review Torrejon *et al*. found the mean age of patients to be over 60 years. [37] According Palaia *et al.* in 2021, PVL affects older women to a greater extent. [19] This was confirmed by, among others, Ramos-Garcia *et al.* in their 2021 systematic review, in which 64.02% of all cases of PVL were diagnosed in women and 35.98% in men. [9] In 2022 de Pauli Paglioni *et al.* reported a higher risk of MT associated with PVL in women. [5] However, in 2025 Mohideen *et al.* identified a higher incidence of PVL in women than in men, with women accounting for 66.22% of all cases and men for 33.78%. Similarly, malignant transformation mainly affected women, who accounted for 74.3% of all cases, compared with 25.7% for men. [21]

PVL is a rare oral disease, [10, 38] an uncommon form of leukoplakia (OL), [28, 39] which, unlike PVL, is considered one of the most frequently occurring OPMDs, [5] with a prevalence of approximately 1–3%. [24, 40] OPMDs affect 2% of the world's population, [12, 18, 24] and the incidence of malignant transformation among them is 7.9%. [12] The aetiology of PVL is unknown, and it has no prognostic biomarkers in clinical practice. [38] This has been confirmed by a study conducted in 2025 (Vergier *et al.*), which showed no risk of increased MT gingival PVL in smokers compared to non-smokers. [31] In their 2021 review Ramos-Garcia *et al.* were unable to clearly identify the relationship between MT and gender, age or the use of stimulants such as smoking or alcohol. [9] This is in contrast to OL, which occurs more often in men and is associated with smoking (as it is 6 times more common in people who smoke tobacco), and also includes additional risk factors such as alcohol consumption and human papillomavirus. [11] In the case of PVL, on the other hand, tobacco and alcohol are not aetiological factors. [19, 35]

Similarly, a statistical review and meta-analysis from 2025 (Mohideen *et al.*) confirmed the lack of statistically significant differences between a group of smoking patients with cancer progression and a group without progression. Similar results were obtained for alcohol consumption. Regarding HPV, a statistically insignificant difference was noted in both patients with and without

cancer progression. [21] On the other hand, de la Cour *et al.* assessed the prevalence of HPV in leukoplakia, lichen planus, and PVL at 20.2%, 23.0%, and 24.7%, respectively. [24] Unfortunately, the relationship between HPV and the occurrence of cancer in PVL has not been clarified. [21] It is not possible to exclude or confirm the role of HPV in the etiopathogenesis of PVL. [19]

Further research is necessary. Similarly, the possible role of microbial factors in the pathogenesis of PVL remains unclear. Understanding the molecular mechanisms offers hope for solving the mystery of MT in PVL. A pilot study revealed dysregulation of 140 genes related to the immune system. This issue requires clarification. [21] As a consequence, there is a lack of prognostic biomarkers in the clinical diagnosis of PVL. [38]

In summary, both the aetiology of PVL and the factors influencing its progression to cancer are unknown. Close monitoring of patients is recommended and, if necessary, biopsies should be performed to assess changes in the appearance, colour, and size of observed lesions, as well as the appearance of subsequent lesions. [21]

The importance of observation and the role of the dentist

PVL carries a particularly high risk of malignant transformation. [11, 12] Furthermore, due to the frequency of multifocal occurrence, [27, 41] this condition remains difficult not only to treat [16, 42] but also to diagnose. [11, 27] Diagnosis is most often retrospective, based on clinical and histopathological examinations, which change over time. [10, 19] The approach of the dentist should be determined by the non-specific presentation of the lesion and necessitate frequent dental examinations of the patient as well as histopathological tests, sometimes multiple times and from multiple sites within the oral cavity. [9] Multiple biopsies are recommended, [21] especially in the case of newly formed red or nodular areas [19] as well as when changes occur in the size or appearance of existing lesions or new lesions emerge. [28] It is important to note that histopathological features may vary depending on the stage of the disease or the biopsy site. [21] Notice should be taken of any leukoplakia lesion whose surface assumes a papillary form, increases in size, or becomes exophytic. [11] Early diagnosis and early treatment of PVL are crucial due to the aforementioned high risk of MT. [12] Therefore, dental check-ups are of pivotal importance in identifying the disease, [11] as treatment depends on prompt diagnosis combined with periodic biopsies and patient observation. [11, 19]

Mortality associated with the disease can be as high as 30–50%. [28] As a consequence, dentists play a key role in the early detection and monitoring of these types of oral lesions. It is also important to educate patients about oncological vigilance. Often, dentists may be the first to observe and diagnose "disturbing changes" in the oral cavity.

If OPMDs lesions are suspected, the physician should refer the patient to a specialist for immediate treatment. A biopsy of the lesion should be performed, and follow-up appointments should be scheduled based on an individual risk assessment. [5] Therefore, early diagnosis, surgical removal, and patient observation are crucial in the treatment of PVL. [12]

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