









Clinical Case Report

Oral verrucous carcinoma in unusual locations: Two case reports



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ABSTRACT

Verrucous carcinoma is a rare, distinct, well-differentiated variant of squamous cell carcinoma, characterized by slow growth, local invasiveness, and low metastatic potential. The tumor predominantly affects the buccal mucosa, followed by the mandibular alveolar ridge, gingiva, and tongue. We report two cases of oral verrucous carcinoma in unusual locations. Case 1 presented as a white verrucous lesion on the lower lip, while Case 2 manifested as a palatal lesion with a cauliflower-like verrucous surface. Microscopically, both cases exhibited an exophytic tumor with a verruciform architecture and invasive growth through broad, bulbous rete ridges. Therefore, the definitive diagnosis for both patients was verrucous carcinoma. Distinguishing verrucous carcinoma from other verrucous lesions poses a significant challenge for pathologists. A deep biopsy incorporating adjacent normal tissue is mandatory for accurate differential diagnosis and to rule out hybrid carcinoma. (Rev Port Estomatol Med Dent Cir Maxilofac. 2026;67(x):xxx-xxx)

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Introduction

Verrucous carcinoma (VC), initially described by Ackerman in 1948, is a rare, distinct, well-differentiated variant of squamous cell carcinoma (SCC), with specific morphological and clinical characteristics.^{1,2} Unlike conventional SCC, VC exhibits slow growth, a local invasion pattern, and low potential for regional and distant metastases.³ Typically, VC af-

fects middle-aged and elderly adults, with a peak in the sixth and seventh decades of life, and is more prevalent in men than in women.^{4,5}

Regarding location, VC predominantly affects the mucous membranes of the head and neck region, particularly the oral cavity, larynx, hypopharynx, and trachea.^{6,7} In the oral cavity, the most common sites are the buccal mucosa, followed by the mandibular alveolar ridge, gingiva, and tongue.^{2,8} Clinically,

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oral lesions present as asymptomatic, well-demarcated exophytic growths with superficial papillary or verruciform projections, often resembling cauliflower. The lesions are usually white but can appear pink or erythematous.⁹

VC accounts for approximately 2–12% of all oral carcinomas.¹⁰ Although the etiology is not well understood, risk factors commonly associated with VC development include smoking, tobacco chewing, alcohol consumption, and poor oral hygiene. Other etiological factors include human papillomavirus (HPV), areca nut chewing, immunosuppression, oral leukoplakia, and oral submucous fibrosis.^{11,12} The treatment of choice for oral VC is surgical excision with wide margins.¹³ Overall, the prognosis is favorable, with a 5-year survival rate of approximately 80%; however, the recurrence rate varies from 30% to 50%.^{14,15}

This paper presents two cases of oral VC in elderly men occurring in unusual locations — the palate and the lower lip, followed by a literature review emphasizing clinical features, histological findings, and treatment modalities.

Case reports

Case 1

A 77-year-old male patient was referred to the (placeholder for the institution's name) for evaluation of a white verrucous lesion on the lower lip with a duration of approximately 1 year. The medical history revealed no systemic diseases or drug allergies. In addition, the patient reported a 63-year history of smoking. On physical examination, no regional lymphadenopathy was observed. A whitish exophytic lesion was present on the vermilion border and mucosa of the lower lip. The plaque was thick and well-circumscribed, with a papillary surface and a sessile base, measuring approximately 3 cm in diameter (Figure 1). Based on these findings, a clinical hypothesis of VC was established. An incisional biopsy was performed under local anesthesia, and the specimen was submitted for histopathological examination.



Figure 1. Extraoral examination showing a white verrucous lesion on the lower lip.

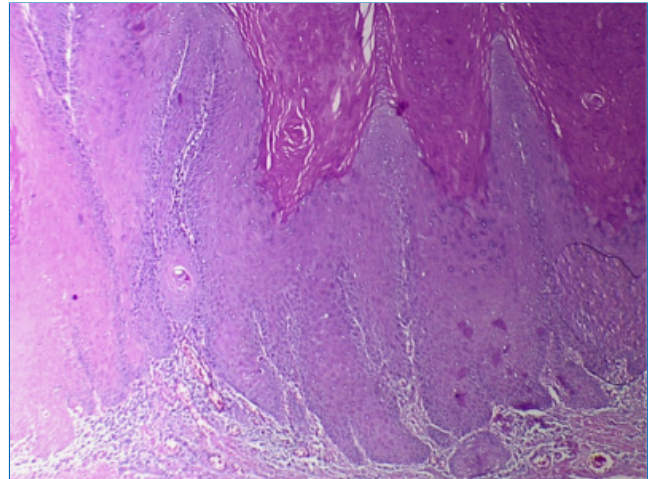


Figure 2. Photomicrograph showing exophytic tumor with church spire sign and broad, bulbous epithelial ridges that appeared to push against the underlying connective tissue (H&E, 40x).

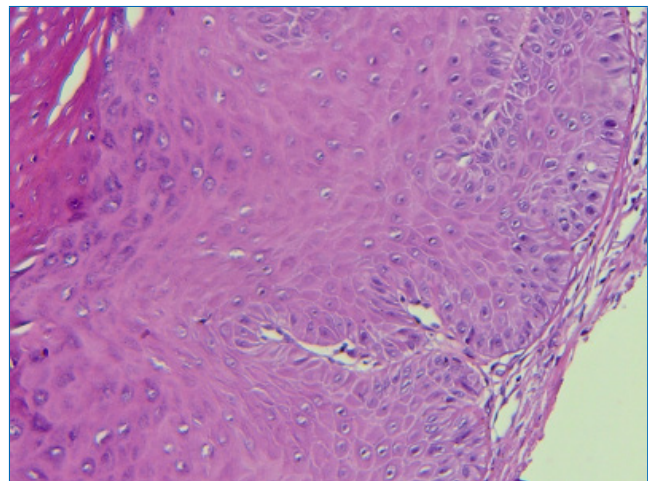


Figure 3. Photomicrograph showing mild cytologic atypia in the basal layer and absence of frank invasion (H&E, 100x).

Microscopic examination demonstrated a well-differentiated stratified squamous epithelium with marked superficial keratinization and verrucous exophytic projections, consistent with the “church spire” sign. Additionally, the hyperplastic epithelium exhibited endophytic growth with broad, bulbous epithelial ridges pushing into the underlying connective tissue (Figure 2). At higher magnification, mild cytologic atypia was observed in the basal layer, with no evidence of overt invasion (Figure 3). The underlying connective tissue showed a chronic inflammatory infiltrate composed predominantly of lymphocytes, as well as rich vascularity and no lymphovascular or perineural invasion. Therefore, a histopathologic diagnosis of VC was confirmed. The patient was referred to a head and neck surgeon and is currently undergoing treatment.

Case 2

A 71-year-old male patient presented to the (placeholder for the institution's name), complaining of a lesion on the palate with unknown duration. During the anamnesis, the medical history was non-contributory, although the patient reported a long-term habit of smoking and chewing tobacco. Extraoral examination revealed no edema or facial asymmetry; however, the right submandibular lymph nodes were enlarged. Intraoral examination revealed an extensive tumor mass located on the palate, extending to the upper alveolar ridge and buccal vestibule. The lesion presented exophytic growth, reddish-white coloration, and a cauliflower-like verrucous surface (Figure 4). Therefore, the diagnostic hypothesis was VC. An incisional biopsy was performed under local anesthesia, and the specimen was sent for histopathological analysis.

Histological analysis revealed acanthotic stratified squamous epithelium with hyperparakeratosis and a verruciform surface, resembling the blunt variant of verrucous hyperplasia (Figure 5). The epithelium showed moderate dysplasia and invasive growth, with blunt, broad epithelial ridges exhibiting pushing borders into the underlying connective tissue, compared to the adjacent normal epithelium (Figure 6). The lamina propria was fibrous, containing dense bundles of collagen fibers and minimal vascularity. Based on the findings, a diagnosis of VC was established. The patient was referred for head and neck surgery and is currently undergoing treatment.

Discussion and conclusions

In 1948, Ackerman¹ first described VC as an exophytic tumor with a verrucoid or cauliflower-like appearance, slow growth, local invasiveness, and low metastatic potential.¹⁵ The clinical characteristics of VC contrast with the ulcerative and nodular appearance of conventional SCC, which is the most frequent malignancy of the oral mucosa.^{15,16} VC accounts for



Figure 4. Intraoral examination showing a lesion on the palate extending to the upper alveolar ridge and buccal vestibule, displaying a cauliflower-like verrucous surface.

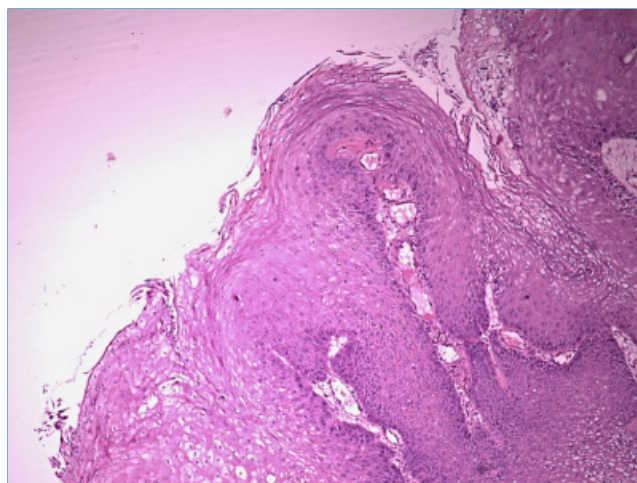


Figure 5. Photomicrograph revealing exophytic tumor with parakeratosis and verruciform surface, similar to the blunt variant of verrucous hyperplasia (H&E, 40x).

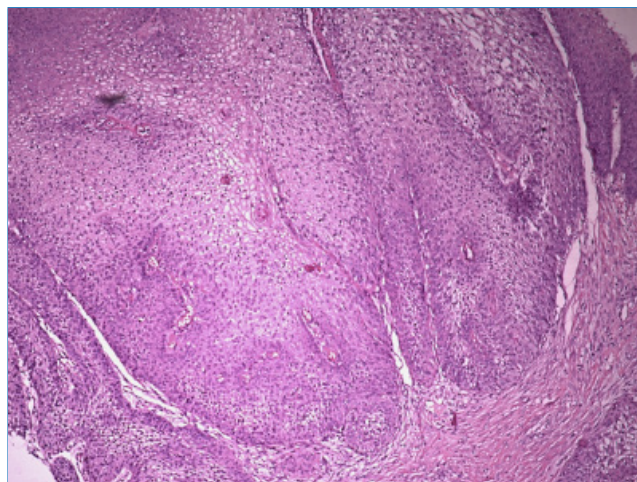


Figure 6. Photomicrograph showing moderate dysplasia and an invasive growth with blunt, broad epithelial ridges (H&E, 100x).

approximately 2–6% of all oral cavity carcinomas and is more prevalent in elderly men, usually in the 6th and 7th decades of life, as observed in the present cases.^{6,7} The most common intraoral locations are the buccal mucosa, gingiva, and tongue; this distribution differs from our cases, which involved the palate and lower lip.¹⁷

The etiology of VC remains controversial, but it is associated with poor oral hygiene, alcohol consumption, smoking (bidis and cigarettes), and, predominantly, smokeless tobacco and areca nut chewing. These etiological factors have been implicated in the malignant transformation of leukoplakia, erythroleukoplakia, submucous fibrosis, lichen planus, and oral lichenoid reaction.^{16,18-20} Conversely, approximately 20% of patients affected by VC do not report any predisposing factors.^{6,15} Regarding proliferative verrucous leukoplakia, the development of VC from this potentially malignant disorder is

less common, particularly as proliferative verrucous leukoplakia is not typically associated with tobacco use.²¹

Another predisposing factor suggested in the etiopathogenesis of VC is the human papillomavirus (HPV), which has been detected in tumor cells.^{22,23} Greere et al.²⁴ demonstrated the presence of HPV in approximately 20% of VC cases, especially subtypes 16 and 18. Although HPV's influence on the oncogenesis of VC is less significant compared to SCC, studies indicate that the pathogenesis of VC involves the abrogation of a tumor suppressor gene by oncogenic HPV subtypes.^{19,25} Additionally, physical factors, such as irritation and repetitive mechanical trauma, may contribute to VC development, alongside chronic inflammatory processes like osteomyelitis, fistulas, ulcers, and lipid necrobiosis.²⁵⁻²⁷ In the cases presented herein, the only etiological factors identified were tobacco use and poor oral hygiene, corroborating literature that smoking and chewing tobacco represent the primary carcinogenic factors in VC.

Regarding differential diagnosis, VC mimics several benign oral tumors; thus, lesions with a similar appearance must be considered in the clinical and histopathological differential diagnosis, such as squamous papilloma, condyloma acuminatum, focal epithelial hyperplasia, pseudoepitheliomatous hyperplasia, chronic hyperplastic candidiasis, verrucous hyperplasia, and proliferative verrucous leukoplakia.²⁸⁻³⁰ Furthermore, a distinction should be made between VC and conventional SCC, especially with SCC that exhibits a "verrucoid" appearance.³¹ The accurate diagnosis of VC can be challenging and requires an adequate incisional biopsy, as well as close cooperation between the clinician and the pathologist. In this context, superficial biopsies may microscopically display only benign features, such as hyperkeratosis, acanthosis, and papillomatosis, leading to an erroneous diagnosis of benign squamous proliferation. Therefore, a broad and deep biopsy is required to obtain an accurate microscopic diagnosis, avoiding the need for multiple biopsies and the misinterpretation of a malignant process as benign.^{1,29,32,33}

The definitive diagnosis of VC is usually based on histopathological examination of clinically suspicious oral lesions and requires the identification of the histological criteria described by Ackerman.¹ Histopathologically, VC typically presents with predominantly exophytic growth of well-differentiated hyperplastic epithelium, exhibiting a heavily keratinized or parakeratinized surface and sharp or blunt epithelial projections. In addition, scarce fibrovascular cores are noted, as well as numerous clefts between epithelial projections filled with keratin (keratin plugging). An intense subepithelial chronic inflammatory infiltrate is often present. Other important histopathological features include an intact basement membrane, preservation of stratification, minimal or absent atypia, and bulbous epithelial ridges, displaying an endophytic growth pattern that compresses the underlying connective tissue.^{13,31,34,35} The abrupt transition between the adjacent normal epithelium and the endophytic growth of the lesion is considered a critical feature for distinguishing VC from benign verrucous processes.³⁶ The cases presented herein showed microscopic findings consistent with these aforementioned features.

Several studies have demonstrated cases of SCC arising within VC. It is suggested that VC may arise *de novo* or from

epithelial dysplasia and subsequently transform into conventional SCC, presenting more or less differentiated foci of SCC. In this context, the presence of at least one focus of SCC transformation within a VC lesion characterizes a hybrid carcinoma.^{30,37,38} In fact, VC cases often present microinvasion, indicating that atypical basal cells have acquired invasive properties.³⁹ During histopathological examination, it is mandatory to rule out SCC and hybrid carcinoma, which require staging and treatment as conventional SCC due to the greater metastatic potential.³¹ To this end, large and deep biopsies are essential, as invasion may be missed in small and superficial specimens, making it unfeasible to exclude an underlying conventional carcinoma.³¹ Gokavarapu et al.⁴⁰ reported that approximately 50% of cases clinically diagnosed as oral VC or its benign precursors actually represented hybrid carcinomas. Therefore, a thorough microscopic examination of the entire specimen, using serial sections to examine deeper tissues, is crucial for identifying potential invasive components.^{9,40} If hybrid VC is identified, the pathologist must quantify the tumor components, define the grade of differentiation of the conventional SCC foci, and determine the depth of invasion, as well as evaluate lymphovascular and/or perineural invasion, in order to assist clinicians in determining adjuvant therapeutic options.⁴¹

Several therapeutic approaches are employed for oral mucosal VC, including surgery, chemotherapy, and radiotherapy.⁴² Among the therapeutic options, wide surgical excision is generally considered the treatment of choice, with recommended safety margins of at least 5 mm to minimize the risk of local recurrence.^{12,16,19,43,44} Neck dissection is typically not required for VC, as the neoplasm tends to invade locally but rarely metastasizes to lymph nodes.^{19,45,46} In this context, cervical lymphadenopathy associated with VC represents, in most cases, reactive changes rather than metastases.³¹ In cases involving extensive lesions, surgery may not be feasible and lead to unsatisfactory functional and cosmetic results.¹⁶ When resection is not indicated, therapeutic alternatives can be used, such as cytostatic drugs, imiquimod, α -interferon, and oral retinoids.⁴ The use of high-dose cytostatic agents has shown benefits in reducing VC size, while the complementary use of interferon (IFN) has the potential to slow tumor growth.⁴⁷ Chemotherapy with bleomycin or methotrexate has demonstrated success, used either as a monotherapy for inoperable cases or as neoadjuvant therapy to reduce the size of extensive lesions prior to surgery.^{48,49}

Regarding radiotherapy, some studies report the occurrence of radiation-induced anaplastic transformation of VC, which usually manifests 2–8 months after the end of treatment.^{50,51} Although the potential for anaplastic transformation remains controversial, radiotherapy is considered a salvage option when surgery is not feasible or when histopathological findings indicate adverse risk features, such as positive lymph nodes or invasion of deeper vital structures (e.g., nerves and blood vessels).^{8,13,52}

Regardless of the treatment modality, local recurrence rates are high, ranging from 30% to 50%, with a tendency to recur in the form of less differentiated carcinomas.³² Nevertheless, the prognosis for oral VC is usually favorable compared to other malignant tumors (7–8% mortality rate), except for hybrid carcinoma cases, which carry a worse prognosis and

must be managed as conventional SCC.^{7,37} The 5-year overall survival rate ranges from 64% to 94%, and the stage at diagnosis is the most relevant prognostic factor.^{7,16,17} In oral VC cases, surgical intervention appears to be related to improved overall survival, whereas advanced age and higher staging are associated with worse overall survival and disease-specific survival rates.⁵³

Furthermore, it is important to distinguish VC from other verrucous lesions, particularly proliferative verrucous leukoplakia and verrucous hyperplasia (VH).³⁸ Proliferative verrucous leukoplakia is an uncommon, progressive form of multifocal leukoplakia characterized by a high rate of malignant transformation to SCC or VC, as well as a significant recurrence rate. Although lesions often begin as simple, flat hyperkeratosis, indistinguishable from conventional leukoplakia, proliferative verrucous leukoplakia presents persistent growth, evolving into exophytic, verrucous forms.³⁸ Thus, current evidence suggests that untreated leukoplakia can progressively evolve into VH and/or VC.¹¹

Clinical distinction between VH and VC is challenging.¹¹ VH is considered an antecedent or early form of VC, and the transition is so consistent that dysplasia, once diagnosed, should be treated as VC.^{4,38,54,55} Strict clinical monitoring of VH is essential to detect any potential transition to VC.¹¹ The differential diagnosis is established microscopically, but an incisional biopsy must be performed with adequate margins to ensure a correct diagnosis. In VH, the hyperplastic epithelium and verrucous processes are superficial to the adjacent normal epithelium. In contrast, in VC, although the verrucous processes are superficial, the epithelial ridges extend deeply relative to the adjacent normal epithelium.^{38,56,57} Finally, VC must also be differentiated histologically from papillary SCC, which exhibits marked cellular atypia and frank invasion of the basement membrane.⁷

It is noteworthy that a thorough microscopic examination is essential for the differential diagnosis and appropriate treatment of VC and other verrucous lesions. Santoro et al.³¹ identified common pitfalls during the evaluation of verrucous lesions: histologically underdiagnosed cases, diagnosed as keratosis or VH, with synchronous or metachronous aggressive growth patterns; clinically undertreated cases, with proliferative verrucous leukoplakia and VC lacking a microscopic diagnosis of epithelial dysplasia; and surgically overtreated cases, including VC managed as conventional SCC, with radical resection and unnecessary lymphadenectomy. Thus, an accurate diagnosis relies on a comprehensive assessment of all characteristics rather than an isolated evaluation of individual findings, and close collaboration between clinicians and pathologists is essential.³¹

VC is a rare variant of oral SCC characterized by locally invasive and typically non-metastatic behavior, carrying a more favorable prognosis than conventional SCC. The differential diagnosis between VC and other verrucous lesions remains a challenge for pathologists, especially when biopsy specimens are insufficient and inaccurate and obtained from unrepresentative areas. An adequate biopsy from the periphery of the lesion, with sufficient depth and inclusion of adjacent normal tissue, is critical for differential diagnosis and for the analysis of potential areas of cytologic atypia, epithelial

dysplasia, and hybrid SCC. Thus, histopathologic distinction is essential for appropriate management, as VC requires more conservative treatment than conventional SCC. In addition, close communication between the clinician and the pathologist is mandatory for a correct diagnosis. Ultimately, the treatment of choice for VC is surgical excision. While the role of radiotherapy remains controversial, some authors suggest it for extensive tumors where surgery is not feasible or for patients with poor general health status.

Conflict of interest

The authors have no conflicts of interest to declare.

Ethical disclosures

Protection of human and animal subjects. The authors declare that the procedures followed were in accordance with the regulations of the relevant clinical research ethics committee and with those of the Code of Ethics of the World Medical Association (Declaration of Helsinki).

Confidentiality of data. The authors declare that they have followed their work center protocols on access to patient data and for its publication.

Right to privacy and informed consent. The authors have obtained the written informed consent of the patients or subjects mentioned in the article. The corresponding author is in possession of this document.

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Carcinoma verrucoso oral em localizações incomuns: Dois casos clínicos

R E S U M O

O carcinoma verrucoso é uma variante rara, distinta e bem diferenciada do carcinoma de células escamosas, que apresenta crescimento lento, padrão de invasão local e baixo potencial metastático. O tumor tem predileção pela mucosa bucal, seguindo-se a crista alveolar mandibular, a gengiva e a língua. Apresentamos dois casos de carcinoma verrucoso oral em localizações incomuns. O caso 1 mostrou uma lesão verrucosa branca no lábio inferior, enquanto o caso 2 revelou uma lesão no palato com uma superfície verrucosa semelhante a couve-flor. Microscopicamente, ambos os casos demonstraram um tumor exofítico com uma superfície verruciforme e um crescimento invasivo com cristas epiteliais bulbosas e largas. Portanto, o diagnóstico definitivo em ambos os casos foi carcinoma verrucoso. O diagnóstico diferencial entre carcinoma verrucoso e outras lesões verrucosas permanece um desafio para os patologistas. Uma biópsia profunda com margens de tecido normal adjacente é essencial para o diagnóstico diferencial e exclusão de um carcinoma híbrido. (Rev Port Estomatol Med Dent Cir Maxillofac. 2026;67(x):xxx-xxx)

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