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Original Article

Verrucous Carcinoma: The Quiet Invader

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ABSTRACT

Verrucous carcinoma isa slow-growing type of squamous cell carcinoma that occurs in the mouth. Smoking tobacco is the main cause of this condition. It presents as a warty cauliflower like lesion that grows slowly and has minimal distant metastasis. It commonly affects buccal mucosa, alveolar ridge gingiva, floor of mouth and tongue. Main histological feature differentiating it from other lesions would be pushing invasion pattern and well differentiated epithelium and Surface epithelium shows thick Para keratin layer with papillary projections. Surgery being the primary mode of treatment having better prognosis.

Keywords: Ackerman's Tumor, Buccal Mucosa, Elderly Male Cauliflower Like, Pushing Margin, Surgery

INTRODUCTION

Head and neck cancer is one of the more common types of cancer, with many new cases reported in 2020.Half of these cases come from the lips and mouth. (1,2) Squamous epithelium is the source of more than 90% of mouth cancer cases. (1) Oral squamous cell carcinoma has a high incidence and mortality rate and is one of the most aggressive tumors in humans. (3) In 1948, Ackerman identified verrucous carcinoma as a different type of oral squamous cell carcinoma. (4) The tumor is minimally invasive and grows slowly. Clinically, it presents as a distinctive, white, exophytic growth that is usually asymptomatic. Histologically, it differs from oral squamous cell carcinoma in its larger base, pushing margins, and intact basement membrane. (5) A case of well-differentiated squamous cell carcinoma of the mouth was first reported by Fridell and Rosenthal in 1941 as "papillary verrucous carcinoma. (6) " Other names for this tumor include Ackerman's tumor, Buschke–Lowenstein tumor, florid oral papillomatosis, epithelioma cuniculatum, carcinoma cuniculatum, and Snuff dippers cancer. (7) It commonly affects the head and neck, but oral cavity, genitalia, larynx, skin and esophagus are generally affected. (5) The buccal mucosa, mandibular alveolar crest, gingiva, and tongue are the most common locations for it to occur in the oral cavity; the most common non-oral location is the glottic larynx. VC is commonly experienced by males in their fifth or sixth decades of life. The major etiological factor would be tobacco in both smokeless and inhaled forms, betel nut and areca nut chewing, alcohol consumption, human papillomavirus's (HPV) and proper oral hygiene. Surgery is still the primary treatment of choice. [8]

Etiology

Oral microbata, alcohol consumption, smoking, and areca nut chewing have clearly demonstrated being associated with OVC.

Areca nut:

One of the carcinogens found in areca nut preparations is N-nitroso amines. These carcinogens destroy single DNA strand and cause mutations aiding in the progression and spread of malignancies. Arecoline component present in areca nut extracts have teratogenic potential leading to significant increase in the oral cancer [9,10]

Smoking:

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Another factor that might lead to OVC is smoking. Saliva can absorb more than 300 carcinogens from tobacco smoke and its water-soluble constituents. Two examples are the aromatic hydrocarbon benz-pyrene and tobacco-specific nitrosamines (TSNs). These carcinogens disrupt DNA replication and harm immune system replicating cells by generating DNA adducts, mainly 06 methyl guanine. [9,11]

Alcohol:

In the early phases of oral carcinogenesis, excessive alcohol use can change the intracellular metabolism of epithelial cells, compromising cellular function (e.g., decreased mitochondrial function and increased DNA alkylation). Additionally, it might act as a solvent to help carcinogens pass through oral cellular membranes. [9,12]

Human Papillomavirus (HPV):

Among other possible causes of OVC, HPV's controversial and unclear pathogenic involvement is alarming.Research on the role of HPV appears to be inconsistent. Further knowledge acquisition requires the use of highly sensitive molecular biology techniques, such PCR, and the selection of an appropriate sample size. [9,13]

Oral microbiota:

They may have a major impact on mouth carcinogenesis by altering the local metabolism of carcinogens brought on by smoking and drinking. Oral cancer has been linked to five bacterial phyla: Firmicutes, Proteobacteria, Bacteroidetes, Actinobacteria, and Fusobacteria. The oral and gastrointestinal tracts are instantly exposed to carcinogenic acetaldehyde following alcohol use because oral bacteria can convert ethanol to acetaldehyde, an in vitro and in vivo genotoxin. Common oral bacteria can boost the activation of carcinogenic nitrosamines from tobacco smoking by converting the carcinogenic (IARC, Group 2A) nitrosodiethylamine (NDEA), a nitrosamine from tobacco smoke, to its hydroxylated product in vitro. [9,14]

Clinical features

The buccal mucosa, alveolar ridge, tongue, lip, gingiva, and mouth floor are frequently affected by verrucous cancer. Verrucous carcinoma frequently affects elderly males 60 years of age and older [8] Oral verrucous carcinoma usually manifests as a gradual exophytic growth with cauliflower-like pebbly, warty lesions. [9] Both distant and regional lymph node metastases are quite uncommon. Verrucous carcinoma becomes locally invasive if left untreated. Reactive lymph nodes are frequently palpable. [6]

Based on clinical signs and prognosis, Tang et al. classified OVC into three categories.

- Exogenic
- Cystoid
- Infiltrative

The exogenic type of OVC is characterized by slow tumor growth, cauliflower-like warty lesions, and exophytic development. The other two types of OVC grow and mature more rapidly than the exogenic type. [13]

Histological features

It is composed of well-differentiated malignant squamous epithelial cells arranged in broad papillary projections. Low-grade malignancy – grows slowly and metastasis is rare. Tumor spreads more laterally than vertically. Surface epithelium shows thick para keratin layer with papillary projections (“church spire” appearance). It shows massive bulbous acanthotic rete ridges pushing into connective tissue and characteristic “pushing margin” (uniform downward growth at same level). Para keratin plugging seen in deep cleft-like spaces. Cells show minimal atypia, rare or absent mitosis, near-normal maturation. Basement membrane usually intact with underlying connective tissue shows chronic inflammatory cell infiltration. It may show epithelial pearls and microcysts. Long-standing lesions can compress muscle and cause bone saucerization. Rarely, may transform into invasive squamous cell carcinoma if untreated. [15]

Differential Diagnosis

The differential diagnosis of verrucous carcinoma can be [13]

Diagnosis	Differential Diagnosis
Oral verrucous hyperplasia	differs from nearby normal mucosal epithelium in that it does not show hyperplastic epithelial invasion into the lamina propria.
Oral squamous papilloma	appears as several long, thin projections that resemble fingers and extend above the mucosal surface.
Oral squamous cell carcinoma (OSCC)	From the epidermis to the dermis, squamous epithelial cells form a nest.

Treatment:

Surgery, chemotherapy, radiation, or combinations of these, cryosurgery and shave incision are the major treatments available for verrucous carcinoma. Wide surgical incision, frequently results in defect and disfigured appearance. Radiation and chemotherapy are considered due to its poor prognosis and anaplastic transformation. With recent advances like photodynamic therapy and CO2 laser therapy are aimed to treat verrucous carcinoma. [13]

CONCLUSION:

Verrucous carcinoma is a well-differentiated squamous cell carcinoma with distant metastases, slow development, and little invasiveness. Even though the prognosis is favorable, a precise diagnosis requires a deeper comprehension of the condition's origin, clinical characteristics, histopathological characteristics and differential diagnosis for early identification and timely intervention. The main option for the treatment is wide surgical excision.

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