An Update on Squamous Carcinoma of the Oral Cavity, Oropharynx, and Maxillary Sinus

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• Squamous carcinoma • Oral cavity • Oropharynx
• Maxillary sinus

There are more than 45,000 new cancer cases involving the head and neck diagnosed each year within the United States. Squamous cell carcinoma (SCC) accounts for the majority of cases, often occurring within the oral cavity and oropharynx. Overall 5-year survival rates (60%) have not changed dramatically over the past few decades.[1] New demographic patterns are also emerging with younger patients (less than age 45 years) and those patients without tobacco or alcohol abuse developing these cancers.[2,3]

The anatomic sites composing the oral cavity are the alveolar ridge/gingiva, retromolar pad, buccal mucosa, floor of mouth, hard palate, and anterior two-thirds of the tongue. Recent literature suggests that cancers of the buccal mucosa and the gingiva are more aggressive than previously thought.

The oropharynx is composed of the tongue base, soft palate, tonsils, and posterior pharyngeal wall. Cancers of the oropharynx typically present with advanced-stage disease due to its rich lymphatic system that facilitates early nodal spread, resulting in a high incidence of early nodal metastases. Current literature suggests a strong link between human papillomavirus (HPV) and oropharyngeal cancers leading to much debate regarding treatment strategies involving these aggressive tumors.[2]

Improvements in technology have resulted in better imaging pretherapy and post-therapy, minimally invasive surgery, improved radiotherapy techniques, and reconstructive options. These advancements are leading to improvements in cancer treatment and quality of life.

This article reviews current literature and various controversial topics involving the diagnosis and treatment strategies for patients with oral cavity/oropharyngeal cancers. Although not considered cancer within the oral cavity, maxillary sinus SCC is discussed. The proximity to the oral cavity and late presentation often make it difficult for clinicians to establish primary origin (maxillary gingiva vs maxillary sinus).

HUMAN PAPILLOMAVIRUS

High-risk oncogenic HPV types (HPV-16 and HPV-18) have a significant role in the pathogenesis of oropharyngeal SCC. Various studies have demonstrated that 35% to 65% of oropharyngeal tumors contain high-risk HPV. This strong association is thought to result from the relationship

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between the oropharyngeal mucosa and the mucosal-associated lymphoid tissue of the pharyngeal and lingual tonsils. The crypt-like tissue harbors persistent HPV infection leading to stable oncoprotein expression and carcinomatous transformation. This correlation has important implications because patients with HPV-associated oropharyngeal SCC have a better prognosis than do those with non–HPV-related tumors. The most recent National Comprehensive Cancer Network guidelines include HPV testing for oropharyngeal SCC. The difference in overall survival in patients with HPV-positive and HPV-negative carcinomas at nonoropharyngeal SCC sites has not been demonstrated. Prevention of HPV-positive oropharyngeal SCC with the HPV vaccination has not yet been demonstrated.[2,4]

The link between oral SCC and high-risk HPV is not as clearly defined. HPV identified within oral carcinomas is variable ranging from 4–80%. The majority of these studies used nonquantitative polymerase chain reaction (PCR) methods to detect HPV DNA, which lacks specificity for oncogenic infection. In a large multicenter trial of 766 oral cancers, HPV DNA was identified in only 4% of tumors.[5] Lopes and colleagues[6] screened 142 consecutive cases of oral SCC using both conventional PCR with consensus primers and type-specific quantitative PCR. The investigators demonstrated a low prevalence of high-risk HPV (2%) and concluded that there is little evidence to suggest that oral cavity SCC is associated with high-risk HPV and that routine testing of oral cancers for HPV could not be justified. One criticism of this article was the lack of stratification between young and old patients with oral SCC who had HPV isolated from the tumors. The prevalence of oral SCC in younger patients is increasing and HPV may be implicated as a causative factor.

PANENDOSCOPY

According to the literature, head and neck synchronous and metachronous second primary cancers have been reported to occur with a frequency varying from 3% to 21%. This risk has been hypothesized due to the field cancerization change thought to occur within the mucosa of the entire aerodigestive tract after exposure to exogenous carcinogens, such as tobacco and alcohol.[7] In the past, many clinicians advocated panendoscopy, involving laryngoscopy, bronchoscopy, and esophogoscopy, in all patients diagnosed with a head and neck malignancy. Arguments against this rationale include increased iatrogenic risk (ie, esophageal perforation), increased cost, and low yield in diagnosis. This controversy is further complicated by improvements in diagnostic imaging (ie, positron emission tomography [PET], MRI, CT, and ultrasound) and office-based flexible endoscopy and by the increased incidence of newly diagnosed patients who have never abused tobacco or alcohol. A recent study comparing diagnostic panendoscopy in patients with a past history of tobacco abuse with nontobacco users identified a synchronous primary rate of 12% in the tobacco user group whereas no second primary carcinomas were found in the nontobacco user cohort. The investigators concluded that panendoscopy is unlikely to result in the identification of a synchronous carcinoma in patients who have never used tobacco.[8] Haerle and colleagues[9] evaluated the accuracy of PET/CT scan versus panendoscopy for detection of second primary tumors in 311 patients. The prevalence of second primary tumors was 6.1% with PET/CT scan compared with 4.5% with panendoscopy. An additional 5 cancers were detected with PET/CT scan that were missed within the field of the panendoscopy. Recent evidence suggests that symptom-directed panendoscopy performed after PET/CT imaging provides better identification of synchronous and unknown primary tumors. Routine panendoscopy should still be considered for patients with significant risk factors, such as tobacco abuse, and in situations requiring better tumor visualization to aid in assessing stage and in planning surgical resection.[10,11]

CANCER OF THE ORAL CAVITY AND OROPHARYNX—SURGERY AND RADIATION

Early-stage cancers of the oral cavity and oropharynx are generally treated with surgery or radiotherapy. Advanced-stage tumors (stage III/IV disease) require multimodality treatment. Most guidelines recommend that advanced resectable oral SCC be treated with surgery followed by radiotherapy or concurrent chemoradiotherapy (CCRT) depending on pathologic features identified post-resection. Surgery as the first-line modality for tumors within the oral cavity has been considered for various reasons. The oral cavity is easily accessible surgically compared with other regions within the head and neck. Surgical removal of the tumor allows for pathologic assessment of tumor histology and margins. Pathologic staging of the tumor accurately determines the adequacy of resection and the need for adjuvant therapy if the tumor is “upstaged.” Improvements in reconstructive surgery (computer-assisted/simulated design and microvascular surgery) have allowed for better functional outcomes than are customary after

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