coma (ES) is very rare and may be misdiagnosed as another SRCT.

Case report. A 37 weeks’ gestational age male was delivered vaginally. An 8 x 7 cm blue to purple mass protruded from the left facial/ear region that was not detected by ultrasound at 20 weeks’ gestation. Diagnostic imaging and clinical impression were interpreted as a vascular tumor, and prednisone and lansoprazole therapy was initiated. The tumor rapidly increased in size and ulcerated; therefore, resection was performed on the eighth day.

Pathology. The tumor was composed of sheets of undifferentiated cells with scant cytoplasm, frequent mitoses, minimal stroma, and no differentiation. Flow cytometry revealed CD56 (neural cell adhesion molecule) and CD38 positivity with no evidence of hematolymphoid malignancy. Immunocytochemistry exhibited CD56 and dot-like Golgi staining with CD99. Myogenic (myogenin, desmin), neuroblastic (NB84), epithelial (epithelial membrane antigen, cytokeratin), germ cell (alpha-fetoprotein, CD30, placental alkaline phosphatase), vascular (CD34, CD31), rhabdoid (integrase interactor 1) and melanocytic (HMB-45, MelanA) markers were negative. Electron microscopy provided evidence for neuroectodermal (ES) derivation with rare neurosecretory granules, bland nuclei, intercellular junctions and lack of neurites. Cytoarchitectures identified by fluorescence in situ hybridization EWS rearrangement and novel EWS-NFATc2 translocation [t(20;22)]. Despite therapy, the child died of disease 4 months after protocol initiation.

Conclusion. Multimodal diagnostics, including cytogenetic and molecular analyses, are important for definitive diagnosis, proper treatment, and discovering novel translocations in neonates with SRCT.

ORAL WARTS IN HIV-INFECTED INDIVIDUALS EXHIBIT LACK OF TISSUE AUTOFLUORESCENCE: A CLINICOPATHOLOGIC STUDY. C. Flaitz, M. Nichols, N. Vigneswaran, J. Bouquot, A. Zuluaga. U Texas Dental Branch, Bering-Omega Dental Clinic, Remicalm, Houston.

HIV-infected individuals experience an increased prevalence of oral warts. Multiple and high-risk human papillomavirus (HPV) genotypes have been identified in these lesions, which suggests a potential risk for HPV-associated squamous cell carcinoma. The purpose of this clinical study was to evaluate HPV-induced oral lesions in HIV-infected individuals, using direct autofluorescence visualization, and to correlate the results with histopathologic findings. Consecutive HIV patients from the Bering-Omega Dental Clinic with oral warts and hyperkerototic lesions were recruited. After informed consent, patient demographics, CD4 count, viral load, medications, and oral cancer risk factors were recorded. Clinical features and location of lesions were evaluated and photographed under white light, violet light–excited autofluorescence (405 nm), and green-amber light reflectance (540-560 nm), using Identafi 3000 (Trimira, Houston, TX). Surgical specimens were submitted for routine microscopic diagnosis. Immunoperoxidase (IHP) studies using HPV Cocktail Broad Spectrum (Biocare Medical, Concord CA) were performed. Twenty-nine patients had HPV lesions and/or squamous cell carcinoma (25 male, 4 female; mean age 47 years; mean CD4 count 339). Four patients were in the control group (3 male, 1 female; mean age 45 years; mean CD4 count 163). In the HPV group, tobacco use was reported in 55% and alcohol use was 38%; the control group reported 100% tobacco and 50% alcohol use. IMP for HPV was positive in wart but not in control subjects. Loss of fluorescence (LOF) was 85% for oral warts, 9% equivocal, and 6% no loss. LOF for the control group was 25% with positive vascular reflectance for lichenoid mucositis. In this pilot study using a multispectral oral examination light, oral warts consistently demonstrated LOF in this high-risk oral cancer group.


Background. Human papillomavirus (HPV) infection has been recently identified as an important etiologic factor in head and neck (HN) pathology, with important treatment and prognostic implications because HPV-positive tumors affect a younger nonsmoking population and have a distinctly better survival after treatment than the HPV-negative cohort.

Study design. The identification and genotyping of high-risk (16, 18, 31, 33, 35, 39, 45, 51, 52, 56, 59, and 70) and low-risk (6 and 11) HPV was investigated in 18 HN cancers and precancerous lesions—8 invasive tonsillar squamous cell carcinomas (5 moderately differentiated [MDSCC] and 3 poorly differentiated [PDSCC]), 5 laryngeal tumors (4 squamous papillomas, 1 of which exhibited moderate dysplasia and 1 MDSCC), 2 well differentiated SCC [WDSCC]) involving the left arytenoid and fossa of Rosenmuller, respectively, and 1 invasive PDSCC of the soft tissues of the neck of unknown primary—were investigated real-time polymerase chain reaction (PCR). Two invasive WDSCC of the dorsal tongue and the floor of the mouth and 1 lesion of the lateral tongue showed keratosis with moderate dysplasia were also included.

Results. Of all lesions, 61.1% (11/18) were HPV-16 positive and none demonstrated low-risk HPV subtypes; 5/6 MDSCC were HPV-16 positive and 1 MDSCC was inconclusive; 3/4 PDSCC demonstrated the HPV-16–positive subtype. Neither the oral lesions nor the tumors of the arytenoid and fossa of Rosenmuller showed HPV presence. All laryngeal papillomas were positive for HPV-16.

Conclusions. Despite the small sample size, this study further confirms the detection of high-risk HPV in oropharyngeal carcinoma and laryngeal papilloma. The absence of HPV in oral malignant and precancerous lesions also delineates the limited contribution of HPV to the development of these lesions. These findings should also prompt the clinicians to investigate the presence of HPV in MDSCC and PDSCC, with less emphasis on WDSCC, via real-time PCR.


Primary intraosseous carcinoma (PIOC) is a rare malignancy of the jaws arising from the remnants of residual odontogenic epithelium. PIOC is most commonly seen in the posterior mandible and has a strong male predilection. The incidence of PIOC is extremely low, with <.50 cases reported in the literature. We report an additional case of PIOC in the mandible of a 73-year-old patient. The lesion was initially discovered as an incidental...
ulocural radiolucency at the apex of tooth #28. Owing to the increasing size and nonhealing nature of the lesion after root canal therapy, an incisional biopsy was performed. Histopathologic examination revealed an epithelial malignant neoplasm of odontogenic origin consistent with PIOC. The patient was treated with anterior mandibulectomy followed by reconstruction with a fibula free flap. In addition to a comprehensive literature review, we discuss the diagnostic criteria and clinical, histopathologic, therapeutic and prognostic features of PIOC.


Adenosquamous carcinoma (ASC) is a rare and aggressive malignant neoplasm with a poor prognosis. It arises from both the surface and the salivary ductal epithelium, with histologic features of both squamous cell carcinoma and adenosquamous carcinoma. The most common site in the upper aerodigestive tract is the larynx, followed by the oral cavity. An exhaustive literature search revealed <20 documented cases of intraoral ASC. The most common intraoral locations are the tonsillar pillars, floor of the mouth, and posterior tongue. We report a case of ASC on the palate of a 72-year-old edentulous patient, who presented with a chief complaint of pain and soreness of 3 weeks’ duration under the denture. Clinically, the lesion presented as a thick diffuse leukoplakia extending from the right vestibule to the alveolar ridge and the hard and soft palate, with cratered ulceration of the left side of the hard palate. An incisional biopsy was performed. The microscopic examination displayed squamous cell carcinoma and adenosquamous carcinoma, favoring a diagnosis of ASC. The patient opted for treatment with chemotherapy and radiation only, without extensive surgery. The clinical and histopathologic features of ASC, differential diagnosis, and review of literature are presented.


Leukoplakia is the most frequently occurring oral lesion with malignant potential: a clinical entity defined by the World Health Organization as “white plaques of questionable risk having excluded (other) known diseases or disorders that carry no increased risk for cancer.” Essentially, leukoplakia is a diagnosis of exclusion. Histologically, leukoplakia may represent hyperorthokeratosis or parakeratosis, with or without acanthosis, inflammation, and/or dysplasia. Cases submitted for histopathologic examination from January 2007 to June 2008 to Pathology Services, a surgical pathology laboratory in Cambridge, Massachusetts, affiliated with the Harvard School of Dental Medicine, were reviewed for inclusion in the study. In total, 1,269 cases were accepted. Among these, 417 were true leukoplakia, and the other 852 were specific benign lesions. Of the 417 true leukoplakias, 241 showed evidence of reactive lesions, and only 176 cases were nonreactive non-specific leukoplakia. Among these 176, 43.2% (76) were dysplastic, and all 176 cases were atypical. Current literature suggests that 80.1% of leukoplakias reveal no histopathologic evidence of epithelial dysplasia. This may be partly because frictional injuries such as benign alveolar ridge keratosis and all classic morsicatio mucosae oris (chronic bite injury) were excluded. This study found 10.9% of leukoplakia to be dysplastic and 89.1% without evidence of dysplasia. After exclusion of these benign frictional and otherwise reactive keratotic conditions, this study found the proportion of cases of true leukoplakia that represent atypia, dysplasia, carcinoma-in-situ, and invasive squamous cell carcinoma to be 43.2%, twice that previously reported.

P38 REGULATES INTERLEUKIN-12–MEDIATED CYTOKINE SECRETION IN HEAD AND NECK SQUAMOUS CELL CARCINOMA. R. Vander Broek, E. van Tubergen, K. Kirkwood, N D. Silva. U Michigan School of Dentistry, Ann Arbor, Medical U South Carolina School of Dentistry, Charleston.

Background. Cytokines and proinflammatory factors are critical mediators of head and neck squamous cell carcinoma (HNSCC). RNA-binding proteins, such as tristetraprolin (TTP), target cytokine mRNA for degradation and decrease cytokine production. However, during an inflammatory response, TTP is functionally inactivated by phosphorylation through p38 activity, leading to increased expression of cytokines. A constitutively active p38 pathway is implicated in tumor survival and interleukin (IL) 6 production. Previously, we showed that increased IL-6 in HNSCC is prognostic for poor disease-specific survival and higher probability of tumor recurrence. Therefore, an active p38 mitogen-activated protein kinase pathway may inactivate TTP and contribute to tumor progression.

Objective. The aim of this study was to delineate the role of p38 activity in regulating cytokine secretion in HNSCC.

Study design. p38 activation was optimized with an IL-12 dose curve. UM-SCC-11A and 81B were transfected with small interfering (si) RNA nontarget (NT) and p38. Conditioned medium was collected from cells transfected with siNT or si-p38 in the presence of IL-12. IL-6, vascular endothelial growth factor, and prostaglandin E2 secretion were quantified by ELISA.

Results. p38 is activated in HNSCC cell lines. IL-12 mediates p38 activation in HNSCC cell lines maximally at 10 ng/mL. p38 knockdown was verified by immunoblot analysis. Maximal knockdown of p38 occurred at 72 hours post transfection. p38 knockdown reduced cytokine secretion even in the presence of IL-12 at 72 hours after transfection.

Conclusions. These findings support the potential for targeting regulators of cytokine secretion, such as p38 or downstream targets of p38, as a practical means for limiting the progression of HNSCC. Future studies will elucidate the mechanisms of p38 regulation of TTP activity in HNSCC. (U Mich School of Dentistry and National Institute of Dental and Craniofacial Research grant nos. R01 DE018512 and K02 DE019513)


In head and neck squamous cell carcinoma (SCC) samples, we observed the intimate association of myeloid dendritic cells (DC) with SCC cells in both primary tumors and their lymph node metastases. In vitro videomicroscopy studies showed that the direct interactions between monocyte-derived DC and SCC cells produced a significant effect on SCC cell migration. Our current research further examines the influence of monocyte-