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 × 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 (integrin alpha 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. Cyogenetics 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 neoplasms 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 papilloma virus (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 (IMP) 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 papilloma virus (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, 42-45, 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 Rosennmuller, 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 Rosennmuller 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