

Findings: Oral cancer OSC2 cells were assessed following IFN γ treatment at specific time-points. DSPP and MMP20 mRNA expression levels, as well as ER stress, UPR and calcium homeostasis-related proteins, including GRP78, SERCA2b, IP3r, PERK and IRE1, were assayed by RT-PCR, while Bcl-2, Bax, PCNA and Cytochrome C protein expression levels were analyzed by Western blot. IFN γ treatment significantly downregulated mRNA levels of major ER stress regulator GRP78, and, to a lesser extent, UPR-related molecule IRE1, but without significant effect on PERK. Furthermore, IFN γ affected the mRNA expression levels of important ER calcium homeostasis molecules, downregulating SERCA2b and upregulating IP3r. Additionally, DSPP and MMP20 mRNA levels were significantly reduced by IFN γ . IFN γ treatment also hampered OSC2 migration (assessed by wound-healing assay), reduced cell viability (evaluated by MTT), and enhanced apoptosis (assayed by Annexin V/FITC flow cytometry). These changes were accompanied by induction of Bax and Cytochrome c and downregulation of PCNA and Bcl-2 protein levels.

Conclusions: IFN γ appears to inhibit oral cancer cell viability and migration, and drive apoptosis, possibly by regulating ER stress and UPR mechanisms. DSPP and MMP20 downregulation appears to correspond to the IFN γ -induced changes in ER calcium homeostasis in OSCC.

HAIRY LEUKOPLAKIA IN A PATIENT UNDERGOING ANTI-RETROVIRAL THER-

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We report a case of hairy leukoplakia that developed in a patient undergoing anti-retroviral therapy. A 53-year old white male presented with mild erythema in the anterior maxillary gingiva and was managed with clobetasol gel, after excluding the possibility of candidiasis. Patient's medical history was significant for HIV, bipolar disorder, high blood pressure, high cholesterol, chronic bronchitis, smoking, and alcohol. His medications included Androgel, Axiron, atorvastatin, bupropion, clonazepam, finasteride, hydrochlorothiazide, lamotrigine, lisinopril, pantoprazole, Prezcofix, Trazadone, Truvada (200 mg emtricitabine, 300 mg tenofovir), Ziprasidone, zolpidem, and baby aspirin. During one of multiple follow-up appointments, an asymptomatic white plaque was identified on right lateral tongue. Clinical differential diagnoses included hairy leukoplakia and hyperkeratosis secondary to trauma. Patient reported that his physician changed Truvada to Descovy (200 mg emtricitabine, 25 mg tenofovir) since his last appointment. At the appointment six weeks later, the white plaque increased in size, and additional white plaques were found on the left dorso-lateral surface and dorsal tongue.

Two biopsies were taken, one from the right lateral and the other from left dorso-lateral tongue. The biopsies showed similar histological features including hyperparakeratosis with shaggy surface and bacterial colonization. Intracellular edema and pyknotic nuclei were noted in the spinous cell layer. Upper spinous cell nuclei were enlarged and glassy appearing, without obvious nucleoli or nuclear beading. An Epstein-Barr encoding region (EBER) in-situ hybridization was performed, which demonstrated presence of EBV. Blood testing, taken five days after the biopsy, showed a CD4 count and viral load within normal

limits. The patient's physician prescribed a course of acyclovir 800mg for treating oral hairy leukoplakia. The oral lesions reduced in size at the follow-up appointment three weeks after completion of acyclovir therapy. Further follow-up information also will be presented.

CLINICAL, HISTOPATHOLOGICAL, AND MOLECULAR CHARACTERIZATION OF CARVAJAL SYNDROME WITH ORAL MANIFESTATIONS. DR. COLBY HAINES, DR. JENNIE ISON, DR. JOHN FANTASIA, DR. KATHLEEN SCHULTZ. ZUCKER SCHOOL OF MEDICINE AT HOFSTRA/NORTHWELL

Introduction: Carvajal syndrome is characterized by woolly hair, striated palmoplantar keratoderma and left-sided ventricular cardiomyopathy. It is inherited as an autosomal recessive disorder due to a homozygous mutation in the gene coding for desmoplakin, which truncates the C-terminal of the protein and maps to chromosome 6p24. Signs and symptoms of Carvajal syndrome include: woolly hair that is present from birth, palmoplantar keratoderma that develops after infancy, follicular keratoses on elbows, knees, face, abdomen and lower limbs, clubbing of fingers and rarely mucosal lesions. The desmoplakin (DSP) abnormality can result in arrhythmogenic ventricular cardiomyopathy.

Clinical Presentation: A 2 month old male of Ecuadorian descent presented with oral ulcerations and poor feeding as reported by his mother. The oral lesions were noted at 2 weeks of age. Bilateral dorsal tongue and palatal erosions with sloughing were noted on oral examination. Skin excoriations were noted at sites of electrocardiogram leads. It was also noted that the child had sparse woolly hair that extended on to the forehead and had a hoarse cry.

Intervention and Outcome: Biopsies of the anterior dorsal tongue, lingual epiglottis, and duodenal, gastric, esophageal and rectosigmoid mucosa were performed. The tongue and epiglottis surface epithelium consisted of discohesive squamous epithelial cells with interspersed inflammation and bacterial colonies. Esophageal biopsy showed suprabasilar separation from underlying lamina propria. Direct immunofluorescence studies were negative. Whole Exome Sequence Analysis revealed patient was compound heterozygous for the c.7623delT and c.7623delG pathogenic variants in the DSP gene.

Conclusion: The mucosal lesions of this syndrome can present intraorally, and have a rather unique histopathology characterized by dyskeratosis and discohesion. Patients with this syndrome require regular cardiac evaluations as the cardiac issues are of paramount importance.

MULTIFOCAL ORAL MUCOSAL MELANOACANTHOSIS IN A TEENAGER WITH ECZEMA, A CASE REPORT. DR. KATHLEEN SCHULTZ, DR. PAUL CRESPI, DR. JOHN FANTASIA. ZUCKER SCHOOL OF MEDICINE AT HOFSTRA/NORTHWELL

Introduction: Oral melanoacanthosis is a rare, benign, mucosal pigmentation characterized by rapid growth which may clinically resemble mucosal melanoma. A biopsy is often indicated to confirm this diagnosis and exclude other pathologies. A reactive etiology is suggested, as melanoacanthosis typically