THE ROLE OF EPIGENETIC AND EPISTATIC INTERACTIONS IN THE PATHOGENESIS OF ORAL SUBMUCOUS FIBROSIS. *PROF. RAGHU RADHAKRISHNAN[A], DR. MOHIT SHARMA[B]. [A] MANIPAL ACADEMY OF HIGHER EDUCATION, [B] READER, DEPARTMENT OF ORAL PATHOLOGY, ITS DENTAL COLLEGE, HOSPITAL AND RESEARCH CENTER, GREATER NOIDA - 201308*

Objectives: Epigenetic factors have shown to play an important role in the development of fibrosis. Persistent injury to oral mucosa because of habitual quid chewing resulting in the upregulation of inflammatory cytokines, leading to myofibroblastic persistence underlies an epigenetic aberration in oral submucous fibrosis (OSF). There is however, a paucity of literature showing the role of epistasis in the pathogenesis of OSF.

Findings: Epistasis of IGF-1, TGF-1, COX and Lipoxygenase (LIOX) on PTEN are some of the relevant epistatic interaction relevant to the pathogenesis of OSF. Additionally, NF-κB is epistatic to PTEN, which is specifically arbitrated via p65 subunit of NF-κB.

Conclusions: Given the importance of epigenetic modification in the pathogenesis of OSF the potential role of DNMT and HDAC inhibitors as a therapeutic option holds promise in OSF. Inhibitory microRNAs against profibrotic genes and/or stimulatory microRNAs against antifibrotic genes could be another viable in-vivo therapeutic alternative for the treatment of OSF.

AMELOBLASTIC FIBRO-ODONTOMA: A DISTINCT ENTITY. *DR. MOLLY SMITH[A], DR. CRAIG FOWLER[A], DR. INDRANEEL BHATTACHARYYA[B], DR. REKHA REDDY[B], DR. DOUGLAS DAMM[A]. [A] UNIVERSITY OF KENTUCKY, [B] UNIVERSITY OF FLORIDA*

Background: Ameloblastic fibro-odontoma (AFO) is a benign odontogenic tumor first described by Hooker in 1967. Its etiology and behavior have long been debated, as some investigators have proposed that AFO may represent a stage in development of an odontoma. For this reason, AFO was eliminated from the most recent Odontogenic and Maxillofacial Bone Tumor section of the World Health Organization (WHO) Head and Neck classification system. Occasional AFOs, however, have been found in patients older than the proposed age for odontoma completion (22 years) or present as large radiolucent lesions consisting mainly of the ameloblastic fibroma (AF) pattern with only foci of mineralized product formation. Herein, we present seven cases of AFO, all of which demonstrate particularly aggressive radiographic and/or histopathologic features and do not support the contention that all AFOs represent maturing odontomas.

Materials and Methods: An IRB-approved retrospective search of the oral pathology biopsy services at the Universities of Kentucky and Florida between January 1, 1975 and January 1, 2018 was completed. Cases with appropriate histopathological and radiographic documentation were selected.

Results: Seven patient cases were identified with ages 8, 8, 12, 16, 17, 27, and 29 years. Six cases were from the posterior mandible, and one was located in the posterior maxilla extending into the maxillary sinus close to the floor of the orbit. Only two of the cases have follow-up information, both of which demonstrate no evidence of tumor following conservative treatment.

Conclusion: Although the majority of cases diagnosed as AFO likely represent developing odontomas, we present seven cases in which the clinical, histopathologic, and/or radiographic features suggest that AFO should exist as a distinct entity and be treated similarly to an AF.

GENETIC POLYMORPHISM AND GENE EXPRESSION OF PI3K GENE IN AMELOBLASTOMA. *PROF. AADITHYA B. URS, DR. HANSPAL SINGH, PROF. MAHESH VERMA. MAULANA AZAD INSTITUTE OF DENTAL SCIENCES, NEW DELHI-02*

Objective: Ameloblastoma is a benign and local aggressive odontogenic tumor. Many genes and their respective signalling pathways are involved in the pathogenesis i.e. Patch, SHH, SMO, PI3K, AKT, mTOR etc. PI3K has an important role i.e. cellular quiescence, proliferation, cancer, and longevity in the pathogenesis of Ameloblastoma through PI3K/AKT/mTOR signalling pathway. The study was designed to evaluate the gene expression and gene polymorphism of PI3K gene.
The present study was a prospective preliminary study, which was carried out in 20 patients of confirmed ameloblastoma cases. 5 tooth germs were taken as control to compare. Biopsy was taken with patient's consent. Genomic DNA was extracted to assess the polymorphism of PI3K gene gene sequencing method in exon 9 and exon 20 in association with immunohistochemical analysis respectively.

Findings: Insertion of AA is noticed as the most common variation among 12 samples out of 20 identified at Exon 9 near to the splice site of PIk3CA (g.24751_24752insAA) (chr3:178890652_178890653insAA). However, no variation at Exon 20 was observed. Variant was neither found in ExAC nor 1000G. No differences were noted in the frequency and type of mutations analyzed by sex, age, or histologic features. The gene expression of PIK3CA was significantly higher in tumor epithelial cells. Such genetic polymorphisms are vital because they can be used as biomarkers that indicate for prognosis of tumor and its biological behavior.

Conclusion: These results suggest that common genetic variations in these pathways may modulate risk and clinical outcomes of ameloblastoma. Further replication and functional studies are needed to confirm these findings. It will be of benefit to the patient, if we target the mutation or aberrant protein products at the appropriate time by intervention of précised therapy.

PLASMA CELL GINGIVITIS DUE TO COSMETICS RELATED IODOPROPYNYL BUTYLCARBAMATE (IPBC) ALLERGY IN A TEENAGE FEMALE PATIENT MASKING AS DESQUAMATIVE GINGIVITIS.. *DR. SONIA SANADHYA[A], DR. RONALD BROWN[B], DR. JOHN BASILE[A], DR. RANIA YOUNIS[A], DR. ROY ESKOW[C]. [A] UNIVERSITY OF MARYLAND, SCHOOL OF DENTISTRY, [B] HOWARD UNIVERSITY COLLEGE OF DENTISTRY, [C] BETHESDA DENTAL IMPLANT CENTER*

Plasma cell gingivitis (PCG) is a rare lesion found on the attached and free gingiva, often extending to the mucogingival junction. Clinically, PCG can appear as sharply delineated erythematous lesions which can be accompanied by edema. We present a case of PCG in a 13 year old female patient which