fetuses done by Stout and Collet in 1969 found evidence of two cystic lesions associated with MAAC. These cysts were named median alveolar cyst (MAC). To the best of our knowledge, we are reporting for the first time a bona fide example of MAAC - MAC in a human being.

Case report: A healthy 14-year-old Saudi female with an anterior maxillary diastema was referred to the orthodontics clinic for consultation. Clinical examination revealed a double frenum connecting the maxillary lip and alveolar vestibule. A panoramic film and a cone beam CT revealed a radiolucency between the maxillary central incisors extending from the alveolar crest to the incisive foramen area. The labial cortical plate was missing while the palatal was intact. The radiologist interpretation was "enlarged nasopalatine canal". No other physical or dental abnormalities were evident. Upon surgical exploration, no labial maxillary osseous plate was found however, soft tissue was present and excised. Microscopic examination of the excised tissue revealed a cystic process lined by acanthotic nonkeratinizing stratified squamous epithelium with intracellular edema. In addition, sebaceous glands, islands of squamous epithelium with keratin pearl formation and lymphoid infiltrates were seen within the cystic wall. A retrospective review of the imaging studies coupled to the microscopic findings resulted in diagnosis of median alveolar cyst associated with a median maxillary anterior cleft.

Conclusion: We report a rare case of MAAC with MAC showing a sebaceous component. It is thought that MAC most likely originates from epithelial invaginations derived from the anterior intermaxillary suture. However, the mechanism involved in the formation of these two conditions remains to be elucidated.

IDENTIFICATION OF NOVEL COPY NUMBER ALTERATIONS IN AMELOBLASTOMA AND AMELOBLASTIC CARCINOMA FROM NIGERIA.

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Background and Objectives: Ameloblastoma is a benign odontogenic neoplasm, characterized by local invasiveness, facial deformity, tooth displacement, a high rate of recurrence, and malignant transformation. It accounts for 63% of odontogenic tumour in Nigeria. Recently, studies in the genomic landscape of ameloblastoma have identified a number of consistent alterations that may be useful for therapeutic intervention. To date, no whole genome survey of ameloblastoma and ameloblastic carcinoma has been published.

Methods: DNA was extracted from RNALater stored tissue using the DNeasy Tissue Kit (QIAGEN), from a cohort of ten ameloblastoma and three ameloblastic carcinoma from UCH, Ibadan, Nigeria. Whole genome analysis was performed using the Oncoscan FFPE Assay Kit (Affymetrix). Data was analysed using Nexus Express for Oncoscan 17.0 and Somatic Mutation Viewer 1.0.1.

Findings: Ameloblastoma (n=10) showed a mean genome change of 9.7%, with a mean of 88.7 copy number (CN) aberrations and 7.5% of loss of heterozygosity (LOH), whereas the ameloblastic carcinomas (n=3) had a mean genome change of 6.8% with a mean of 87.3 copy number (CN) aberrations and 3.6% of loss of heterozygosity (LOH). All tumours (benign and malignant) showed CN gain at 8q23.3, affecting the CSMD3 gene. Other commonly affected regions included LOH at 1p34.2-p34.1 and 2q11.2, among others. Ameloblastoma and ameloblastic carcinomas shared somatic mutations in BRAFV600E, EGFR, KRAS and PTEN genes. One ameloblastoma showed a mutation in TP53 and two (66.7%) ameloblastic carcinomas showed a mutation in the PIK3CA gene, which was not observed in the ameloblastoma cohort.

Conclusions: Ameloblastoma and ameloblastic carcinoma do not show extensive genome changes indicative of genomic instability. We have identified novel areas of CN gain and LOH that require further investigation. The mutational profile of these lesions is similar to that reported in the literature. Funding: Pathological Society of Great Britain.

THE IMPORTANCE OF IMMUNOHISTOCHEMISTRY AND MOLECULAR STUDIES FOR DIAGNOSING EWING'S SARCOMA OF THE MANDIBLE: A CASE REPORT..

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Introduction: Ewing's sarcoma (ES) is a malignant small round cell neoplasm primarily affects the bone. It was first described by James Ewing in 1921. ES accounts for 6-10% of all primary malignant bone tumors. It is most commonly found in children between 10-15 years of age. 1% to 2% of cases of ES affect the craniofacial bones. Only a few cases have been reported in the mandible. Here we report a case of EW in the mandible and the use of immuno-histochemistry and molecular studies to confirm its diagnosis.

Clinical presentation: A 16 year old female patient was seen at the Department of Oral and Maxillofacial Surgery in King Fahad Medical City. Extra-oral examination revealed diffuse painless swelling on left side of the mandible with reduced mouth opening. Intraorally, an ulcerated large mass was present. CBCT revealed ill-defined radiolucency involving the posterior part of the mandible extending to the ramus. MRI showed a destructive mass in the left mandible with a soft tissue component occupying the left masticator space. PET/CT showed a FDG avid left cervical large mass. An incisional biopsy was taken. Microscopically, the specimen revealed the presence of islands and sheets of monotonous malignant cells infiltrating the bone. The nuclei of the malignant cells were round to oval in shape with fine dispersed chromatin and one or two indistinct nucleoli. The neoplastic cells were positive for CD99 and Fli1 and negative for SATB2. Chromosomal translocation t (11:22) involving the EWS and FLI-1 gene was identified using FISH. Patient was treated with chemotherapy.

Conclusion: We reported a case of a malignant tumor with an immunoprofile of Ewing Sarcoma that was confirmed with the identification of chromosomal translocation by molecular study.

CONDYLAR HYPERPLASIA: RADIOLOGICAL, HISTOLOGICAL AND IMMUNOHISTOCHEMICAL COMPARATIVE ANALYSIS.

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