

rare with only 16 cases reported in the literature, none of which have been reported in the mandible.

Case Report: A 14-year-old female presented with a lobulated gingival nodule measuring 1.2×0.8 cm between teeth #20 and 21. Radiographically, the lesion was a well-circumscribed, non-corticated multilocular radiolucency between the roots of #20 and #21 measuring 1.4×1.0 cm, extending from the alveolar crest to close to the root apices. An incisional biopsy was performed. Microscopically, the lesion consisted of a non-encapsulated, multilobular tumor composed of a proliferation of spindle and epithelioid cells within a delicate myxochondroid stroma. Tumor cells were positive for S-100, vimentin, neuron-specific enolase (NSE) and epithelial membrane antigen (EMA) and negative for CAM5.2, AE1/3, SMA, SOX-10, CD57, glial fibrillary acidic protein (GFAP), and p63. Ki-67 labeled less than 5% of the cells. These findings are suggestive of STM, and the bone and soft tissue consultant pathologist concurred with this diagnosis.

Discussion: The putative cell of origin for this tumor is a stem cell within the soft tissues that differentiates towards a cell with myoepithelial phenotype. Unlike salivary gland myoepithelioma which harbors *PLAG1* and *HMG2* rearrangements, STM harbors the *EWSR1* rearrangement in up to 44% of the cases. Ectomesenchymal chondromyxoid tumor may represent the same entity since it has similar histomorphology and immunohistochemical profile but exhibits *EWSR1* rearrangement in only 25% of cases. To our knowledge, this is the first report of a STM occurring in the mandible.

PROGNOSTIC CLASSIFIER FOR ORAL POTENTIALLY MALIGNANT DISORDERS: AN INTEGRATED HISTOPATHOLOGICAL AND MOLECULAR APPROACH. DR. HANS PRAKASH SATHASIVAM, PROF. PHILIP SLOAN, DR. RALF KIST, DR. MAX ROBINSON. NEWCASTLE UNIVERSITY

Objectives: Oral squamous cell carcinoma (OSCC) is associated with a high degree of morbidity and mortality. OSCCs are often preceded by oral potentially malignant disorders (OPMD) which have a higher propensity to undergo malignant transformation (MT) compared to clinically normal oral mucosa. Currently there is no reliable method to determine which OPMD cases will undergo MT. This study was performed to construct a prognostic classifier for patients with OPMD by integrating clinical, histopathological and molecular factors and to discover a gene expression signature that characterises OPMD with a high risk of undergoing MT.

Findings: Statistical analysis of an OPMD patient cohort (23 MT vs. 25 with no MT) showed that site of initial OPMD ($p=0.043$), binary oral epithelial dysplasia (OED) grading ($p=0.009$) and loss of heterozygosity at 3p/9p/17p ($p=0.026$) were statistically significant. Other demographic factors, clinical features and the WHO 3-tiered OED grading system were not statistically significant. Gene expression experiments revealed several genes that were differentially expressed between OPMD that underwent MT and those that did not [false discovery rate of < 0.05]. Statistical model building was performed, and the outputs were used to construct a prognostic classifier.

Conclusions: Our findings show that a classifier combining histopathological and molecular factors outperforms conventional methods for prognosticating clinical outcome in patients with OPMD. We have also shown that formalin-fixed paraffin-embedded tissue can be used to generate a molecular classification with clinical utility.

SOMATIC DRIVER MUTATIONS IN ORAL AND SINONASAL MUCOSAL MELANOMA. A REFERRAL CENTRE EXPERIENCE IN MEXICO CITY. DR. JESSICA LISSETTE MALDONADO MENDOZA^A, DR. VELIA RAMIREZ-AMADOR^A, DR. GABRIELA ANAYA SAAVEDRA^A, DR. ERIKA RUÍZ GARCÍA^B, DR. HÉCTOR MALDONADO MARTÍNEZ^B, DR. EDITH FERNÁNDEZ FIGUEROA^F, DR. ABELARDO MENESES GARCÍA^B. ^A UNIVERSIDAD AUTÓNOMA METROPOLITANA XOCHIMILCO, ^B NATIONAL CANCER INSTITUTE OF MEXICO, ^F COMPUTATIONAL GENOMICS, NATIONAL INSTITUTE OF GENOMIC MEDICINE

Objective: Oral and sinonasal mucosal melanomas (OSNMM) are aggressive tumors with low survival and few therapeutic alternatives. The aim of this study was to describe the prevalence of mutations in *NRAS*Q61K, *BRAF*V600E, *CKIT*L576P, *K624E*, *MITF*E318K and *PTEN*R130; and to analyze the clinic-pathological features present.

Findings: Cross-sectional and observational study that included cases with OSNMM from the National Cancer Institute of Mexico City and the Oral Pathology Laboratory of UAM-X (January 2000-December 2016). Demographic and clinical data were obtained, and histopathological diagnosis was confirmed. Genomic DNA was obtained and molecular analysis was carried out through quantitative polymerase chain reactions (qPCR) (Customized Biomarker somatic mutation Array[®], Qiagen). The statistical analysis was performed using the SPSS v20 software.

Forty-eight cases were included, 56.2% were sinonasal melanomas (SNM) and 43.7% oral melanomas (OM). The median age of the individuals was 60 years (Q1-Q3 = 51-74), 54.2% of the cases were men. Higher symptomatology percentages were found among SNM (100% vs. 52.9%, $p < 0.001$). At the histopathological analysis, 97% of the tumors showed vertical and infiltrative growth, SNM showed a greater amount of necrosis (68% vs. 32%, $p = 0.006$) in comparison with OM. Eight (16.6%) OSNMM presented at least one mutation: 6/28 (21.4%) SNM cases and 2/20 (10%) OM. From 48 OSNMM, three (6.6%) showed V600E *BRAF* mutation, 3/48 (6.2%) Q61R mutation *NRAS* and 2/48 (4.1%) K624E mutation *KIT*. No mutations were found in *MITF* or *PTEN*.

Conclusions: A low prevalence of mutations was found. Somatic driver mutations might not be related with OSNMM development; thus, the current biological agents (vemurafenib and imatinib) may probably be ineffective against OSNMM. It is necessary to continue the search of other molecular alterations to suggest therapeutic alternatives for these tumors, such as: proteins amplification (c-*KIT*) or epigenetic mechanism which might regulate the genetic expression (miRNAs).

APPLICATION OF DEEP LEARNING ALGORITHMS IN DETECTION OF MITOTIC EVENTS IN ORAL SQUAMOUS CELL CARCINOMA USING CELLPHONE IMAGES. DR. AMBER KIYANI^A, DR. HASSAN AQEEL^B, MR. ASJID TANVEER^B, MR. WAJAHAT NAWAZ^B, DR. SYED ALI KHURRAM^C. ^A RIPHAH INTERNATIONAL UNIVERSITY, ^B NATIONAL UNIVERSITY OF SCIENCE AND TECHNOLOGY, ^C UNIVERSITY OF SHEFFIELD