

consistent and reproducible results. Factors such as endogenous peroxidase blocking, antibody concentration and incubation time, chromogen system and counterstains are important parameters that should be optimized for individual studies.

**EXPRESSION OF THE CANCER STEM CELL MARKER ALDH1 IS INCREASED IN THE BUDDING AREA OF ORAL SQUAMOUS CELL CARCINOMA.** DR. GIOVANNA RIBEIRO SOUTO<sup>A</sup>, DR. HELVÉCIO MARANGON JUNIOR<sup>B</sup>, MS. VICTÓRIA VASCONCELLOS MOREIRA MELO<sup>A</sup>, MS. ÂNGELA BRAGA CAIXETA<sup>A</sup>, DR. PAULO EDUARDO ALENCAR SOUZA<sup>A</sup>, DR. MARIA CÁSSIA FERREIRA AGUIAR<sup>C</sup>, DR. MARTINHO CAMPOLINA REBELLO HORTA<sup>A</sup>. <sup>A</sup> SCHOOL OF DENTISTRY, PONTIFICAL CATHOLIC UNIVERSITY OF MINAS GERAIS, <sup>B</sup> CENTRO UNIVERSITÁRIO DE PATOS DE MINAS, <sup>C</sup> SCHOOL OF DENTISTRY, FEDERAL UNIVERSITY OF MINAS GERAIS

**Objectives:** This study aimed to evaluate the expression of the cancer stem cell marker ALDH1 and its association with tumor budding, a morphological marker of cancer invasion, in oral squamous cell carcinoma (OSCC).

**Findings:** 163 OSCC samples were obtained by incisional biopsies. Immunohistochemistry was performed to detect positive cells for ALDH1 (cancer stem cell marker) and for AE1/AE3 (multi-cytokeratin to identify OSCC cells in tumor budding evaluation). A positive expression of ALDH1 was observed in 47.24% of the samples. In the tumor budding evaluation, samples were classified as low-or high-intensity tumor budding. Association between the ALDH1 expression and tumor budding was assessed using the chi-square test. However, no association was observed ( $p > 0.05$ ). In samples with high-intensity tumor budding, differences in the ALDH1 expression between the budding area and the area outside the budding were evaluated using the McNemar test. The ALDH1 expression was higher in the budding area than in the area outside the budding ( $p < 0.05$ ).

**Conclusion:** The findings reinforce the idea that cells at the tumor budding area show phenotypic characteristics of cancer stem cells. The debate concerning the model of oral carcinogenesis by cancer stem cells was also strengthened. Supported by FAPEMIG (APQ-01806/14 and PPM-00653-16).

**MESENCHYMAL CHONDROSARCOMA OF THE MAXILLA: A CASE REPORT AND REVIEW OF THE LITERATURE.** DR. MARK LERMAN. TUFTS UNIVERSITY

Mesenchymal chondrosarcoma (MCS) is a rare subtype of chondrosarcoma accounting for less than 2% of all chondrosarcomas, first described by Lichtenstein and Bernstein in 1959. The majority develop as intraosseous lesions, and the jawbones are among the most common primary sites. The peak incidence is between ages 10-30. In this report, we present a case of MCS diagnosed in the maxilla and review the literature for previously reported cases. A 20-year-old female presented to oral and maxillofacial surgery with a five-month history of sinus congestion. A panoramic radiograph demonstrated a diffuse radiopacity of the right maxillary sinus and a CT scan revealed extension of the lesion to the orbit. A biopsy exhibited a

proliferation of cells with basophilic cytoplasm varying in appearance from round to spindled. Numerous atypical mitotic figures were noted and foci of chondroid material were scattered throughout the lesion. Immunohistochemical studies revealed diffuse reactivity of the cellular proliferation with CD99 and positivity of S-100 within the cartilaginous tissue. These findings were consistent with a diagnosis of mesenchymal chondrosarcoma and genetic studies confirmed HEY1-NCOA2 fusion to support the diagnosis. The patient was referred to a sarcoma center for further management. The literature was reviewed for previous cases of MCS of the maxilla. Including the current case, there are 41 cases with a male:female ratio of 1:1.4. The age at diagnosis ranged from 9-83 years with a mean age of 30 years and median age of 26 years.

MCS is a rare high-grade malignancy with a ten-year survival of 10-54%. While some studies have suggested that MCS of the jawbones may have an improved prognosis compared to those originating in other sites, others have disputed that finding. Familiarity with the radiographic and histopathologic features of MCS may aid in the diagnosis of this rare sarcoma.

**PHEOPHORBIDE A-MEDIATED PHOTODYNAMIC THERAPY INDUCED ENDOPLASMIC RETICULUM STRESS, LEADING TO INDUCTION OF APOPTOSIS IN HUMAN ORAL SQUAMOUS CARCINOMA CELLS.** PROF. JUNG-HOON YOON<sup>A</sup>, PROF. JUN LEE<sup>B</sup>. <sup>A</sup> DEPARTMENT OF ORAL AND MAXILLOFACIAL PATHOLOGY, COLLEGE OF DENTISTRY, WONKWANG BONE REGENERATION RESEARCH INSTITUTE, DAEJEON DENTAL HOSPITAL, WONKWANG UNIVERSITY, DAEJEON 35233, REPUBLIC OF KOREA, <sup>B</sup> DEPARTMENT OF ORAL AND MAXILLOFACIAL SURGERY, COLLEGE OF DENTISTRY, WONKWANG BONE REGENERATION RESEARCH INSTITUTE, DAEJEON DENTAL HOSPITAL, WONKWANG UNIVERSITY, DAEJEON 35233, REPUBLIC OF KOREA

Photodynamic therapy (PDT) has been developed as an alternative for malignant tumors that uses a photosensitizer. Our group recently synthesized a photosensitizer Pheophorbide a (Pa) from chlorophyll-a. However, the molecular mechanisms by which it causes anti-cancer activity in oral squamous cell carcinoma (OSCC) are not well understood. Here, we showed that Pa-PDT inhibited effectively the proliferation of FaDu cells. Flow cytometry and western blot showed that Pa-PDT induced intrinsic apoptosis cell death pathways in FaDu cells. Next, we checked Pa-PDT induced ER stress in FaDu cells that it was observed as demonstrated by accumulation of the ER stress marker. Pa-PDT also induced autophagy in FaDu cells was evidenced by the increased levels of the autophagic protein marker expression. Inhibition of ER stress pathway using 4-phenylbutyric acid (PBA) 1mM decreased CHOP, with induced inhibition of cell deaths. Also, the inhibition of ER stress enhanced Pa-PDT mediated autophagy. This result suggest that Pa-PDT induced ER stress trigger apoptosis and inhibition of ER stress decreased Pa-PDT mediated cytotoxicity through an increase of autophagy. This study was supported by Basic Science Research Program through the National Research Foundation of Korea (NRF) funded by the Ministry of Education, Science and Technology (No. NRF-2016R1D1A1B01006388).