

NON-LASER PHOTOBIMODULATION AND BENZODIAZEPINES IN BMS MANAGEMENT

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Aim: the aim of this study was to compare a photobiomodulation device (Biopton™) used as light therapy and Benzodiazepines (BDZ) in burning mouth syndrome (BMS) treatment.

Methods: clinical and demographical data of 43 patients with a clinical diagnosis of BMS were randomized in two split groups. Group A was treated with Biopton™ light therapy, whereas group B was treated with BDZ. Visual Analogue Scale (VAS) was used to assess the therapeutic effects of the given treatments in both groups. Outcomes were recorded at the beginning of the treatment (t0) and at the end (tf).

Results: reduction of VAS was recorded in both groups with a significant improvement ($p < 0.05$). In Group A, the VAS decreased significantly (t0: 7.07: tf: 4.64). Also, in group B, mean

VAS decreased significantly from 7.52 to 6.41. This study does not show a statistically significant difference between the two therapies ($P = 0.064$; $p > 0.05$).

Conclusion: results of the present study showed that both treatment were effective in reducing the patient-perceived pain and discomfort, although Biopton™ light treatment showed a slightly bigger reduction in VAS points. As Biopton™ light therapy has no known side-effects, and is a non-invasive procedure, this can allow for management of BMS patients in which customary medicaments with BZD may not be feasible. Further studies may focus on using light therapy and pharmaceuticals in combination, to try and understand if they may work better together in helping BMS patients.

IMMUNOHISTOCHEMICAL DETECTION OF BRAF V600E MUTATION IN AMELOBLASTOMA

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Aim: the ameloblastoma is the most frequent odontogenic tumor. Despite its benign nature, the local aggressiveness and a high recurrence rate characterize some entities. Literature data suggest the BRAF V600E mutation is involved in 46-82% of ameloblastomas, offering a biological rationale for developing new therapeutic strategies. The study aims to evaluate the correlations between the presence of BRAF V600E mutation and the clinicopathological data in a cohort of ameloblastoma patients.

Methods: 50 ameloblastomas were subjected to immunohistochemical analysis using VENTANA anti-BRAFV600E Mouse Monoclonal Primary Antibody. A uniform, cytoplasmic staining of viable tumor cells was considered as “positive” staining. Differences among ameloblastoma groups were established by

Chi-square and Mann-Whitney tests. A p-value < 0.05 was accepted as significant.

Results: the 46% of ameloblastomas harbored the BRAF V600E mutation. Its presence was significantly associated with the mandibular site ($p = 0.0004$) and unicystic histotype ($p = 0.0383$). All BRAF unicystic ameloblastomas arose in the mandible ($p < 0.0001$). There was a trend between the presence of BRAF and the late onset of recurrent ($p = 0.0521$), without reaching a significance difference in term of disease-free survival time.

Conclusion: our results suggest the MAP-kinase pathway could contribute on ameloblastic tumorigenesis. Moreover, they could indicate the anatomical specificity of the driving mutations of mandibular tumors, providing a biological rationale for developing new targeted therapies.