ministration of the photosensitizer, 5-Aminolaevulinic Acid (5-ALA) in 20% oral gel, 90' before laser exposure. The laser adopted was a 635nm diode (Lambda Spa, Vicenza, Italy). The irradiation was performed twice a week, each second day. The protocol previewed a maximum of 6 applications, less in case of clinical disappearance of the lesion. Laser power was 100mW, in CW with scanning no contact movement and total energy of 100mJ. The exposure time was 1000 seconds, in 5 cycles of 3', with intervals of 3' to permit the synthesis of the Protoporphirin IX, and a final cycle of 100 seconds. After 5 applications the lesion totally disappeared, without side effects scarring or pain both in immediate and follow up period. Controls were scheduled each 3 months; at 18 months no recurrence was observed. In this case the PDT resulted very helpful in the management of VPL. More studies and cases are needed to evaluate its real efficacy for the treatment of these lesions.

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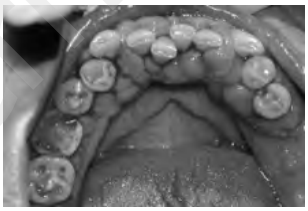
Case Report

Drug-induced gingival hyperplasia, treatment with diode laser



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Background. Gingival overgrowth/hyperplasia can be attributed to several causes, but drug-induced gingival overgrowth/hyperplasia arises secondarily to prolonged use of antihypertensive drugs, anticonvulsants and immunosuppressants. A gingivectomy performed with a laser is a short, easy procedure that produces an immediately dramatic effect. Compared to a scalpel gingivectomy, there is excellent hemostasis, which improves visualization, requires less need for periodontal packing, and results in minimal postoperative discomfort[1,2].

Case report. A 70-old-male patient treated with antihypertensive therapy with "Calcium channel blockers" for about 1 year (amlodipine). He had severe gingival overgrowth, hyperemia, easy bleeding, pain and masticatory function compromised. Changing of antihypertensive drug and the execution of a professional treatment of oral hygiene did not allow the resolution of hyperplasia. A diode 810 laser was used to excise the gingival overgrowth. The used parameter was 2W in continuous wave with a activated fiber of 400 mm. This treatment permitted the resolution of the case without any complication.

Conclusion. The use of diode laser in the present case proved to be effective in the removal of large amounts of hyperplasic gingival tissue and resulted in fast heeling and mild discomfort.

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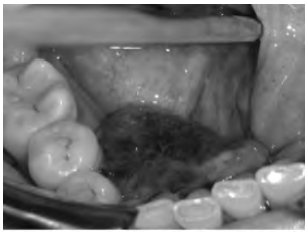
Case Report

Mandibular brown tumor as the first manifestation of primary hyperparathyroidism: a case report



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Background. Brown tumors are erosive bony lesions caused by rapid osteoclastic activity and peritrabecular fibrosis

due to hyperparathyroidism, resulting in a local destructive phenomenon. These lesions are non-neoplastic and they appear as a mass with partly cystic and partly solid areas.
Clinically, they are slow-growing lesions that can be locally destructive resulting in variety of symptoms such as significant bone swelling, pain and pathological fracture; they can be mistaken for a neoplasm.
The classical "brown tumor" is commonly seen in ends of long bones, the pelvis and ribs. Facial involvement is rare and, when present, usually involves the mandible.
Parathyroid adenoma (and most rarely adenocarcinoma) is the commonest cause of primary hyperparathyroidism and usually presents with symptoms/signs of hypercalcaemia.
Case Report. We report a case of 71-year-old male with a mandibular brown mass in absence of symptoms. The lesion was expansive and osteolytic, with invasion of the floor of the mouth. Histology revealed the presence of an intrabone giant cell lesion. Blood tests demonstrated elevations in parathyroid hormone (PTH) concentrations. This suggested the diagnosis of hyperparathyroidism initially manifesting as a brown tumor of the mandible. Posterior explorations confirmed the existence of a parathyroid adenocarcinoma as the cause of the condition.
Conclusion. The aim is to alert the clinicians to include this entity although extremely rare in the differential diagnosis of red/brown oral masses.

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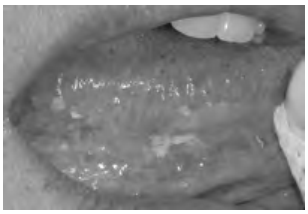
Case Report

Loss of p53 protein expression in leukoplakias may reveal the presence of an oral squamous cell carcinoma: a case series analysis



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Mutations in the TP53 gene leading to loss of function are the most common genetic damages found in human cancers and Oral Squamous Cell Carcinoma (OSCC). TP53 mutations may result in a over-production of p53 inactive proteins which accumulate in the epithelium either due to blocking by another protein or due to partial degradation, or instead in an abrogation of p53 wild function by epigenetic mechanisms. Immunoistochemical analysis of p53 is a simple method used for the detection of p53 protein, and its evaluation has been widely recommended in oral leukoplakias to a better understanding of the potential risk of malignancy. In the present series report we describe four patients referred to our Department following the appearance of an asymptomatic lesion of the tongue. Patients underwent an incisional biopsy for histology and immunoistochemical analysis of p53 protein. In all cases, the lesions were clinically and histologically classified as oral leucoplakias with sign of mild dysplasia. Immunoistochemical analysis showed in all cases a negative staining of p53 protein. All four lesions were subsequently surgically removed and histological analysis of the whole lesion showed in all cases the presence of a microinvasive carcinomas. Thus, in all four cases the histological conclusions from an incisional biopsy as a "snapshot" of the whole lesion have understimate the true nature of the whole lesion. Predictive value of p53 negative staining is not well described in literature while p53 over-expression has been proposed in numerous studies as a reliable marker associated to oral carcinogenesis, although most of the studies utilized antibodies that cannot discriminate in the single case wild or mutated form[1]. Instead, the presence of p53 negative staining could be considered a specific value representative of an arrest of p53 protein synthesis and seems to be a useful marker for early diagnosis of OSCC also in absence of histological recognizable signs.

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