Collagen turnover induced by cellular connective tissue cytokines of drug induced gingival overgrowth and hereditary gingival fibromatosis (Histological and immunohistochemical comparative study)

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Abstract

Background
Gingival overgrowth (GO) is usually associated with multiple factors including immunosuppressive agents as cyclosporine (CsA) and Tacrolimus (TAC), and hereditary gingival fibromatosis (HGF).

Objective
To compare the expression of TGF-ϐ1, PDGF, TIMP-1 and MMP-9 at the molecular and cellular levels in patients receiving (CsA or TAC) and patients manifested (HGF), to cast some light on the pathogenic mechanism potentially involved in the collagen (COL) turnover of both conditions.

Subjects
and methods: Gingival tissue samples were obtained from patients undergoing therapy with CsA (n = 6), TAC (n = 6), HGF (n = 3) as well as control tissues from systemically healthy control (n = 6). Tissue sections were immune-stained by labeled streptavidin-biotin (DAB) technique, using monoclonal antibodies against TGF-ϐ1, PDGF-ϐ, TIMP-1 and MMP-9.

Results: comparison of type of expression among the studied groups, showed significant diffuse expression of TGF-ϐ1 and PDGF-ϐ in group I and II with P value = 0.58 and 0.38 respectively. The expression of MMP-9 was significantly diffuse in TAC or CsA group when compared to HGF group with P value = 0.38, mean while there was a significant diffuse expression of TIMP-1 in HGF group when compared to TAC or CsA group with P value = 0.38.

Conclusions
In conclusion the biological mechanisms behind the drug induced gingival overgrowth (DIGO) and HGF is targeting COL turnover but in different ways. Also, this may explain the need for periodic surgical correction of the gingival form and architecture in HGF cases, unlike the DIGO which can be overcame by replacement of CsA by TAC with improvement of oral health.

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