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Immunohistochemical expression of epithelial mesenchymal transition markers (vimentin, e-Cadherin and alpha –smooth muscle actin) in hereditary gingival fibromatosis in comparison to inflammatory gingival hyperplasia.

A Thesis

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Abstract

Background:

Gingival enlargement is the most common characteristics of the gingival disease. Clinically described as gingival enlargement or overgrowth. Gingival enlargement may be hereditary or acquired. Inflammatory gingival enlargement is an inflammatory restraint to local irritant correlating with the gingiva, the clinical manifestation begin as ballooning of the papilla or marginal gingiva, the enlargement gradually increase in size and extent to become generalized. Hereditary gingival fibromatosis is a rare and heterogeneous group of disorder that develop as slowly progressive, local or diffuse enlargement, it affects both gender equally. The molecular and biochemical mechanism that induce this pathological condition is not well understood. Epithelial mesenchymal transition theory is one of the diverse theories attempted to explain the mechanism of advancement of this benign lesion.

Aims:

To evaluate and compare the Immunohistochemical expression of Vimentin, E chadherin and Alpha-smooth muscle actin in hereditary gingival fibromatosis and in inflammatory gingival hyperplasia.

Material and method

The study was performed on thirty one formalin- fixed paraffin embedded tissue blocks, fifteen of which diagnosed as inflammatory gingival overgrowth, which were obtained from archives of the department of Oral Pathology, Collage of Dentistry, University of Baghdad from 1971 to 2018.

The second group was composed of sixteen formalin- fixed paraffin embedded tissue blocks which diagnosed as hereditary gingival fibromatosis, obtained from archives of the department of Oral Pathology, Collage of Dentistry, University of Baghdad from 1971 to 2018. The diagnosis of each case

was confirmed by the examination of hematoxylin and eosin (H&E) sections by a specialized pathologist. Tissue preparation was done by fixing all tissue specimens in 10% buffered formalin for at least 24 hours, and processed routinely into paraffin blocks. Serial sections From each paraffin embedded tissue block (samples and controles) were cut; Sections of 4µm thickness were mounted on glass slides, stained with hematoxylin and eosin (H&E), and histopathologically re-evaluated. Re-evaluation and conformation of diagnosis were made for each case by specialized pathologist. For each case, at least four tissue sections of 4um thickness were cut and mounted on positively charged slides for immunohistochemical staining with polyclonal antibodies (vimentin, e-chadherin and alpha- smooth muscle actin). Immunohistochemical signal specifity was demonstrated by the absence of immunostaining in the negative control slides and its presence in the recommended positive controls. Fibroblast cells with brown staining were considered positive cell, while the fibroblast cells without brown stain were considered as negative cells. Blindly assessment for all slides without prior knowledge of the clinicopathological parameters was done, the histological examination were calibrated by a specilized pathologist.

Result:

Increase in the immunohistochemical expression of vimentin and e-chadherin (score and percent), while weak immunohistochemical expression of alpha-smooth muscle actin (score and percent) both in hereditary gingival fibromatosis and in inflammatory gingival hyperplasia,

Non-significant statistical difference in the immunohistochemical expression of vimentin, e-cadherin and alpha smooth muscle actin between the hereditary gingival fibromatosis and inflammatory gingival hyperplasia.

Conclusion:

Epithelial mesenchymal transition process could operate in the pathogenesis of hereditary gingival fibromatosis and inflammatory gingival hyperplasia.