Morphology and distribution of CD1a positive Langerhans cells in normal and malignant buccal mucosae.

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AIM & OBJECTIVES: To study the morphology and distribution of CD1a positive Langerhans cells in the normal and malignant buccal mucosae and to look for correlation with the method of tobacco consumption as well as the clinical staging of malignancy.

METHODS:

With the approval of the Institutional Review Board, consented patients were recruited for the study. Normal buccal mucosa was collected from 16 patients who underwent substitution urethroplasty using buccal mucosal graft and malignant buccal mucosa was collected from 15 patients who underwent excision of buccal carcinoma. The tissues were fixed in neutral formalin, processed for immunohistochemistry and embedded in paraffin. 4-5 µm thick sections were taken, mounted on PLL coated slides and stained with Rabbit monoclonal CD1a antibody. The stained slides were studied at 40X magnification under a microscope. The CD1a positive LCs were identified, their number counted and classified into morphological types based on the presence of processes and branching pattern across a length of 25 mm epithelium per tissue. The diameter of one hundred CD1a positive LCs were measured. The morphometric analysis was done using Cellsens image processing software (version 1.4) and statistical analysis was done using SPSS version 16.

RESULTS AND CONCLUSION:

Two types of CD1a positive Langerhans cells (LCs) were identified in the normal and malignant buccal mucosae- a) typical dendritic LCs and b) non-dendritic LCs. These non-dendritic CD1a positive LCs were identified for the first time in the buccal mucosa using CD1a marker. While most of the typical CD1a positive LCs were located in the suprabasal layer, the non-dendritic CD1a positive LCs were present in the basal layer. In the well differentiated squamous cell carcinoma they were scattered throughout all the layers of the epithelium. CD1a positive LCs formed aggregations around a) basement membrane in-folds, b) intraepithelial capillaries, c) intraepithelial lymphocytic aggregations, d) subepithelial lymphocytic aggregations as well as e) migrating across the basement membrane. Apposition of the LCs with the lymphocytes were observed in the epithelium and the subepithelium. There was no significant difference in the number and diameter of LCs between the normal and malignant buccal mucosae. Of the LCs showing typical dendritic morphology the cells with a single process were predominant. Overall the number of CD1a positive non-dendritic LCs were significantly higher in the normal compared to malignant buccal mucosa. The mean diameter of the CD1a positive LCs was significantly lesser in patients who used smokeless forms of tobacco. There was a significant decrease in the mean diameter of the CD1a positive LCs as the tumour stage progressed.

The results of the current study might throw light into the role of LCs in the buccal mucosal immunity.