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PLENARY ABSTRACT

Oral lichen planus – the controversy continues……

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Oral lichen planus (OLP), lichenoid contact reactions (LCR), lichenoid drug reactions (LDR) and graft versus host disease (GVHD) can affect the oral mucosa and belong to the group of lichenoid reactions (LR). Thus, LR does not represent one disease entity but should be considered as a pathological reaction pattern instigated by different etiological entities. This is supported by the fact that the four different lichenoid reactions involve different immunopathological mechanisms. OLP is a mucocutaneous disease which presumably involves autoreactive T-cells. LCR are seen in oral mucosa in contact with dental materials, and LCR is considered to be a delayed hypersensitivity reaction against the filling material. Recently it has been suggested that bacterial antigens may also induce LCR. LDR are elicited by pharmacological drugs which may act as haptens linked to self-proteins. GVHD is observed in patients treated with allogeneic stem cell transplantation. Immune cells from the donor recognize the recipient's tissue as foreign causing a T cell dominated inflammatory cell infiltrate in the mucosa underlying the oral epithelium. The explanation behind the different clinical manifestations of LR is related to the magnitude of the subepithelial inflammation. A mild degree of inflammation may provoke the epithelium to produce hyperkeratosis, whereas more intense inflammation will lead to partial or complete deterioration of the epithelium, histopathologically perceived as atrophy, erosion or ulceration. This corroborates with the fact that most erythematous and ulcerative lesions are surrounded by white reticular or papular structures. An inflammatory gradient may be formed where the central part comprise an intense inflammatory process while the periphery is less affected and the epithelial cells are able to respond with hyperkeratosis.

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