Pathogenesis of Adamantiades-Behçet's disease.

Abstract:
The aetiology of Adamantiades-Behçet's disease remains unknown and its pathogenesis is not fully understood. Linked intrinsic and extrinsic factors are thought to contribute to the development of the disease, which probably occurs by environmental triggering of a genetically determined disorder. Transmission is solely vertical, indicating that the disease is not contagious. Genetic factors have been investigated and a significant link of HLA-B51, especially of HLA-B5101, has been identified. However, none of the functional correlates of the disease appear to be restricted by HLA-B51. Recently, the role of the genes encoding TNF, Tap proteins and MICA has been emphasised. Extrinsic pathogenetic candidates have been identified, including bacterial (Streptococcus sanguis, Mycoplasma fermentas) and viral (human herpes virus) antigens and environmental pollution, which may cross-react with oral mucosal antigens and induce immunological mechanisms. A common factor linking some of the possible pathogenetic agents is extrinsically induced tissue stress or heat shock proteins, which react with host tissues and elicit significant Th1 cell responses. Neutrophils may also play a role in the pathogenesis of the disease, as they are attracted by macrophages and activated endothelial cells, which release cytokines and chemokines (especially IL-8) at the site of the lesions, and thus contribute to tissue damage and self maintenance of inflammation. Endothelial activation leading to a
chronic local inflammation process together with platelet and serum factors enhance coagulation and thrombosis.

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