0062

EBV Seroprevalence and Salivary Viral Load in Oral Lichen Planus

J. Kakar[1], L. Sokolovska[2], S. Svirskis[2], I. Cema[1], M. Murovska[2]
[1]Clinic of Oral and Maxillofacial surgery and Oral medicine, Stomatology Institute, Riga Stradins University, Riga, Latvia, [2]Institute of Microbiology and Virology, Riga Stradins University, Riga, Latvia

Objectives Oral lichen planus (OLP) is a chronic inflammatory condition of the oral mucosa with suspected autoimmune origins. Epstein-Barr virus (EBV) has been implicated in autoimmune diseases due to its ability to modulate immune responses. This study aims to investigate the association between EBV serological markers, viral load in saliva and OLP clinical severity.
Methods 24 OLP patients who visited RSU Clinic of Oral Medicine, RSU Institute of Stomatology during 17.06.2021- 02.03.2023 were included in the study. Demographic data and clinical characteristics of OLP were recorded. Serum samples were collected and EBV-specific IgG and IgM class antibody levels were measured using ELISA. Virus-specific genomic sequences were detected in DNA samples isolated from saliva by real-time PCR. JMP17 and GraphPad Prism9 were used to analyse correlation between levels of IgG and IgM class antibodies, EBV load in saliva and clinical parameters
Results EBV-specific IgG class antibody level surpassed the upper detection limit (>750 IU/ml), suggestive of past EBV exposure or persistent infection in 9 (37.5%) patients. In almost all patients except one virus-specific IgM class antibody level was below detection threshold (<10 IU/ml), indicating absence of acute infection or reactivation of persistent EBV infection. EBV load was detected in 17 (70.8%) OLP patients saliva samples and in 6 it was $>10^5$ copies/ml. Out of those 6 patients, 5 with high EBV load in saliva had elevated IgG antibody levels in blood serum and 80% had reticular form of OLP. Histopathology showed dense lymphocyte/macrophage accumulation beneath mucosal epithelium, epithelial cell apoptosis, vacuolar damage in lower mucosal layers, and basal layer degeneration, suggesting viral infection.
Conclusions Our findings suggest a potential association between EBV infection and OLP. Elevated EBV IgG class antibody levels and presence of viral genomic sequences in saliva indicates that EBV could be involved in pathogenesis of OLP through immune dysregulation.