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Authors:
Turriziani Ombretta Università Sapienza, Roma ~ Roma ~ Italy
Falasca Francesca Università Sapienza, Roma ~ Roma ~ Italy
Donato Giuseppe Università Sapienza, Roma ~ Roma ~ Italy
Meacci Arianna Università Sapienza, Roma ~ Roma ~ Italy
Greco Nicoletta Università Sapienza, Roma ~ Roma ~ Italy
Di Loreto Fausta Università Sapienza, Roma ~ Roma ~ Italy
Borghini Raffaele Università Sapienza, Roma ~ Roma ~ Italy
Picarelli Antonio Università Sapienza, Roma ~ Roma ~ Italy

Topic: 3.3 SMALL INTESTINAL - COELIAC DISEASE: BASIC

Abstract Title ANALYSIS OF VIRAL NUCLEIC ACIDS IN DUODENAL BIOPSIES FROM ADULT PATIENTS WITH ACTIVE CELIAC DISEASE: IN SEARCH FOR AN ETIOLOGICAL RELATIONSHIP.

BACKGROUND AND AIM: Celiac Disease (CD) is a multisystemic chronic inflammatory autoimmune disease which develops in genetically predisposed subjects and it is triggered by the ingestion of gluten. After the interaction between HLA-DQ2/DQ8 and gluten-derived peptides, lymphocytes T CD4+ start a specific immune response which ends in a chronic inflammation and mucosal damage. CD pathogenesis is complex and not entirely understood, probably due to an alteration in the gastrointestinal immune system or to its aberrant regulation. Furthermore, many environmental and immune factors could be involved, particularly viral infections. The aim of the study was to observe possible relationships between CD and infections from HHV-6 A/B, EBV, CMV, adenovirus and rotavirus.

MATERIAL AND METHODS: Thirty-nine adult patients (aged 18-65 yrs) have been enrolled: specifically, 24 duodenal biopsies from active CD patients and 15 biopsies from non-CD patients were analyzed. CD diagnosis has been performed by means of serological antibodies, histology of duodenal biopsies and duodenal biopsy organ culture. Viral nucleic acids were extracted from duodenal biopsies and then amplified using Real-Time PCR technique.

RESULTS: HHV-6B was found in 62.5% of CD patients and in 73.3% of non-CD patients (p=0.13). EBV was found in 4.5% of CD patients and 6.7% of non-CD patients (p=0.35). Nucleic acids from HHV-6A, CMV, adenovirus and rotavirus were not detected in any group. HHV-6B viral load in CD patients was higher than in non-CD patients, but data were not statistically significant (p=0.54). CD patients with HHV-6B viral load >50000 copies/ml resulted to be younger and had lower anti-tTG antibody titers found at organ culture than patients with lower HHV-6B viral load (p>0.05).

CONCLUSIONS: There seems to be no difference in viral load and/or in the detection of viruses between CD and non-CD patients. Thus, our data do not support the possible relationship between CD and viral infections, although a larger population is needed to confirm our study results.

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