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Pyroptosis, Autophagy–Lysosomal Axis, and T-Cell Immune Regulation in Celiac Patients Lacking the DQ-Risk Genotype

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Abstract

Objective: Although celiac disease is predominantly associated with the HLA-DQ2.5 and/or DQ8 genotypes, it can also occur in individuals who do not carry these risk alleles. This suggests that mechanisms other than classical HLA-mediated pathogenesis may contribute to disease development. This study investigated the potential roles of selected single nucleotide polymorphisms (SNPs) associated with inflammasome–pyroptosis, autophagy-lysosomal system, T-cell activation, immune regulation, and cell migration in celiac patients without two risk DQ alleles.

Materials and Methods: A total of 89 celiac patients and 99 healthy controls were included in the study. DNA was isolated from peripheral blood samples, and HLA-DQ genotyping was performed to confirm inclusion criteria. Selected SNPs represented pathways including inflammasome/pyroptosis (IL18RAP rs917997, NLRP3 rs35829419), T-cell activation (TAGAP rs1738074), autophagy (FYCO1 rs2133660), and chemotaxis-cell migration (CCR3 rs6441961). SNP genotyping was performed using the Real-Time PCR method. Genotype and allele distributions were statistically analyzed and interpreted in the context of biological relevance.

Results: No significant difference was observed between groups in terms of age (p=0.324), whereas gender distribution differed significantly (p=0.0019). The T allele of IL18RAP (rs917997) and NLRP3 (rs35829419) was more frequent in the patient group. The TAGAP (rs1738074) TC variant and FYCO1 (rs2133660) CC variant were less frequent in patients. The CCR3 (rs6441961) TT genotype was found to be higher in patients. These findings suggest increased inflammasome activation, altered T-cell activation, impaired autophagic flux, and increased Th2/eosinophil-mediated chemotaxis in the patient group.

Conclusion: Our findings suggest that celiac disease development in patients without two risk DQ alleles cannot be explained solely by HLA-mediated mechanisms. Alterations in inflammasome activation, pyroptosis, autophagy-lysosomal pathway, and T-cell responses may represent an alternative immune axis. This study supports a distinct immune sub-phenotype in DQ-negative celiac disease and may contribute to future research on diagnostic biomarkers and targeted therapies.

Keywords: Celiac disease, inflammasome–pyroptosis, autophagy–lysosomal axis, t-cell immunoregulation

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