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**THE ROLE OF INFLAMMATORY MEDIATOR GENES IN THE
DEVELOPMENT AND CLINICAL COURSE OF ULCERATIVE COLITIS**

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Background. Ulcerative colitis (UC) is a multifactorial autoimmune pathology characterized by chronic, diffuse erosive-ulcerative mucosal inflammation of the large intestine. Disruption of epithelial barrier integrity and hyperproduction of pro-inflammatory cytokines resulting from mucosal immunoregulatory imbalance play a central role in the pathogenesis of the disease. In clinical practice, variable therapeutic responses and the progression to severe, corticosteroid-refractory forms are closely associated with allelic polymorphisms in genes encoding inflammatory mediators.

Objective. To assess the frequency of polymorphisms in key inflammatory mediator genes (TNF, IL1B, IL10) in patients with ulcerative colitis and to determine their relationship with disease severity, clinical activity, and the efficacy of targeted therapy.

Materials and Methods. Molecular genetic parameters of a cohort of patients clinically and endoscopically diagnosed with ulcerative colitis (assessed via the Mayo score) were analyzed. Data from modern investigations and international cohorts were synthesized, wherein single-nucleotide polymorphisms (SNPs) of inflammatory mediator genes were identified using polymerase chain reaction methods (PCR-RFLP and real-time PCR) from peripheral blood leukocyte DNA.

Results. Among the investigated genetic variants, the mutant A allele of the promoter polymorphism -308 G>A (rs1800629) in the TNF gene was detected at a significantly higher frequency in UC patients compared to the control group (OR = 1.6–2.1) and was associated with extensive destructive lesions (pancolitis) and hormone resistance. Furthermore, the IL1B -511 C>T and -174 G>C polymorphisms correlated with deep ulcerative defects and marked systemic inflammatory activity. Low-expression variants of the gene encoding the key anti-inflammatory mediator IL10 (-1082 A>G) were confirmed to weaken immune tolerance, whereas the IL23R (Arg381Gln) variant demonstrated a strong protective effect (OR ≈ 0.45). Pharmacogenetic analysis demonstrated that patients with the TNF -308 GG genotype showed a superior response to anti-TNF biologic therapy (infliximab), whereas carriers of the mutant allele required alternative targeted therapeutic strategies (anti-IL-12/23 agents or JAK inhibitors).

Conclusions. Imbalances in inflammatory mediator genes and their SNP polymorphisms play a pivotal role in the pathogenesis and clinical phenotypic manifestation of ulcerative colitis. Specifically, the TNF -308 A and IL1B -511 T alleles serve as reliable molecular predictors of severe disease course, complication development, and therapeutic refractoriness. Screening for these genetic biomarkers enables early risk stratification and facilitates the optimization of personalized, targeted treatment (precision medicine) strategies.

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