

Investigating associations between HLA-A, HLA-B and HLA-C allele Polymorphisms, H.pylori infection, and anti-CagA IgA among Turkish gastritis patients

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We aimed to determine the distribution of human leukocyte antigen (HLA-A, HLA-B and HLA-C) in Turkish gastritis patients with active gastritis excluding cancer cases. The relationship between the presence of H. pylori and metaplasia in the gastric mucosa and anti-CagA IgA seropositivity was investigated. Patients (n=201) were admitted for gastroendoscopy procedures and underwent high-resolution HLA-A, HLA-B and HLA-C typing. Serum anti-CagA IgA levels were analyzed by ELISA, and H. pylori and metaplasia were classified as positive or negative in gastric mucosal tissue slides. H. pylori and metaplasia were found to be positive in 86,3% and 13,9% of gastric mucosa slides, respectively. and the serum anti-CagA IgA positivity rate was 23,0%. H. pylori was found to be significantly lower in those carrying the HLA-A *03:01 allele (55,3%) than in the total allele pool (72,2%) (p=0.0145, Pearson chi-square). Carrying the HLA-A*03:01 allele reduces the incidence of H. pylori in gastric mucosa by 2,304 times (p=0.017, logistic regression). The presence of metaplasia was found to be significantly increased in carriers of the HLA-B*50:01 allele (33.3% vs 13,9%) compared to metaplasia positivity among all allele pools (p=0.048; Pearson chi-square). No significant correlation was found between anti-CagA IgA, serum positivity and HLA-A, -B and C polymorphisms. The presence of H. pylori and metaplasia in the gastric mucosa may be affected by the polymorphisms of the HLA-A*03:01 allele and HLA-B*50:01 allele. HLA-A, HLA-B, and HLA-C polymorphisms showed no effect on anti-CagA IgA production among gastritis patients with Turkish descent.

Key Words: Anti-CagA IgA, Helicobacter pylori, HLA-A*03:01; HLA-B*50:01, HLA class I