Evaluation of H.pylori Infection and IL23R Gene polymorphism in dyspeptic subjects

Farid Zandi, Hedayatollah Shirzad, Nader Bagheri, Ghorbanali Rahimian, Loghman Salimzadeh, Fateme Azadegan, Kambiz yousefzadeh Eshkevari, Fateme Fatahi, Abbas Ahmadi, Alireza Gharib, Sara Gholami and Behnam Zamanzad.

Abstract:

CagA strains of H. pylori (Hp) are known to be associated with gastroduodenal diseases. Polymorphisms in inflammation related genes, such as cytokines and their receptors, were thought to partly determine the outcome of Hp infection and the progression of gastritis. It is supposed that interleukin 23 receptor (IL23R), a basic cytokine receptor in the inflammatory IL-17/IL-23 axis, may be related to gastritis. In the present study, we evaluated the association of IL23R +2199 rs10889677 polymorphism and cagA positivity with chronic gastritis. In addition, we studied the infiltration of polymorphonuclear (PMN) and mononuclear (MN) Leukocytes into surrounding tissues of corpus. Biopsies taken from the corpus of the patients were classified as two groups: Hp-infected and Hp- uninfected. The severity of gastritis was graded from normal to severe, chronic gastritis and chronic active gastritis.

Virulence factor, cagA, was evaluated using PCR and the polymorphism in IL23R was investigated by polymerase chain reaction-restriction fragment length polymorphism (PCR-RFLP). AA and AC carriers of IL23R +2199 polymorphism, but not CC genotype in Hp-uninfected patients, were not associated with cellular infiltration and gastritis in both groups ($p > 0.05$). CagA positivity was significantly associated with increased risk of PMN ($P=0.013$), but not with MN infiltration ($P= 0.069$). Also gastritis was found to be associated with cagA positivity ($P=0.044$).Our results show decreased Hp infection probability in patients with CC genotype of 2199 +IL23R. According to the clinical and pathological features in Hp-infected group, IL23R polymorphism doesn't influence chronic gastritis and chronic active gastritis.