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Clinical Investigation

Up-regulated CCL18, CCL28 and CXCL13 Expression is Associated with the Risk of Gastritis and Peptic Ulcer Disease in *Helicobacter Pylori* infection

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Abstract

Background

Helicobacter pylori (*H. pylori*) infection causes inflammation and increases the risk of developing peptic ulcer disease (PUD); however, the exact molecular mechanisms of PUD development remain unclear. The aim of this study was to investigate the expression of CCL18, CCL28, and CXCL13 in *H. pylori*-positive subjects in comparison with *H. pylori*-negative subjects, and to determine its association with different clinical outcomes and virulence factors.

Methods

In total, 55 *H. pylori*-positive subjects with gastritis, 47 *H. pylori*-positive subjects with PUD, and 48 *H. pylori*-negative subjects were enrolled in this study. CCL18, CCL28, and CXCL13 expression were determined using real time polymerase chain reaction (PCR). The virulence factors of *H. pylori* such as *cytotoxin-associated gene A* (*cagA*), *outer inflammatory protein A* (*oipA*), *blood group antigen-binding* adhesin (*babA*), and *vacuolating* cytotoxin *A* (*VacA*) genes were evaluated using PCR.

Results

CCL18, CCL28, and CXCL13 expression in *H. pylori*-positive subjects were significantly higher than *H. pylori*-negative subjects. CCL18 and CXCL13 expression in *H. pylori*-positive subjects with $oipA^{+}$ and $babA2^{+}$were significantly higher than *H. pylori*-positive subjects with $oipA^{-}$ and $babA2^{-}$. CCL18 and CXCL13 expression were found to be significantly elevated in *H. pylori*-positive subjects with gastritis compared with *H. pylori*-positive subjects with PUD. CCL28 expression was significantly higher in *H. pylori*-positive subjects with PUD compared with *H. pylori*-positive subjects with gastritis.

Conclusions

The increased of CCL18 and CXCL13 may be involved in the pathogenesis of *H. pylori*-associated gastritis, while the increased of CCL28 may be involved in the pathogenesis of *H. pylori*-associated PUD.