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Assessment of gastric caused by *Helicobacter pylori* and pathologic elements correlation with -511 IL1-β and -308 TNF-α polymorphisms in gastritis patients

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ABSTRACT

Helicobacter pylori (*H.pylori)* is the main reason for gastric disorders including gastric lymphoma, ulcer disease, gastric carcinoma (GC), and chronic atrophic gastritis. *H.pylori has two more* significant virulence factors named *cagA* and *vacA*. Some host cytokines polymorphisms (Interleukin (IL-1) and tumor necrosis factor (TNF-α)) may contribute to *H. pylori*-related diseases. In the present study, we investigated the association of *H. pylori* gastritis and its pathogenic genes as well as the association of IL-1β and TNF-α polymorphisms in patients with gastritis. We collected gastric biopsy samples from patients with gastritis. After extracting DNA from biopsy specimens infected with *H. pylori*, *cagA* + and *vacA* + were detected by the polymerase chain reaction (PCR). To genotyping TNF-α polymorphism at position −308 and IL-1β polymorphism at position −511, PCR-based restriction fragment length polymorphism analysis was performed. Our study indicated that IL-1β-511 polymorphism, unlike TNF-α-308 polymorphism ($P = 0.030$), did not show a significant relationship between patients infected with *H. pylori* ($p = 0.219$). Also, our results indicated that alleles C and T of polymorphism of IL-1β-511 and alleles G of TNFα-308 were not significantly correlated with *cagA* status among patients infected with *H. pylori* ($p = 0.793$, $p = 0.674$, $p = 0.179$, respectively) unlike allele A of TNFα −308 ($p = 0.016$).

1. Introduction

H. pylori is a gram-negative spiral bacterium in the stomach that was firstly isolated from gastric biopsy in 1982 by Marshall and Warren (Kalsoom et al., 2020; Abolfathi et al., 2019). *H. pylori* is recognized to be the main reason for gastric lymphoma, ulcer disease, gastric carcinoma and chronic atrophic gastritis (Kalsoom et al., 2020; Shakhatreh et al., 2020; Rajindrajith et al., 2009). *cagA* and *vacA* are the most significant virulence factors of *H. pylori*, which are commonly used in genotyping of this bacterium in clinical isolation (de Brito et al., 2018). *cagA* and *vacA* genes are known as significant factors in increasing the pathogenicity of *H. pylori*. The *vacA* gene has two regions of signal (s) and middle (m) that diversity in these regions causes different levels of cytotoxicity (Akeel et al., 2019). The Vacuolating activity of this gene is mostly correlated with changes in the signaling region, while changes in the middle region of the *vacA* gene determine cell property with the efficacy of toxin on the connection of host cells (Rajindrajith et al., 2009). The vacuolating cytotoxin was firstly discovered and following that, *cagA* as the cytotoxin associated with the A gene was identified but itself lacks cytotoxic activity (Rajindrajith et al., 2009; de Brito et al., 2018; Baj et al., 2020). *H. pylori* infection can cause the manufacturing of cytokines, such as interleukin (IL-1) and tumor necrosis factor (TNF-α) (Kalsoom et al., 2020). These cytokines are efficient in increasing the inflammatory response of the gastric mucosa, which is a significant factor in causing *H. pylori* infection. Host cytokines and their polymorphisms may be among the host factors that contribute to *H. pylori*-related diseases. TNF-α is encrypted by the TNFα gene, located on the little arm of human chromosome 6 (6p21.3), among HLA-DR and HLA-B.

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