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Editorial

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Helicobacter Pylori Induce Gastric Upset

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Introduction

Helicobacter pylori is a micro-aerophilic, helical-form gram-negative aggressive bacteria. Accordingly, the idiom "Helico" intimates its helical appearance, "bacter" symbolizes bacteria, while "pylori" denotes stomach due to the first and common site of this bacteria living. Further, Marshall B. and Warren R. observed and described it in 1982. Then, the followed investigators studied this bacterium in detail with its consequences and complexities [1].

Gastric upset (Indigestion), dyspepsia: means impaired gastric digestion. Accordingly, the patient complains of upper abdominal pain, heartburn, belching, nausea, even feeling earlier gastric fullness than expected while eating. Furthermore, there are many causes of indigestion like gastroesophageal reflux disease, ulcer disease, gastritis, and even gastric cancer. Hence, unexplained recent onset dyspepsia in older people may need additional examinations. Moreover, one of the common causes is *Helicobacter pylori* infection, which needs laboratory and endoscopic examination [2].

Argument

Many theories investigated the etiology and pathogenesis of Helicobacter pylori infection, concerning chronic or acute gastritis. Hence, gastric upset is the main presentation of both types of gastritis.

Evidences

1. The genotype is valuable in determining the dominant *Helicobacter pylori* strains as the isolates were different genetically plus heterogeneous distribution. Accordingly, the vac and cag markers operate a significant function in defining clinical consequences. These virulence agents are present in a subset of *Helicobacter pylori* strains isolates like cagA, iceA, vacA, and ureC. Moreover, the cagA causes cytotoxins induction by the gastric epithelial cell as Interleukin 8 [3].
2. The molecular intercommunication researches exhibit that the act of acarus calamus in hindering biofilm formation in *Helicobacter pylori* is due to the inhibitory impact of phytobio-active component, β-sitosterol, on the quorum sensing molecules-ToxB, PhnB, DnaA, plus Sip. Consequently, this opinion may suggest the molecular mechanism of *Helicobacter pylori* in producing the acid-related complaints and gives a clue to a new therapy [4].
3. *Helicobacter pylori* infection causes lncRNA risk impression linked to *H. pylori* in gastric cancer patients and can prognosticate the prediction of these patients [5].
4. There was a close relationship between raised serum IgE levels in *Helicobacter pylori* infected patients [6].

Counterargument

The laboratory investigations of *Helicobacter pylori* infection depend on several factors like the fluctuations of serum antibody titers in a time series, the antigene detection in stool tests, the false-positive results of lab tests, or the manner of endoscopic biopsy collection. Furthermore, other factors like the variations in Cytotoxin-Associated Gene A (CagA) in East Asian patients. Moreover, the gastric nodularity or atrophy, the patient's age, the severity of the gastric mucosal infection are causes of variations in *Helicobacter pylori* detection at the time of the investigation [7].

Refutation

The significant markers of *H. pylori*, the presence of the vacuolating cytotoxin (vacA), the cytotoxin-associated gene A (cagA), which induced by the direct communication with gastric epithelium factor antigen (iceA gene), and the presence of urease C gene (ureC). Consequently, all these factors play the principal factors in deciding the gastric consequences of Helicobacter infections.

Conclusion

Helicobacter pylori induce gastric upset by several mechanisms to form numerous Gastric diseases.

Conflict of Interest:

The author do not have any conflict of interest to declare.

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