Role of *Helicobacter pylori* in Damaging the Gastroduodenal Mucosa

Marcellus Simadibrata

Division of Gastroenterology, Department of Internal Medicine, Faculty of Medicine, Universitas Indonesia/Dr. Cipto Mangunkusumo General National Hospital, Jakarta

Helicobacter pylori is a gram negative helix-shaped bacteria which is pathogen and can live in the stomach, between the mucus and the epithelial mucosa.[1] Helicobacter pylori can damage the Gastroduodenal mucosa, causing inflammation, ulceration, MALT lymphoma and cancer. It was identified in 1982 by Australian scientists Barry Marshall and Robin Warren, and in 1994, the International Agency for Research on Cancer classified *Helicobacter pylori* as a carcinogen, or cancer-causing agent, in humans.[2]

Virulence factors of *Helicobacter pylori*, include Cag A, Vac A, Bab A etc, induce proinflammatory cytokine production and release, proliferative epithelial cells signaling, and epithelial damage.[3,4] There are two possible mechanisms currently being investigated by which *Helicobacter pylori* could cause cancer. The first mechanism involves the production of free radicals near Helicobacter pylori as well as an increased rate of host cell mutation.The second mechanism involves enhancement of the transformed host cell phenotype by altering cell proteins such as adhesion proteins. So Helicobacter pylori causes inflammation and locally high levels of TNF-α and/or interleukin 6.[5,6]

The inflammatory changes on gastroduodenal mucosa caused by Helicobacter pylori infection can be seen macroscopically by endoscopy examination and microscopiccally by histopathology examination.7,8 Tissue damage due to free radicals can be examined by measuring the malondialdehyde compound.[9,10]

Laura Dairi et al in their study reported that from 20 patients with *Helicobacter pylori* (+), the average level of malondialdehyde was 1.58 umol/mL while in 20 other patients with *Helicobacter pylori* (-), the malondialdehyde level was 1.19 umol/mL with p value 0.013. They concluded that the mean serum levels of malondialdehyde was higher in Helicobacter pylori positive gastritis than *Helicobacter pylori* negative.[11]

We have some consensus and guidelines for the *Helicobacter pylori* eradication therapy using triple or quadruple drug regiments.[12] Some experts suggest that rebamipide can prevent *Helicobacter pylori* infection, reduce inflammation, accelerate healing after eradication, promote ulcer healing, and prevent progression of preneoplastic resions.[13]

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