Peptic Ulcer or Inflammatory Gastric Disease?

Until now, the hypothesis "no acid, no ulcer" has been validated in the pathogenesis of peptic ulcer disease. In this hypothesis aggressive factors like acid-pepsin overcome defensive mechanisms like bicarbonate secretion, mucus production, blood flow, cell renewal and prostaglandins. However, this hypothesis was not sufficient in explaining why ulcers were more frequent in the minor curvature of the stomach and bulbus of the duodenum. The treatment of peptic ulcer was focused on acid suppression using very potent acid suppressive agents. In addition, after successful treatment, the recurrence rate of an ulcer was almost 90 % within a year (1,2). The need for lifetime acid suppresion has been debated.

After the description of a relationship between *Helicobacter pylori* and peptic ulcer by Warren and Marshall (3), the attention of gastroenterologists was directed with great enthusiasm towards this microorganism. *Helicobacter pylori* and its relation to gastritis, peptic ulcer and cancer has been described in detail. Today, the primary goal in the treatment of gastritis, peptic ulcer and MALT lymphoma is the eradication of Helicobacter pylori. In the future, gastric cancers may be prevented by Helicobacter pylori eradication.

While *Helicobacter pylori* increases aggressive factors like acid-pepsin and gastrin, it also destroys mucosal protective factors by inhibition of blood flow, bicarbonate and mucus secretion. Somatostatin is inhibited by the infection of this microorganism. That means an important inhibitor of gastrin is eliminated and the synthesis of an important cytoprotective mediator like PGE_2 is decreased. A mucosal aggressive mediator-like nitric oxide is observed to be increased.

Is *Helicobacter pylori* an initiator of the pathogenic mechanism of peptic ulcer? The answer is "Yes" considering the new information about the bacteria and peptic ulcer. Thus, the term "no *Helicobacter pylori*, no ulcer"' should be thought to be more appropriate insted of "no acid, no ulcer". Or, one may say "If there is no *Helicobacter pylori*, there is no gastritis or ulcer". This raises another question: Is " Inflammatory Gastric Disease (IGD)" a more appropriate term in the description of the disease? This question reflects an important issue. "No acid, no ulcer" aimed to suppress acid secretion in the treatment of ulcer but not considered *Helicobacter pylori* infection as an important factor in ulcer pathogenesis. However, IGD comprises the relationship between *Helicobacter pylori*, the aetiologic agent and peptic ulcer, gastritis and cancer. It also implies that the therapy should be focused on the eradication of *Helicobacter pylori* with antibiotics.

The presentation of this term for discussion may help us find the right direction in the description of gastric diseases in the future.

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