

Functional Dyspepsia

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Letter to Editor

Functional dyspepsia is a clinical syndrome defined by chronic or recurrent pain or discomfort in the upper abdomen of a variable origin. A general agreement exists on the irrelevant role played by *Helicobacter pylori* in the pathophysiology of most cases of functional dyspepsia worldwide [1,2].

Diagnosis of *H. pylori* is based on the clinical symptoms and detection of *H. pylori* serum antibodies. The following clinical symptoms are considered; upper gastrointestinal pain, burping, gastric distension, halitosis, and hyperacidity. Specific sensitive diagnostic tests are available but *H. pylori* serum antibodies, though non-specific, is suggested because of being cost effective as the matter of *H. pylori* dyspepsia is a typical subject of cost-effectiveness [3].

It is necessary to effectively deal with *H. pylori* dyspepsia due to its associated risk with gastric carcinoma, lymphoma and many reasons of chronic illness such as acid peptic disease, diabetes, hypertension, thyroiditis, carditis, dermatitis and nephritis through immune or different reasons [2].

The efficacy of antibiotic treatment for non-ulcer dyspepsia is controversial, different trails have given conflicting results. Overall, antibiotic eradication treatment for non-ulcer dyspepsia symptoms had no significant effect on quality of life compared with placebo and was found more costly if compared to antacid treatment [4,5] Bio-organic acids; lactic and acetic, have been proved effective in symptomatic and clinical cure of dyspepsia due to interference with *H. pylori* energy metabolism or its respiratory chain metabolism as the main source of energy for *H. pylori* are via pyruvate and the activity of the pyruvate dehydrogenase complex is controlled by the rules feedback regulation and product inhibition; lactate and acetate are demonstrated among the end products of pyruvate metabolism [6-8].

Eradication of clinical symptoms of *H. pylori*-related dyspepsia is considered a clinical cure; patients who are rendered asymptomatic after treatment do not need further investigation or treatment, they can just return for re-assessment if they develop further symptoms. Evaluation of eradication after *H. pylori* treatment markedly increases cost with no clear improvement in results [9].

H. pylori is not just a bad bug in all instances; the juxta-mucosal ammonia produced by *H. pylori* protects the gastric wall from its acid if it goes in excess. The residual ammonia inside the lumen of the stomach resulting from the buffering between the ammonia and the gastric acid is not toxic, it is even beneficial; functioning as smooth muscle tonic maintaining the integrity of the gastro-esophageal sphincter and hence preventing reflux [7].

Data from observational studies have proposed a protective role of *H. pylori* against the development of gastro-oesophageal reflux disease, and suggested that *H. pylori* eradication treatment may increase the incidence of reflux symptoms. It was observed that the prevalence of *H. pylori* has been decreasing in developed countries, while the prevalence of gastro-esophageal reflux disease and esophageal adenocarcinoma has been increasing since 1930s [9].

It was amazing to the team working with the author of this letter to get the news of a nine years old Saudi girl living with her family in Switzerland to have a diagnosis of reflux disease. The symptoms of this kid just all disappeared after stopping un-necessary antibiotics for every throat infection, restriction of outside-home meals and fast food in addition to intake of natural probiotics. Normal behavior *H. pylori* is supposed to be protective; it should not be kicked out from the stomach but saved.

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