INTRODUCTION

Helicobacter pylori, a Gram negative curved or S shaped rods that colonizes the stomach has worldwide prevalence. More than 50% of the world's populations are infected with higher prevalence in developing countries (Brown, 2000). Nobel Prize in Medicine has been given to Barry Marshal and Robin Warren who, in the early 1980s, cultured H. pylori, proved its infectious nature, and described its clinica symptoms **(*Nobelprize, 2005).***

Infection with this organism is strongly associated with type B antral gastritis, peptic ulcer and gastric cancer. Several studies have been carried out on the association between H. pylori infection and various extra-digestive diseases such as cardiovascular, immunological and a variety of other pathologie **(*Gasbarrini et aL, 1999).***

Recently, several Seroepidemiological studies have suggested an association between iron deficiency anaemia (IDA) and Helicobacter pylori infection **(*Milman et al., 1998*).** Improvement in IDA after H pylori eradication has been demonstrated in case series and in clinical trials in both children and adults with otherwise unexplained IDA ***(Choe et al., 1999*).**

The possible mechanisms by which H pylori is involved in the development of IDA, in the absence of bleeding lesions, still remains to be elucidated, and given the high prevalence of the infection it is stil unclear why only some patients with H pylori develop IDA ***(Bini, 2010).***

The possibility that some H pylori strains have a specific ability to interfere with iron metabolism seems unlikely as neither virulence factors such as Cag-A ***(Berg et al., 2010***) nor mutations in the bacterial genes involved in iron uptake have been demonstrated to be associated with IDA ***(Choe et al. 2010).***

The extension of gastritis to the corporal mucosa is observed in a higher percentage of patient with H pylori infection and IDA compared with non-anaemic infected controls. This corporal involvemen results in an increase in the intragastric pH ***(Capurso et al., 2001***). H pylori infection is also involved in the development of atrophic body gastritis ***(Annibale et al., 2001***) that can in turn cause decreased gastric acid secretion and IDA ***(Dickey, 2002)***.

About 80% of dietary iron needs an acidic intragastric pH to be reduced to ferrous and be absorbed. This reaction is promoted by ascorbic acid (AA) which is therefore considered the most poten enhancer of iron absorption ***(Condrad et al., 1999).***

Ascorbic acid is actively secreted from plasma to the gastric juice and is reversibly depleted in the gastric juice of patients with H pylori gastritis (***Zhang et al., 1998***).

However, the possibility that alterations in gastric acidity and in the concentration of AA in the gastric juice might link H pylori infection with the development of IDA has not yet been fully investigated***(Dickey, 2002)***.