INTRODUCTION AND AIM OF THE WORK

Allover the world the human upper gastrointestinal tract, is commonly affected with *H. pylori*. This urea shunting bacterium is now considered to be a causal agent in a spectrum of human diseases ranging from a limited antral gastritis to frank duodenal ulceration, in addition to an association with carcinoma and lymphoma and recently in hepatic encephalopathy ***(Cordoba et al., 1997)***.

Hepatic encephalopathy, also called hepatic coma or Portal - Systemic encephalopathy ***(Sherlock and Dooley, 1997)*** is a neuropsychiatric syndrome that occurs only with significant liver dysfunction and has a potential for full reversibility. Two distinct forms can be identified in patients with cirrhosis, the overt form which is easy to diagnose, and the subclinical form ***(Rajiv et al, 1997).***

Several theories have been proposed to explain the pathogenesis of this syndrome. One of these theories is elevated blood ammonia levels resulting from impaired hepatic clearance and portosystemic shunting. Presently, colonic bacterial protein breakdown is considered to be the main source of the ammonia ***(Savarino et al., 1996).***

However, the stomach in the presence of *Helicobacter pylori* may be a notable source of systemic hyperammonaemia and may play a role in producing symptoms of hepatic encephalopathy ***(Miyaji et al., 1997).***

This study was done to determine the role of gastric *Helicobacter pylori* infection on the blood level of ammonia in patients with decompensated liver cirrhosis and its possible role in the pathogenesis of hepatic encephalopathy.