SUMMARY

Hepatic encephalopathy is a common complication affecting patients with cirrhosis although its pathogenesis and selective occurrence are not fully explained, ammonia toxicity is strongly incriminated. Encephalopathy is precipitated by compounds such as urea, which can contribute to the elevated blood ammonia levels frequently present in symptomatic patients. Bacterial urease contributes significantly to absorbed ammonia. Although this activity is usually attributed to fecal bacteria, the stomach, which possesses strong activity when infected with *H pylori* is an alternative site. Urea readily diffuses from blood to the gastric lumen where, in the presence of *H. pylon,* it is hydrolised to ammonia, which is then rapidly absorbed.

As a result *H pylori* infection could potentially contribute to the development and severity of encephalopathy.

In the present study we assessed the urease activity, Ig against *H pylori* infection together with intragastric and blood ammonia levels in 40 patients with advanced liver cirrhosis and in 20 healthy controls.

Furthermore, patients were furtherly divided according to presence or absence of H.E. symptoms.

All the studied groups were subjected to the following:

1-Complete history with special emphasis on encephalopathic symptoms.

2-Full clinical assessment.

3-Laboratory studies:

a- Complete blood picture and erythrocyte sedimentation rate.

b-Liver function tests.

c- Kidney function tests.

d-Blood sugar levels (fasting and postprandial).

e- Urine and stool examinations.

4-Abdominal ultrasonography.

5-Gastroscopy through which the followings were obtained:

a- Antral biopsies to diagnose *helicobacter* infection by:

- Rapid urease test.
- Histopathological examination.

b-Five to ten ml of gastric juice for analysis of gastric ammonia level.

6-Immediately after gastroscopy, arterial blood sampling will be withdrawn to measure:

- Serum ammonia level in all patients.
- Immunoglobulin levels (IgA, IgG) in all patients.

Results showed a significant rise of intragastric and blood ammonia levels in these patients and the rise was more obvious in those with H.E. and so presence of *H. pylori* may be a contributory factor in increasing or precipitating symptoms of H.E.