INTRODUCTION

The close association between gastric inflammatory disease and the Gram negative bacterium Helicobacter pylori (*H. pylori*) is now well established. Many studies have confirmed a close association between the presence of *H. pylori* in the gastric mucosa and chronic gastritis, peptic ulcer and even gastric cancer (Blazer, 1990).

Helicobacter pylori is a spiral shaped microaerophilic gram negative bacterium, often observed colonizing the human gastroduodenal mucosa (Soltesz *et al.*, 1992).

Fedotin (1993) reported that *H. pylori* is found in more than 95% of patients with duodenal ulcers and in about 70% of patients with gastric ulcers.

Eradication of H. pylori is indicated in all infected patients with peptic ulcer disease. In those cases, however, reliable diagnosis of the infection is a prerequisite (Thijs *et al.*, 1995).

A wide variety of diagnostic test have been developed to detect infection with this organism. These include the non invasive indirect assay tests, such as the urea breath test and serodiagnostic tests. The other alternative in diagnosis is the direct demonstration of H. pylori in gastric biopsy specimens after invasive endoscopy. This is possible through culture, assay for urease activity, immunofluorescence technique and recently by molecular biological technique based on polymerase chain reaction (PCR) (Weiss *et al.*, 1994).

For isolation of *H. pylori* either non selective media as sheep blood agar or selective media as Skirrow`s selective medium can be used. Suspected oxidase positive, catalase positive colonies are confirmed as *H. pylori* by morphology, positive rapid urease reaction (El-Ghazzawy *et al.*, 1995).

Patients with *H. pylori* gastritis develop a local and systemic humoral response with IgG and IgA antibodies (Talley *et al.*, 1991).

H. pylori secrets a chemotactic factor for human neutrophils and monocytes. A more likely role for this factor is in the production of *H. pylori* related gastritis with the recruitment of the inflammatory cells into the stomach wall (Criag *et al.*, 1992).

H. pylori elicits chlorinated toxic oxidant production from neutrophils and these substances play a critical part in *H. pylori* associated gastric mucosal damage (Suzuki *et al.*, 1994).

However gastritis associated with *H. pylori* infection may acts as predisposing factor for the action of ulcerogenic drugs, smoking and stress (Taylor *et al.*, 1991).