## INTRODUCTION

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Biliary reflux means regurgitation of duodenal contents into the stomach through the pylorus when the pyloric sphincter is resected, bypassed, or incompetent (Wormsely, 1972).

Biliary reflux is recorded in 20-30% of cholecystectomized Persons (Nudo, 1989), this was proved by Lorusso et al., (1993) and it was associated with increased histological damage to the mucosa of the antrum. In this instance the biliary and pancreatic secretions are thought to enter the duodenum continuously secondary to the lack of the gallbladder reservior function or the absence of the ampullary sphincter. Providing an excess duodenal contents available for reflux into the stomach (William, et al., 1991).

The exact mechanism for the production of alkaline reflux gastritis remains unknown. Attempts to elucidate the pathophysiology have led investigators to concentrate their efforts on the motility disorders and the cytotoxic effects of bile and pancreatic enzymes on the susceptible mucosa. Although alkaline reflux gastritis is associated with a constellation of specific symptoms, there has been a great deal of crossover between these symptoms and the symptoms of various other lesions e.g. peptic ulcer disease, atrophic gastritis, gastric polyps, gastric cancer and mechanical obstruction. Endoscopic examination with multiple mucosal biopsies has proven to be the most important step in confirming the diagnosis of alkaline reflux gastritis (Wiliam, et al., 1991).

The main symptoms of biliary reflux are constant burning pain in the midepigastrium that is worse after meals, unrelieved by antacids, nausea, bilious regurgitation and vomiting (Van Heerden et al., 1975).

Endoscopically, there is a gastric mucosal hyperaemia associated with the presence of bile in the stomach (William et al., 1991).

The aim of this work is to throw the light upon the relation between cholecystectomy and alkaline reflux gastritis as regard the incidence, the possible causes, pathophysiology, diagnosis and mangement.