



cholecystectomy and that the biliary dyskinesia is associated with GERD and gastritis [15].

Bile reflux and regurgitation are associated with chronic cholecystitis. *H. pylori* are sensitive to bile salts, because the bile salts have toxic effect on *H. pylori*. *H. pylori* were present in the stomachs of the patients with chronic lithic cholecystitis and a bile reflux. It shows that a high incidence rate of *H. pylori* infection existed in the stomach in the presence of a bile reflux, so *H. pylori* could live in the basic condition and even aggravate gastritis, this could suggest the existence of a kind of resistant *H. pylori* to bile salts. Bile reflux and regurgitation may play a role in selecting the kind of *H. pylori* so that the *H. pylori* resistant to bile salts could survive and, in combination with the bile, aggravate the injury of gastric mucosa. Resistant *H. pylori* could survive under basic circumstances and could enter the gallbladder via a reverse route [16].

Colonization of the gallbladder with *H. pylori* would be the cause of chronic inflammation similar to the association of *H. pylori* in chronic gastric inflammation. Moricz found that there is a high prevalence of Helicobacter species in patients with chronic cholecystitis and cholecystolithiasis and proposed that the bacterial infection may be associated with a pathological mechanism [17]. Chen showed the association of gastric metaplasia of gallbladder mucosa with chronic cholecystitis which this might be related to the *H. pylori* infection in the gallbladder. *H. pylori* can harm the gallbladder mucosa epithelial cells through mediating inflammation and immunoreaction [16]. Figure 1 shows the pathophysiology of *H. pylori* infection and chronic cholecystitis.

**Conclusion:** Gallstone is a major public health concern worldwide, and is one of the most prevalent digestive disorders needing

hospitalization. The etiology and pathogenesis of gallstone formation is unclear. Gallstone formation may be related to a collaboration of genetic and environmental aspects like female sex, family history, and ethnicity. Gallbladder movement disorder (biliary dyskinesia), hyperlipidemia (high cholesterol due to high-cholesterol diet), and medications (e.g., ceftriaxone, and Clonazepam), affect gallstone formation through the increasing in the activity of  $\beta$ -Hydroxyl Methyl Glutaryl-CoA (HMG-CoA) reductase enzyme or increase in the liver absorption of cholesterol from the blood [18]. Some other conditions and diseases such as obesity, pregnancy, nutrition, Crohn's disease, terminal ileum resection, stomach surgery, hereditary spherocytosis, sickle cell anemia, thalassemia, Hepatitis C Virus (HCV) infection and gallbladder polyps may affect gallstone formation [18,19]. Several liver enzymes like aspartate Aminotransferase (AST), Alanine Aminotransferase (ALT), Alkaline Phosphatase (ALP), and  $\gamma$ -Glutamyl Transferase (GGT) are associated with gallstone formation [19].

Gastro duodenal environment has an important role in the presence of gallstones and *H. pylori* are believed to be an arbitrating factor for gastric and extra gastric disease. The gallbladder and bile duct may be affected by the effect of chronic





- org/10.1155/2020/9287157) pylori and Biliary Tract Cancers: A Meta-analysis. *Can J Infect Dis Med Microbiol* 2020: 1-7.
37. Hassan EH, Gerges SS, El-Atrebi KA, El-Bassyouni HT (2015) The role of infection in gall bladder cancer: Clinicopathological study. *Tumour Biol* 36: 7093-7098.
38. Boonyanugomol W, Chomvarin C, Baik SC, Song JY, Hahnvajanawong C, et al. (2011) Role of cagA-positive