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

Bile re ux and regurgitation are associated with chronic cholecystitis. *H. pylori* are sensitive to bile salts, because the bile salts have toxic e ect on *H. pylori*. *H. pylori* were present in the stomachs of the patients with chronic lithic cholecystitis and a bile re ux. It shows that a high incidence rate of *H. pylori* infection existed in the stomach in the presence of a bile re ux, 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 re ux 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 in ammation similar to the association of *H. pylori* in chronic gastric in ammation. 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 in ammation and immunoreaction [16]. Figure 1 shows the pathophysiology of *H. pylori* infection and chronic cholecystitis.

Cholelithiasis: Gallstone is a major public health concern worldwide, and is one of the most prevalent digestive disorders needing hospitalization. e 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., ce riaxone, and Clo brate), a ect gallstone formation through the increasing in the activity of β-Hydroxyl Methyl Glut aryl-CoA (HMG-CoA) reeducates 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 a ect gallstone formation [18,19]. Several liver enzymes like aspartate Aminotransferase (AST), Alanine Aminotransferase (ALT), Alkaline Phosphatase (ALP), and γ-Glut Amyl Transferase (GTP) 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. e gallbladder and bile duct may be two of the targets of chronic *H. pylori* infection. Kawaguchi rst detected *H. pylori* in the gallbladder's mucosa of a patient with gallstones and cholecystitis who underwent cholecystectomy in 1996 for the rst time [19,20]. ere are controversial results from di erent studies, showing in favor of [21-24] or against [11,25-30] the theory of role of H. pl-6(a o)1. in gallstone formatioH. everal p-91(o)11(t64(en)19(t)5(li

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