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# ความชุกของการติดเชื้อเฮลิโคแบคเตอร์ ไพโลไร ในผู้ป่วยตับแข็งที่ได้รับการส่องกล้องทางเดินอาหาร เพื่อคัดกรองภาวะหลอดเลือดโป่งพองในหลอดอาหารของโรงพยาบาลลำปาง

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**คำสำคัญ**:
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## บทคัดย่อ

**ภูมิหลัง**: ผู้ป่วยตับแข็งมีความเสี่ยงในการติดเชื้อเฮลิโคแบคเตอร์ ไพโลไร ทำให้เพิ่มภาวะแทรกซ้อน ซึ่งยังไม่เคยมีการศึกษาความชุกและปัจจัยเสี่ยงในการติดเชื้อนี้ สำหรับผู้ป่วยตับแข็งในประเทศไทย

**วัตถุประสงค์**: เพื่อศึกษาความชุกและปัจจัยเสี่ยงของการติดเชื้อเฮลิโคแบคเตอร์ ไพโลไร ในการส่องกล้องทางเดินอาหาร เพื่อคัดกรองภาวะหลอดเลือดโป่งพองในหลอดอาหารในผู้ป่วยตับแข็ง

**วัสดุและวิธีการ**: เป็นการศึกษาแบบตัดขวาง ในผู้ป่วยตับแข็งที่ได้รับการส่องกล้องทางเดินอาหารเพื่อคัดกรองภาวะหลอดเลือดโป่งพองในหลอดอาหาร โดยตรวจการติดเชื้อเฮลิโคแบคเตอร์ไพโลไร เพื่อหาความชุกและปัจจัยเสี่ยงของการติดเชื้อนี้

**ผลการศึกษา**: มีผู้ป่วยเข้าร่วมศึกษา 108 ราย พบการติดเชื้อเฮลิโคแบคเตอร์ ไพโลไร 32 ราย (ร้อยละ 29.6) สาเหตุของตับแข็งที่พบบ่อยที่สุด คือ การดื่มแอลกอฮอล์ รองลงมา คือ ไวรัสตับอักเสบบี และไวรัสตับอักเสบซี การวิเคราะห์ univariate analysis ไม่พบปัจจัยเสี่ยงที่สัมพันธ์กับการติดเชื้อเฮลิโคแบคเตอร์ ไพโลไร ในผู้ป่วยตับแข็งอย่างมีนัยสำคัญทางสถิติ

**สรุป**: ผู้ป่วยตับแข็งที่เข้ารับการส่องกล้องทางเดินอาหารเพื่อคัดกรองภาวะหลอดเลือดโป่งพองในหลอดอาหาร พบความชุกของการติดเชื้อเฮลิโคแบคเตอร์ ไพโลไร ร้อยละ 29.6

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Original Article

# Prevalence of *Helicobacter pylori* Infection Among Cirrhotic Patients Undergoing Endoscopic Screening for Esophageal Varices at Lampang Hospital

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## Abstract

**Background**: *Helicobacter pylori (H. pylori)* infection in patients with chronic liver disease has been associated with the exacerbation of complications. However, to date, no studies have assessed the prevalence and risk factors of *H. pylori* infection specifically in cirrhotic patients in Thailand.

**Objective**: To determine the prevalence of *H. pylori* infection and identify associated risk factors among cirrhotic patients.

**Methods**: A cross-sectional study was conducted at Lampang Hospital, including cirrhotic patients undergoing esophagogastroduodenoscopy (EGD) for esophageal varices screening. *H. pylori* infection was diagnosed using either a rapid urease test or histopathological examination. Patients were classified into *H. pylori*-infected and non-infected groups.

**Results**: Of the 108 cirrhotic patients included in the study, 32 (29.6%) were diagnosed with *H. pylori* infection. Alcoholic cirrhosis was the most common etiology, followed by cirrhosis due to hepatitis B virus and hepatitis C virus. Univariate analysis revealed no significant associations between the examined risk factors and *H. pylori* infection in cirrhotic patients.

**Conclusion**: Among cirrhotic patients undergoing EGD for esophageal varices screening, the prevalence of *H. pylori* infection was 29.6%.

## Introduction

*Helicobacter pylori (H. pylori),* a gram-negative bacterium, is primarily transmitted through person-to-person contact and colonizes the gastric mucosa, promoting the development of ulcers and inflammation in both the stomach and duodenum[1]. Its pathogenicity is attributed to its ability to induce gastric inflammation and stimulate the production of proinflammatory cytokines, such as interferon-β and tumor necrosis factor, thereby increasing susceptibility to gastric cancer. The World Health Organization recognizes *H. pylori* as a significant risk factor for stomach cancer[2, 3].

The global prevalence of *H. pylori* infection is approximately 50%, with rates reaching up to 70% in developing countries, compared to about 30% in developed countries. In Thailand, the prevalence ranges from 26.3% to 61.4%[4–8], indicating a relatively high incidence. Among cirrhotic patients in developed countries, the prevalence of *H. pylori* infection has been reported to range from 10% to 49%[9–12]. Studies also indicate a significantly higher incidence of gastrointestinal ulcers in patients with cirrhosis compared to the general population, with ulcer prevalence ranging from 5% to 20% in cirrhotic patients versus 2% to 4% in the general population[9,13,14]. This increased ulcer susceptibility may be partly attributed to *H. pylori,* among other contributing factors. However, the precise mechanisms underlying this vulnerability remain poorly understood.

Complications in cirrhotic patients—such as jaundice, ascites, coagulopathy, gastrointestinal bleeding, and hepatic encephalopathy—substantially elevate morbidity and mortality rates[15]. Notably, *H. pylori* infection in individuals with chronic liver disease can worsen the clinical course, leading to heightened complications, including hepatic encephalopathy, peptic ulcers, and gastrointestinal hemorrhage[16].

Current guidelines recommend esophagogastroduodenoscopy (EGD) for screening esophageal varices in patients with compensated cirrhosis, particularly those with liver stiffness exceeding 20 kilopascals and a platelet count below 150,000 /mm$^3$[17]. However, limited data are available on the prevalence of *H. pylori* infection among cirrhotic patients undergoing EGD screening for esophageal varices screening at Lampang Hospital. The findings will help inform the development of clinical guidelines for the management and treatment of these patients. The primary objective of this study was to determine the prevalence of *H. pylori* infection among cirrhotic patients undergoing EGD screening for esophageal varices. The secondary objective was to identify factors associated with *H. pylori* infection in this patient population.

## Materials and Methods

This retrospective cross-sectional study reviewed medical records of cirrhotic patients who underwent EGD for esophageal varices screening at Lampang Hospital, Thailand, between January 2021 and December 2022. Inclusion criteria were as follows: cirrhotic patients aged over 18 years who underwent EGD for esophageal varices screening and *H. pylori* testing in the outpatient department, with liver cirrhosis confirmed by ultrasound. Exclusion criteria included patients diagnosed with liver cancer, liver metastases, fulminant hepatic failure, a history of stomach surgery, or recent gastrointestinal bleeding within the past two weeks, as these conditions could interfere with the interpretation of *H. pylori* results.

The patient data included in this study consisted of general information such as name, medical record number, age, sex, and risk factors for H. pylori infection. These risk factors included the etiology of cirrhosis, the severity of cirrhosis as assessed by the Child-Turcotte-Pugh (CTP) score (which comprises serum albumin, total bilirubin, international normalized ratio, ascites, and encephalopathy), endoscopic findings, smoking history, and history of nonsteroidal anti-inflammatory drug (NSAID) use. Patients included in the study were required to have complete data for all variables under investigation. The research protocol was approved by the Ethics Committee of Lampang Hospital (EC 092/65).

## Outcome Measurement

*H. pylori* infection was diagnosed using either a rapid urease test (RUT), also known as the Campylobacter-like organism (CLO) test, which detects the urease enzyme produced by *H. pylori* that hydrolyzes urea into ammonia and carbon dioxide, or through histopathological examination. Biopsies were performed by gastroenterologists, and the histopathology results were interpreted by pathologists.

## Statistic analysis

Sample size estimation was calculated using the formula.

$$n = \frac{NZ^2_{\alpha/2}P(1\text{-}P)}{d^2(N-1) + Z^2_{\alpha/2}P(1-P)}$$

According to relevant research, a study by Kirchner et al. (9) reported an *H. pylori* infection rate of 45% among patients with liver cirrhosis (P = 0.45). Based on survey data indicating that approximately 150 cirrhotic patients undergo EGD for esophageal varices screening annually at Lampang Hospital (N = 150), and assuming a 5% absolute precision (d = 0.05) and a $Z\alpha/2$ value of 1.96, the calculated sample size was 108 patients.

The data were analyzed using descriptive statistics. Continuous variables were summarized as means with standard deviations (SD), and between-group comparisons were conducted using Student's t-test. Categorical variables were presented as numbers and percentages, with group comparisons made using either the Chi-square test or Fisher's exact test. Patients were categorized into *H. pylori* infection and non-*H. pylori* infection groups.

To examine the association of *H. pylori* infection in patients with liver cirrhosis, logistic regression analysis was employed. In univariate analysis, results were expressed as odds ratios (ORs) with 95% confidence intervals (CIs). Statistical significance was defined as a p-value < 0.05. All statistical analyses were performed using STATA version 15.0.

## Results

There were 139 cirrhotic patients who underwent EGD for esophageal varices screening; of these 24 were not tested for *H. pylori* infection, and 7 patients were excluded due to liver cancer. A total of 108 patients were enrolled in this study, including 32 patients (29.6%) with *H. pylori* infection. *H. pylori* was detected in 31 patients (96.8%) by rapid urease test (RUT) and in 3 patients (9.4%) by histopathological examination. Among those with positive RUT results, 2 patients also underwent histopathological examination. One patient was diagnosed using histopathology alone. Baseline characteristics—such as average age, sex, etiology of cirrhosis, CTP score, endoscopic findings, smoking history, and NSAID use—did not differ significantly between the *H. pylori* infection group and the non-*H. pylori* infection group.)

In the *H. pylori* infection group, 25 patients (78.1%) were male. Alcoholic cirrhosis was the most common etiology, followed by cirrhosis due to hepatitis B virus (HBV) and hepatitis C virus (HCV). Other causes included primary biliary cholangitis (PBC) in 3 patients, autoimmune hepatitis (AIH) in 2 patients, and hemochromatosis in 1 patient. HBV-related cirrhosis was observed in 20 patients (18.5%), all of whom received antiviral therapy: 18 patients (90.0%) were treated with lamivudine, 1 patient (5.0%) with tenofovir, and 1 patient (5.0%) with entecavir. HCV-related cirrhosis was present in 18 patients (16.7%), of whom 11 (61.1%) received antiviral treatment: 4 patients (22.2%) with sofosbuvir/velpatasvir, 2 patients (11.1%) with pegylated interferon alfa-2a, and 1 patient (5.6%) with ledipasvir/sofosbuvir.

Most patients in both groups had a CTP score of A. In the *H. pylori* infection group, infection was identified across all CTP categories: 20 patients (62.5%) with CTP A, 8 patients (25.0%) with CTP B, and 4 patients (12.5%) with CTP C. The most common endoscopic finding was gastritis/duodenitis. *H. pylori* infection was associated with gastroduodenal ulcers/erosions in 5 patients (15.6%).

Further details on baseline characteristics are provided in Table 1. Univariate analysis did not identify any significant risk factors associated with *H. pylori* infection in patients with liver cirrhosis. These results are summarized in Table 2.

**Table 1.** Baseline characteristics comparing H. pylori infection and non-H. pylori infection groups (N =108).

| Factors | H. pylori infection (n=32) | Non H. pylori infection (n=76) | P-value |
|---|---|---|---|
| **Male,** n (%) | 25 (78.1) | 58 (76.3) | 0.839 |
| **Age,** years, mean ± SD | 57.0 ± 1.5 | 57.6 ± 1.0 | 0.745 |
| **Cause of cirrhosis,** n (%) | | | |
| Alcoholic cirrhosis | 20 (62.5) | 56 (73.7) | 0.247 |
| HBV infection | 9 (28.1) | 11 (14.5) | 0.101 |
| HCV infection | 4 (12.5) | 14 (18.4) | 0.454 |
| **Severity of cirrhosis,** n (%) | | | |
| CTP A | 20 (62.5) | 43 (56.6) | reference |
| CTP B | 8 (25.0) | 23 (30.3) | 0.555 |
| CTP C | 4 (12.5) | 10 (13.2) | 0.817 |
| **Endoscopic finding,** n (%) | | | |
| Gastroduodenal ulcer/erosion | 5 (15.6) | 15 (19.7) | 0.616 |
| Gastritis/duodenitis | 25 (78.1) | 61 (80.3) | 0.801 |
| Portal hypertensive gastropathy | 13 (40.6) | 38 (50.0) | 0.374 |
| **History of smoking,** n (%) | 5 (15.6) | 12 (15.8) | 0.983 |
| **History of NSAID use,** n (%) | 4 (12.5) | 7 (9.2) | 0.607 |
| **Liver function test,** mean ± SD | | | |
| Total bilirubin | 2.1 ± 2.5 | 1.9 ± 1.7 | 0.690 |
| Direct bilirubin | 0.7 ± 1.3 | 0.7 ± 0.8 | 0.795 |
| AST | 62.7 ± 50.2 | 72.3 ± 66.5 | 0.465 |
| ALT | 36.5 ± 27.1 | 42.0 ± 42.1 | 0.498 |
| ALP | 137.0 ± 66.4 | 133.1 ± 56.0 | 0.758 |
| Albumin | 3.5 ± 0.6 | 3.4 ± 0.7 | 0.503 |
| Globulin | 3.9 ± 0.9 | 4.0 ± 0.8 | 0.683 |

**Table 2.** Univariate analysis of risk factors associated with H. pylori infection in patients with cirrhosis.

| Factors | Odds Ratio | 95% CI | P-value |
|---|---|---|---|
| **Male** | 1.10 | 0.41–2.99 | 0.839 |
| **Age** | 0.99 | 0.94–1.04 | 0.745 |
| **Cause of cirrhosis** | | | |
| Alcoholic cirrhosis | 0.60 | 0.25–1.43 | 0.247 |
| HBV infection | 2.31 | 0.85–6.29 | 0.101 |
| HCV infection | 0.63 | 0.19–2.10 | 0.454 |
| **Severity of cirrhosis** | | | |
| CTP A | 1.00 | Reference | |
| CTP B | 0.74 | 0.29–1.96 | 0.555 |
| CTP C | 0.86 | 0.24–3.08 | 0.817 |
| **Endoscopic finding** | | | |
| Gastroduodenal ulcer/erosion | 0.75 | 0.25–2.28 | 0.616 |
| Gastritis/duodenitis | 0.88 | 0.32–2.41 | 0.801 |
| Portal hypertensive gastropathy | 0.68 | 0.30–1.58 | 0.374 |
| **History of smoking** | 0.99 | 0.32–3.08 | 0.983 |
| **History of NSAID use** | 1.41 | 0.38–5.19 | 0.607 |
| **Liver function test** | | | |
| Total bilirubin | 1.04 | 0.85–1.28 | 0.690 |
| Direct bilirubin | 1.05 | 0.71–1.57 | 0.795 |
| AST | 1.00 | 0.99–1.00 | 0.465 |
| ALT | 1.00 | 0.98–1.01 | 0.498 |
| ALP | 1.00 | 0.99–1.01 | 0.758 |
| Albumin | 1.23 | 0.672–2.26 | 0.503 |
| Globulin | 0.90 | 0.55–1.47 | 0.683 |

## Discussion

The prevalence of *H. pylori* infection in cirrhotic patients in this study was 29.6%, aligning with previous reports indicating prevalence rates ranging from 10% to 49%[8–11]. The prevalence of *H. pylori* infection in Thailand shows significant regional variation. A nationwide study reported an overall prevalence of 45.9% among patients undergoing EGD, with the highest rates observed in the Northeast (60.6%), followed by the North (46.9%), Central (39.0%), and South (14.4%) regions[7]. While

much of the existing data are from developed countries, the prevalence in developing countries like Thailand appears comparable. Kirchner et al.[8] reported a 45% prevalence of *H. pylori* in cirrhotic patients, higher than that found in this study. It is well established that *H. pylori* is associated with gastroduodenal ulcers and erosions. Previous studies have reported a higher prevalence of these lesions than was observed here, which may partly explain differences in *H. pylori* prevalence across studies.

*H. pylori* infection in individuals with chronic liver disease has been shown to worsen the clinical course, contributing to complications such as hepatic encephalopathy, peptic ulcers, and gastrointestinal hemorrhage[16]. Given the 29.6% prevalence observed in this study, endoscopists should prioritize the diagnosis and eradication of *H. pylori* in cirrhotic patients undergoing EGD screening. Timely detection and management may help reduce complications in this population.

In this study, no statistically significant risk factors for *H. pylori* infection were identified among cirrhotic patients undergoing EGD. However, cirrhosis due to HBV infection emerged as a potential risk factor, with an odds ratio of 2.31, although this did not reach statistical significance. The lack of significance may be attributed to the relatively small sample size, as the study's primary focus was on determining prevalence rather than associated factors. Nevertheless, the findings support the hypothesis that HBV-related cirrhosis may be linked to *H. pylori* infection, consistent with earlier studies.

This is the first study to examine *H. pylori* infection in cirrhotic patients undergoing esophageal varices screening in Thailand. Data were collected between January 2021 and December 2022 at a single center in Lampang Province. The results highlight the potential co-occurrence of *H. pylori* in this population. *H. pylori* has been associated with an increased risk of hepatic encephalopathy, partly due to its urease activity, which converts urea to ammonia—a major contributor to hyperammonemia and hepatic encephalopathy pathogenesis. Notably, eradication of *H. pylori* has been shown to reduce the risk of hepatic encephalopathy by up to 64%[18,19]. Furthermore, *H. pylori* is implicated in the development of peptic ulcer disease (PUD) in cirrhotic patients, who already face elevated risks for gastrointestinal bleeding. Therefore, managing *H. pylori* infection in this group is clinically important, with growing evidence supporting its role in reducing both hepatic encephalopathy and PUD[20]. Routine screening and appropriate eradication therapy should be considered part of comprehensive cirrhosis care.

This study has several limitations. Data on socioeconomic status, contact with infected individuals, dietary habits, and prior antibiotic use were unavailable. Future research should involve a larger sample size to better explore the association between *H. pylori* infection and cirrhosis in patients undergoing EGD for varices screening.

## Conclusion

The prevalence of *H. pylori* infection among cirrhotic patients undergoing EGD for esophageal varices screening was 29.6%.

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## References

1. Mitchell H, Katelaris P. Epidemiology, clinical impacts and current clinical management of Helicobacter pylori infection. Med J Aust. 2016;204(10):376–80.
2. Chey WD, Howden CW, Moss SF, Morgan DR, Greer KB, Grover S, Shah SC. ACG Clinical Guideline: treatment of Helicobacter pylori infection. Am J Gastroenterol. 2024;119(9):1730-53.
3. Ahn HJ, Lee DS. Helicobacter pylori in gastric carcinogenesis. World J Gastrointest Oncol. 2015;7(12):455–65.
4. Suchartlikitwong S, Lapumnuaypol K, Rerknimitr R, Werawatganon D. Epidemiology of upper gastrointestinal bleeding and infection: review of 3,488 Thai patients. Asian Biomedicine. 2015;9(1):87–93.
5. Kaosombatwattana U, Charatcharoenwitthaya P,

Pausawasdi N, Maneerattanaporn M, Limsrivilai J, Leelakusolvong S, et al. Value of age and alarm features for predicting upper gastrointestinal malignancy in patients with dyspepsia: an endoscopic database review of 4664 patients in Thailand. BMJ Open. 2021;11(10):e052252.
6. Tunruttanakul S, Wairangkool J. Prevalence of Helicobacter pylori infection in patients with perforated peptic ulcer in a tertiary hospital in Thailand: a single tertiary hospital study. Siriraj Med J. 2018;70:139–44.
7. Uchida T, Miftahussurur M, Pittayanon R, Vilaichone RK, Wisedopas N, Ratanachu-Ek T, et al. Helicobacter pylori infection in Thailand: a nationwide study of the CagA phenotype. PLoS One. 2015;10(9):e0136775.
8. Shoosanglertwijit R, Kamrat N, Werawatganon D, Chatsuwan T, Chaithongrat S, Rerknimitr R. Real-world data of Helicobacter pylori prevalence, eradication regimens, and antibiotic resistance in Thailand, 2013–2018. JGH Open. 2020;4(1):49–53.
9. Kirchner GI, Beil W, Bleck JS, Manns MP, Wagner S. Prevalence of Helicobacter pylori and occurrence of gastroduodenal lesions in patients with liver cirrhosis. Int J Clin Exp Med. 2011;4(1):26–31.
10. Wu CS, Lin CY, Liaw YF. Helicobacter pylori in cirrhotic patients with peptic ulcer disease: a prospective, case controlled study. Gastrointest Endosc. 1995;42(5):424–7.
11. Chen JJ, Changchien CS, Tai DI, Chiou SS, Lee CM, Kuo CH. Role of Helicobacter pylori in cirrhotic patients with peptic ulcer. A serological study. Dig Dis Sci. 1994;39(7):1565–8.
12. Calvet X, Navarro M, Gil M, Mas P, Rivero E, Sanfeliu I, et al. Seroprevalence and epidemiology of Helicobacter pylori infection in patients with cirrhosis. J Hepatol. 1997;26(6):1249–54.
13. Siringo S, Burroughs AK, Bolondi L, Muia A, Di Febo G, Miglioli M, et al. Peptic ulcer and its course in cirrhosis: an endoscopic and clinical prospective study. J Hepatol. 1995;22(6):633–41.
14. Chen LS, Lin HC, Hwang SJ, Lee FY, Hou MC, Lee SD. Prevalence of gastric ulcer in cirrhotic patients and its relation to portal hypertension. J Gastroenterol Hepatol. 1996;11(1):59–64.
15. D'Amico G, De Franchis R. Upper digestive bleeding in cirrhosis. Post-therapeutic outcome and prognostic indicators. Hepatology. 2003;38(3):599–612.
16. Qu H, Sun Y, Zhang J, He X, Ji S. Helicobacter pylori morbidity in chronic hepatitis B patients: a case-control study. Biomedical Research 2017; 28(13):5785–9.
17. Jakab SS, Garcia-Tsao G. Evaluation and management of esophageal and gastric varices in patients with cirrhosis. Clin Liver Dis. 2020;24(3):335–50.
18. Li J, Yu H, Wang Y, Wang B, Zhang R, Chen S, et al. A meta-analysis of the association between Helicobacter pylori infection and risk of hepatic encephalopathy. J Public Health (Oxf). 2023;45(2):321–9.
19. Agrawal A, Gupta A, Chandra M, Koowar S. Role of Helicobacter pylori infection in the pathogenesis of minimal hepatic encephalopathy and effect of its eradication. Indian J Gastroenterol. 2011;30(1):29-32.
20. Wei L, Ding HG. Helicobacter pylori infection and peptic ulcer disease in cirrhotic patients: an updated meta-analysis. World J Clin Cases. 2021;9(24): 7073–84.