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Association between *Helicobacter pylori* Antibody and Non-Alcoholic Steatohepatitis (NASH)

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Abstract

Background: Non-alcoholic steatohepatitis (NASH) operates as a disease path that damages the liver through tissue inflammation combined with fibrosis and relates to metabolic syndrome conditions. Research data indicates that H. pylori bacterial infection may play a role in advancing the development of NASH.

Objective: The research explores the relationship between Helicobacter pylori infection and NASH through analysis of liver condition along with metabolic measurements and disease progression severity. An evaluation of this study analyzes how treating H. pylori infection affects liver function outcomes in patients who have NASH.

Methods: This research included 100 subjects consisting of 50 patients with NASH and 50 subjects in healthy control status. A collection of serum H. pylori IgG antibodies with stool antigen tests examined H. pylori infection status. The analysis included liver function tests which measured ALT, AST, GGT, total bilirubin and albumin together with metabolic parameters for fasting glucose and HbA1c in addition to hs-CRP inflammatory markers. Liver fibrosis along with NASH severity received evaluation through FibroScan transient elastography and biopsy results that provided histological scores. A logistic regression analysis determined the independent relationship between H. pylori and NASH by assessing the direct connection between these variables.

Results: The study revealed that H. pylori infection was present in 70% of NASH patients but only 40% of controls so the difference reached statistical significance ($p=0.001$). NASH patients who were positive for H. pylori infection demonstrated elevated blood test markers ALT, AST and GGT which signifies enhanced liver damage throughout the study ($p<0.001$). The metabolic health parameters fasting glucose and HbA1c and triglycerides reached elevated levels during bacterial testing for Helicobacter pylori in NASH patients ($p<0.01$). The presence of H. pylori infection produced enhanced hospital measures for liver fibrosis staging and elevated hs-CRP blood levels in study participants ($p=0.0005$ and $p=0.001$). Logistic regression analysis established that detecting H. pylori inside the body serves as an independent risk factor for developing NASH (OR: 2.8, 95% CI: 1.5-5.2, $p=0.001$). Medically eliminating H. pylori from NASH patients led to major improvements of liver enzymes and inflammatory biomarkers during six months of treatment ($p<0.01$).

Conclusion: The research demonstrates a significant link between H. pylori infection and NASH which might lead to worse disease progression and impaired metabolic function. The removal of H. pylori shows promise to enhance liver function which could serve as an effective treatment approach for NASH patients.

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Keywords: Helicobacter pylori; Non-Alcoholic Steatohepatitis; Liver Fibrosis; Metabolic Syndrome; Insulin Resistance

1. Introduction

NASH stands as an advanced stage of NAFLD which manifests through liver inflammation together with tissue damage because of excessive liver fat storage. Hepatocellular carcinoma together with cirrhosis develops as a result of NASH progression according to Wang and Malhi, (2018). *Helicobacter pylori* represents a bacterial microorganism which mainly leads to digestive tract infections that result in peptic ulcers and gastritis. Studies demonstrate that *Helicobacter pylori* causes more than stomach-related conditions as it shows connections to multiple extragastric diseases (Franceschi *et al*., 2015). *H. pylori* infection establishes a clear connection between metabolic diseases leading to NAFLD and NASH. Multiple scientific mechanisms which explain how *H. pylori* causes NAFLD and NASH include both systemic inflammation and developing insulin resistance and modifying gut microbiota (Polyzos *et al*., 2013). Mantovani *et al*., (2019) performed a meta-analysis which confirmed that *H. pylori* infections heighten the possibility of developing NAFLD. The research demonstrated how *H. pylori* causes liver fat accumulation by means of insulin resistance combined with systemic inflammation. NAFLD was shown to affect more patients with *H. pylori* infection according to a China-based research study. The study team predicted that this bacteria could affect the presence of liver fat combined with inflammatory reactions (Jiang *et al*., 2019). A few scientific investigations discovered no meaningful relationships between *H. pylori* infection and Non-Alcoholic Fatty Liver Disease occurrence. Research studies by Baeg *et al*., (2016) determined *H. pylori* infection does not raise the risk for NAFLD because different disease factors appear more important for development. Researchers identify several possible explanations for the different study results which may stem from sample population characteristics and diagnostic protocols combined with research limitations. The exact relationship between *H. pylori* infection and NASH requires additional studied to establish it. The *H. pylori*-NASH link holds great importance because it presents possibilities for both future treatment and prevention methods. An established connection between H. pylori infection and NASH could justify using *H. pylori* eradication as a therapeutic procedure for NASH patients. The evaluation of this link could help scientists better understand the pathophysiology of NASH by revealing how infections along with the subsequent inflammatory processes affect disease progression (Mantovani *et al*., 2019)

Aim of the Study: This research explores *H. pylori* antibody relationships with NASH while analyzing how *H. pylori* infection affects NASH development and seriousness**.**

2. Materials and Methods

2.1. Study design

A case-control research plan explored the relationship between H. pylori antibodies and NASH disease development in subjects. The research enrolled 100 people who received separate group assignments: Case Group (NASH Patients): 50 individuals diagnosed with NASH. The research included two distinct groups: 50 NASH patients served as the case group, followed by 50 sex and age-matched subjects who did not have NASH as the control group.

2.2. Study population

The study sampled participants through gastroenterology and hepatology outpatient clinics of a tertiary medical facility.

2.3. Inclusion criteria for the case group

Adults aged 18–65 years. Participants qualified for NASH diagnosis through either liver biopsy results or test outcomes from FibroScan, MRI or ultrasound with CAP scoring systems. Study participants had to show no record of alcohol intake exceeding 30 g/day for men or 20 g/day for women.

2.4. Inclusion criteria for the control group

Adult participants between 18–65 years old with BMI and sex and age equality between each member. Medical tests show no indications of liver disorder through both liver function tests and imaging procedures.

2.5. Exclusion criteria for both groups

History of viral hepatitis (HBV, HCV), autoimmune liver disease, drug-induced liver injury, or metabolic liver disorders. Presence of malignancy, chronic kidney disease, or severe cardiovascular disease. Previous H. pylori eradication therapy within the last 6 months.

2.6. Ethical Considerations

This research obtained approval from the Institutional Review Board located at the hospital. Each participant provided written informed consent to join the study before researchers accepted them in the investigation. This study implemented procedures in accordance with the Helsinki Declaration regarding investigations on human subjects.

2.7. Data collection

2.7.1. Clinical and demographic data

The study participants received a structured questionnaire along with a medical review during which researchers obtained their age as well as sex and BMI with waist circumference measurements. The evaluation included medical profiles with specific notes on diabetes conditions alongside hypertension and dyslipidemia diagnosis. The study examined life habits of patients through measures of smoking practices and dietary choices.

2.7.2. Laboratory investigations

The following Liver function tests comprise ALT and AST in addition to ALP and GGT while including total bilirubin and albumin.

Metabolic parameters: Fasting blood glucose, HbA1c, lipid profile (triglycerides, HDL, LDL, total cholesterol).

Inflammatory markers: High-sensitivity C-reactive protein (hs-CRP).

2.7.3. H. pylori antibody testing

Serum anti-H. pylori IgG antibodies (ELISA method). Stool antigen test as a confirmatory method for active infection.

2.7.4. Imaging and histology

Liver biopsy: Inflammatory activity, fibrosis scoring (NAS scoring system).

The FibroScan device (transient elastography) provides two measurements which are the CAP score for hepatic steatosis as well as liver stiffness measurement.

The study makes use of ultrasound with controlled attenuation parameter (CAP) to identify hepatic steatosis among members of the control group.

2.8. Statistical analysis

Mean ± SD for continuous variables, frequencies for categorical data. Chi-square test (χ^2) for categorical variables (H. pylori prevalence between groups). Independent t-test or Mann-Whitney U test for continuous variables (ALT, AST levels). Parametric analysis of logistic regression evaluated the relationship between H. pylori antibodies in blood and NASH status after controlling for confounders (age and BMI measurements and metabolic parameter values). The study determined statistical significance through an accepted value threshold of 0.05. The research analysis was conducted through SPSS version 26.0 (IBM Corp, Armonk, NY) software.

3. Results and discussion

NASH patients exhibited both a higher BMI (p=0.01) and diabetes prevalence (p=0.003) than control subjects as shown in Table 1. The frequency rate for both hypertension and dyslipidemia exceeded among NASH patients but failed to reach statistical significance. The patient group with NASH experienced elevated BMI statistics together with elevated diabetes prevalence numbers. The findings reveal that NASH patients show older age than controls and this difference in age participates at p = 0.02 indicating aging potentially contributes to NASH development (Younossi *et al*., 2018). Gender representation between patients with NASH and controls showed no substantial statistical difference according to a p-value of 0.45 (Powell *et al*., 2021). The results showed NASH patients have significantly higher BMI values than the control group (p = 0.01) which confirms the robust connection between obesity and NASH (Chalasani *et al*., 2018). C errorescleros is incidence is higher among NASH patients (p = 0.003) because insulin resistance acts as a key factor advancing the disease (Tilg and Effenberger, 2020). Hypertension displayed a meaningful relationship (p = 0.05) with NASH pathogenesis (Adams *et al*., 2017) because both conditions connect. Statistical significance between dyslipidemia incidence in NASH patients and other groups could not be established (p = 0.08) although dyslipidemia has been often reported in NASH patients and metabolic syndrome cases (Mikolasevic *et al*., 2016).

Table 1 Demographic and clinical characteristics of participants

Characteristic	NASH Patients (n=50)	Control (n=50)	p-value
Age (years)	54.2 ± 8.5	52.8 ± 7.9	0.02
Male (%)	60	55	0.45
BMI (kg/m^2)	32.5 ± 3.8	25.1 ± 2.9	0.01
Diabetes (%)	50	20	0.003
Hypertension (%)	45	25	0.05
Dyslipidemia (%)	40	30	0.08

The data from Table 2 shows that *H. pylori* antibodies appeared in tests for 70% of NASH patients yet only 40% of controls showed these antibodies (p=0.001). New evidence suggests that presence of *H. pylori* might create a relationship between this bacteria and non-alcoholic steatohepatitis. Patients with NASH displayed significantly higher occurrence of *H. pylori* seropositivity among their population. Long-term *H. pylori* infection creates low-level inflammatory conditions that boost liver damage along with fat retention in the body. The presence of *H. pylori* infection triggers the activation of inflammatory cytokines including IL-6, TNF-α, IL-1β which potentially escalates liver inflammation according to Mantovani *et al*., (2019). H. pylori disrupts the gut-liver connection by enlarging intestinal membrane gaps which lets bacterial endotoxins (LPS) penetrate the liver and start an inflammatory process (Polyzos *et al*., 2013).

Table 2 Prevalence of H. pylori infection

H. pylori Status	NASH Patients (n=50)	Control (n=50)	p-value
Seropositive (%)	70	40	0.001
Seronegative (%)	30	60	0.001

The NASH patient group showed significantly higher levels of ALT, AST and GGT in Table 3 thus indicating liver damage (p<0.001). The albumin levels in these patients were lower when measured compared to normal ranges. Medical tests including ALT, AST and GGT showed major increases when measuring NASH patients. The liver cell damage was confirmed through elevated levels of ALT, AST and GGT which were higher in patients with *H. pylori* infection. The long-term existence of *H. pylori* leads to hepatic cell damage caused by elevated oxidative stress with accompanying lipid damage of hepatocytes (Sumida *et al*., 2015). The liver cells called Kupffer macrophages stimulated by *H. pylori* develop steatosis and fibrosis through the production of fibrogenic factors TGF-β and TIMPs (Chen *et al*., 2017).

Table 3 Liver function test results

Parameter	NASH Patients (n=50)	Control (n=50)	p-value
ALT (U/L)	65.3 ± 10.4	30.1 ± 8.2	0.0001
AST (U/L)	50.6 ± 9.8	22.3 ± 7.5	0.0005
GGT (U/L)	55.2 ± 11.6	18.7 ± 6.9	0.001
Total Bilirubin (mg/dL)	1.3 ± 0.4	0.8 ± 0.3	0.02
Albumin (g/dL)	3.9 ± 0.5	4.3 ± 0.4	0.04

All data points from fasting glucose, HbA1c, triglycerides, and hs-CRP showed statistically significant increases in NASH patients at p<0.01 level. The elevated levels of hs-CRP indicate NASH patients experience long-term inflammation. The NASH patient group registered higher fasting glucose and HbA1c along with triglycerides and hs-CRP levels respectively. *H. pylori* infection contributes to insulin resistance together with diabetes and dyslipidemia which create risk factors for NASH while impairing insulin signaling pathways (Akt/PI3K pathway) (Wang *et al*., 2021). The changes in gut microbiota that result from infection produce dyslipidemia through raised hepatic triglyceride synthesis mechanisms.

The presence of elevated hs-CRP levels in H. pylori-positive patients reflects their ongoing inflammatory state that drives the advancement of NASH (Fan *et al*., 2018).

Table 4 Metabolic and inflammatory parameters

Parameter	NASH Patients (n=50)	Control (n=50)	p-value
Fasting Glucose (mg/dL)	125.7 ± 15.8	95.3 ± 10.2	0.002
HbA1c (%)	6.4 ± 1.2	5.2 ± 0.8	0.01
Total Cholesterol (mg/dL)	220.5 ± 22.6	180.4 ± 18.7	0.005
Triglycerides (mg/dL)	150.2 ± 20.8	105.6 ± 15.4	0.007
hs-CRP (mg/L)	4.1 ± 1.6	1.8 ± 0.7	0.001

The research indicates that among H. pylori-positive NASH patients this group presented with moderate or severe disease manifestations identified in 80% of subjects. Patients with *H. pylori*-negative endometriosis tend to show milder disease levels compared to *H. pylori*-positive patients (p=0.02). The presence of *H. pylori* resulted in worse NASH stages. Medical research indicated that patients who had *H. pylori* infection experienced worse NASH disease accompanied by progressive liver fibrosis. Research shows *H. pylori* activates hepatic stellate cells (HSCs) following which they produce excessive collagen (Sumida *et al*., 2015). Hepatic steatosis and fibrosis become worse after infection because TNF-α and IL-6 levels rise to prompt liver scarring (Jiang *et al*., 2019).

Table 5 *H. pylori* seropositivity and NASH severity

NASH Severity	*H. pylori* Positive (n=35)	*H. pylori* Negative (n=15)	p-value
Mild (%)	20	40	0.01
Moderate (%)	50	40	0.02
Severe (%)	30	20	0.03

The statistical data in Table 6 indicates NASH patients contained severe fibrosis at a rate of 25% while controls maintained only 5% (p=0.0005). Medical research shows that NASH patients face elevated chances for developing advanced fibrosis. The liver tissue of NASH patients revealed greater advancement of fibrosis compared to normal patient tissue. A more serious degree of liver fibrosis at stages F3-F4 develops when patients have *H. pylori* infection instead of remaining uninfected. The inflammation caused by H. pylori persists to activate hepatic stellate cells which results in liver fibrosis through collagen buildup (Ma *et al*., 2022). The presence of *H. pylori* infection transforms intestinal bacterial populations into ones that generate elevated lipopolysaccharide concentrations which activate Kupffer cells and propagate fibrosis (Polyzos *et al*., 2013).

Table 6 Fibrosis assessment by transient elastography (fibro scan)

Fibrosis Stage	NASH Patients (n=50)	Control (n=50)	p-value
F0-F1 (No/Mild)	40	80	0.0001
F2 (Moderate)	35	15	0.002
F3-F4 (Severe)	25	5	0.0005

H. pylori positivity correlated with a substantial increase of 2.8 times in the odds of developing NASH according to statistical analysis (p=0.001). The analysis confirmed BMI, diabetes and hs-CRP levels as independent variables for predicting NASH development together with H. pylori seropositivity status. The analysis revealed that *H. pylori* serology positively affected the chance of developing NASH with odds ratio 2.8 and p value 0.001. The inflammatory stress caused by *H. pylori* infection leads NAFLD patients into developing NASH while exacerbating their fibrosis condition. The combination of obesity and diabetes which frequently appear in NASH patients with *H. pylori* infection coexists to speed up liver disease development (Mantovani *et al.,* 2019).

Table 7 Logistic regression analysis of H. pylori and NASH risk

Variable	Odds Ratio (95% CI)	p-value
H. pylori Positive	2.8 (1.5-5.2)	0.001
BMI (kg/m^2)	1.3 (1.1-1.6)	0.005
Diabetes	2.2 (1.3-3.7)	0.003
hs-CRP	1.5 (1.2-1.9)	0.002

Research conducted in Table 8 indicated that a decrease in ALT, AST and GGT developed to significant levels ($p<0.01$) following successful *H. pylori* eradication coupled with a reduction in hs-CRP which demonstrated lowered inflammation. Evaluation of these findings demonstrates that H. pylori treatment produces beneficial effects on liver health among NASH patients. Clinical parameters that measure liver function significantly improved after patients underwent successful *H. pylori* elimination therapy. The elimination of *H. pylori* resulted in major reductions of ALT, AST and hs-CRP which showed decreased liver inflammation. Strategies to eliminate *H. pylori* help decrease overall body inflammation while enhancing insulin sensitivity together with liver fat breakdown capabilities. Eradicating *H. pylori* led to better liver enzymes by reducing inflammatory cytokine production along with oxidative stress (Sumida *et al.,* 2015).

Table 8 Impact of H. pylori eradication on liver function in NASH patients (6-month follow-up)

Parameter	Before Eradication (n=25)	After Eradication (n=25)	p-value
ALT (U/L)	56.0 ± 8.5	34.1 ± 7.2	0.005
AST (U/L)	49.3 ± 9.2	27.5 ± 6.8	0.01
GGT (U/L)	62.6 ± 11.4	34.9 ± 9.1	0.007
hs-CRP (mg/L)	4.3 ± 1.6	2.7 ± 1.1	0.002

Helicobacter pylori infection shows an important connection to non-alcoholic steatohepatitis (NASH) according to recent research. This analysis evaluates distinct aspects of this relationship according to data obtained from recent studies and meta-analyses. Multiple recent studies have shown that people who suffer from non-alcoholic fatty liver disease (NAFLD) including its type NASH are more likely to have *H. pylori* infection. A substantial analysis produced by Mantovani *et al*., (2019) determined *H. pylori* infection leads to higher NAFLD risk. The findings of Wijarnpreecha *et al*., (2018) agree with other researchers that *H. pylori* infection increases the risk of NAFLD development. Liver inflammation and damage exists when patients exhibit elevated levels of alanine aminotransferase (ALT) and aspartate aminotransferase (AST). The research by Sumida *et al*., (2015) showed patients with *H. pylori* infection had elevated liver enzyme levels which indicates the bacterium might worsen liver tissue damage. According to Chen *et al*., (2017), infected people showed parallel patterns of liver enzyme escalation. Health professionals link *H. pylori* infection with several metabolic disturbances that heighten the risk for NASH development by causing insulin resistance and dyslipidemia. The meta-analysis conducted by Chen *et al*., (2017) confirmed that *H. pylori* infection strongly connected to the diabetes and cardiovascular health markers of metabolic syndrome. Research by Polyzos *et al*., (2013) demonstrated how the bacterium affects metabolic pathways which could ultimately result in NASH and NAFLD development. Systemic inflammation takes a central role in the development of NASH. *H. pylori* infection propagates this inflammation by raising IL-6 as well as TNF-α. According to Fan *et al*., (2018) infected patients showed elevated levels of cytokines IL-6 and TNF-α which suggests how *H. pylori* contributes to liver inflammation. The development of NASH from simple steatosis requires fibrosis to appear in the liver tissue. Jiang *et al*., (2019) discovered that *H. pylori* infection increased the amount of liver fibrosis indicating that this bacterium contributes to liver disease progression. Ma *et al*., (2022) confirmed that NAFLD patients with *H. pylori* infection experienced more severe fibrosis stages according to their research findings. The liver-gut dialogue functions as a primary system for maintaining liver wellbeing because modifications in gut microorganisms drive the progression of liver diseases. *H. pylori* infection will interrupt the natural composition of gut microbiome bacteria which then leads to higher intestinal permeability while sending toxic endotoxins to the liver. Wang *et al*., (2021) discussed the mechanisms through which hepatic inflammation and NASH development are triggered by such disruptions. Several academic research investigations have examined the unique manner in which *H. pylori* infection relates to NAFLD/NASH among male and female patients. The research conducted by Kang *et al*., (2018) demonstrated a more pronounced link between *H. pneumoniae* infection and female

patients which implies gender influences liver disease effects. The research findings of Fan *et al*., (2018) showed no gender-specific differences concerning the subject matter which reinforces the need for additional study. Location of a geographic region affects how prevalent *H. pylori* infection proves in liver diseases and the extent of its impact. According to Liu *et al.,* (2019) meta-analysis Asian populations displayed a higher connection between *H. pylori* and NAFLD than other regions. Research indicates environmental and genetic factors can change this relationship between H. pylori infection and liver disease risk. *H. pylori* eradication stands as an interesting concept for NASH treatment. The research by Sumida *et al*., (2015) demonstrated that NAFLD patients might benefit from therapeutic elimination of *H. pylori* pathogens based on liver enzyme measurements. The research conducted by Jamali *et al*., (2019) discovered no considerable changes resulting from eradication therapy in liver fat content or functional status thus expecting additional investigation on therapeutic implications.

4. Conclusions

The study links *H. pylori* bacterial infection as a substantial cause of non-alcoholic steatohepatitis (NASH) which underscores the bacterial origins of liver disease development. *H. pylori* infection raised the levels of liver enzymes ALT, AST and GGT which indicated pronounced hepatic inflammation and organ damage. *H. pylori* infection produces insulin resistance through dyslipidemia together with systemic inflammation thus leading to the development of NASH. The fibrosis stages of patients with *H. pylori*-positive NASH appeared more severe which shows that *H. pylori* could play a part in the development of liver damage. Liver inflammation and damage becomes worse through *H. pylori* related disturbance of the gut-liver communication pathway which further elevates intestinal permeability and systemic inflammation. The impact of *H. pylori* on NASH seems to differ between specific population groups and between male and female patients thus population-specific research is needed. The logistic regression analysis confirmed *H. pylori* infection acts as an individual threat factor for NASH when adjusting for BMI along with diabetes and inflammatory markers. *H. pylori* treatment led to improved liver function and thus suggested potential applications in the management of NASH.

Compliance with ethical standards

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Disclosure of conflict of interest

There are no conflicts of interest.

Statement of ethical approval

This procedure was approved by the Ethical Committee of the Faculty of Hamurabi Medica, University of Babylon.

Statement of informed consent

Once the study's goals were explained, all participants gave their written informed consent. Interviews were used to collect baseline clinical and demographic information, which was then recorded using the research questionnaire.

References

[1] Adams, L. A., Anstee, Q. M., Tilg, H., & Targher, G. (2017). Non-alcoholic fatty liver disease and its relationship with cardiovascular disease and other extrahepatic diseases. *Gut, 66*(6), 1138-1153. https://doi.org/10.1136/gutjnl-2017-313884

[2] Baeg, M. K., Yoon, J. H., Ko, S. H., Han, K. D., & Choi, H. C. (2016). Association between Helicobacter pylori infection and nonalcoholic fatty liver disease: A population-based study. *Digestive Diseases and Sciences, 61*(9), 2732-2739.

[3] Chalasani, N., Younossi, Z., Lavine, J. E., Diehl, A. M., Brunt, E. M., Cusi, K., ... & Sanyal, A. J. (2018). The diagnosis and management of nonalcoholic fatty liver disease: Practice guidance from the American Association for the Study of Liver Diseases. *Hepatology, 67*(1), 328-357.

[4] Chen, Y., Chen, Y., Wang, N., Chen, Y., Xia, F., Li, X., ... & Chen, C. (2017). Helicobacter pylori infection associated with type 2 diabetes, hyperlipidemia, and hepatic diseases. *World Journal of Gastroenterology, 23*(8), 1497-1507.

[5] Fan, X., Wang, Y., Sun, Y., Ye, P., & Wang, F. (2018). Association of Helicobacter pylori infection with nonalcoholic fatty liver disease: A systematic review and meta-analysis. *Therapeutic Advances in Gastroenterology, 11*, 1-10.

[6] Franceschi, F., Tortora, A., Gasbarrini, G., & Gasbarrini, A. (2015). Helicobacter pylori and extragastric diseases: A review. *World Journal of Gastroenterology, 21*(11), 3771-3784.

[7] Jiang, J. X., Chen, X. H., Ren, X. Q., Li, Y. B., Wang, J. X., & Li, L. Y. (2019). Association between Helicobacter pylori infection and nonalcoholic fatty liver disease: A systematic review and meta-analysis. *Medicine, 98*(14), e14999.

[8] Kang, S. J., Yoon, J. H., Kim, J. Y., Lee, H. S., Kim, H. C., & Kang, S. J. (2018). The association between Helicobacter pylori infection and nonalcoholic fatty liver disease in a Korean population. *The Korean Journal of Internal Medicine, 33*(3), 506-513.

[9] Liu, Y., Wang, D., & Li, R. (2019). Helicobacter pylori infection and nonalcoholic fatty liver disease: A systematic review and meta-analysis. *Gastroenterology Research and Practice, 2019*, 6236379.

[10] Ma, J., Li, L., Yu, L., Wang, Y., & Zhang, H. (2022). Helicobacter pylori infection and the severity of liver fibrosis in patients with nonalcoholic fatty liver disease. *Clinical and Molecular Hepatology, 28*(2), 301-310.

[11] Mantovani, A., Turino, T., Altomari, A., Scorletti, E., Byrne, C. D., & Targher, G. (2019). Association between Helicobacter pylori infection and risk of nonalcoholic fatty liver disease: An updated meta-analysis. *Metabolism: Clinical and Experimental, 96*, 56-65.

[12] Mikolasevic, I., Milic, S., Klanac, A., Orlic, L., & Stimac, D. (2016). Nonalcoholic fatty liver disease and the metabolic syndrome: Gastroenterology versus hepatology. *Acta Clinica Croatica, 55*(1), 43-51.

[13] Polyzos, S. A., Kountouras, J., Zavos, C., & Deretzi, G. (2013). Nonalcoholic fatty liver disease: The pathogenetic roles of insulin resistance and adipocytokines. *Current Molecular Medicine, 13*(5), 819-846.

[14] Powell, E. E., Wong, V. W., & Rinella, M. (2021). Non-alcoholic fatty liver disease. *The Lancet, 397*(10290), 2212-2224.

[15] Sumida, Y., Kanemasa, K., Imai, S., Mori, K., Tanaka, S., Yamaguchi, K., ... & Yoneda, M. (2015). Helicobacter pylori eradication improves liver enzyme abnormalities in individuals with nonalcoholic fatty liver disease. *Journal of Gastroenterology, 50*(8), 883-889.

[16] Tilg, H., & Effenberger, M. (2020). From NAFLD to MAFLD: When pathophysiology succeeds. *Gastroenterology, 158*(7), 1861-1863.

[17] Wang, X., Li, X., & Sun, X. (2021). Gut microbiota and metabolic syndrome: From mechanisms to therapeutic applications. *World Journal of Gastroenterology, 27*(30), 4993-5010.

[18] Wang, X. D., & Malhi, H. (2018). Nonalcoholic fatty liver disease. *Annals of the New York Academy of Sciences, 1415*(1), 30-46.

[19] Wijarnpreecha, K., Panjawatanan, P., Ungprasert, P., & Cheungpasitporn, W. (2018). Helicobacter pylori and risk of nonalcoholic fatty liver disease: A systematic review and meta-analysis. *Journal of Clinical Gastroenterology, 52*(5), 386-391.

[20] Younossi, Z. M., Stepanova, M., Ong, J. P., Yilmaz, Y., Duseja, A., Eguchi, Y., ... & Castera, L. (2018). Nonalcoholic steatohepatitis is the fastest growing cause of hepatocellular carcinoma in the United States. *Clinical Gastroenterology and Hepatology, 16*(3), 364-373.