

ORIGINAL ARTICLE

Association of Helicobacter Pylori Infection with Nonalcoholic Fatty Liver Disease among Adults: A Hospital-Based Cross-Sectional Study

MUHAMMAD AMJAD FAROOQ¹, MUHAMMAD OSAMA RIAZ², SYED MUHAMMAD AZEEM³, MUHAMMAD FAHAD SHAHID⁴, USAMA JAVED⁵, ISTEHSAN AHMED⁶

¹PGR Medicine, Arif Memorial Teaching Hospital/Rashid Latif Khan University, Lahore.

²Medical Officer Medicine, SIDER, Shalamar Hospital, Lahore

³Medical Officer Gastroenterology, Bahria International Hospital, Lahore. **Email:** Syed.m.azeem1040@gmail.com

⁴Medical Officer, Punjab Kidney Liver Institute, Lahore. **Email:** fahadshahid629@gmail.com

⁵Medical Officer, Shalimar Hospital, Lahore. **Email:** usamajaved2929@gmail.com

⁶House Officer, Department of Paediatrics, Services Hospital, Lahore. **Email:** Istehsanahmed3@gmail.com

Correspondence to: Dr Muhammad Amjad Farooq, **Email:** amjadsmc@gmail.com

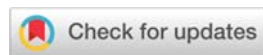
This article may be cited as:

Farooq MA, Riaz MO, Azeem SM, Shahid MF, Javed U, Ahmed I; Association of Helicobacter Pylori Infection with Nonalcoholic Fatty Liver Disease among Adults: A Hospital-Based Cross-Sectional Study. Pak J Med Health Sci, 2026; 20(06): 4-9.

Received: 02-03-2026

Accepted: 15-06-2026

Published: 30-06-2026



© The Author(s) 2026. This is an open-access article distributed under the terms of the [Creative Commons Attribution 4.0 International License \(CC BY 4.0\)](https://creativecommons.org/licenses/by/4.0/), which permits unrestricted use, distribution, and reproduction in any medium, provided the original author(s) and source are credited.

**ABSTRACT**

Background: There has been some recent evidence to indicate that Helicobacter pylori (H. pylori) infection has been shown to play a role in NAFLD pathogenesis as it causes inflammatory and metabolic effects, although this association is debated. To recount the relation between the H. pylori infection and nonalcoholic fatty liver disease on adult patients in a teaching hospital, in the tertiary sector.

Methods: This cross-sectional research was done at a hospital department-Gastroenterology and Internal Medicine department- in Arif Memorial Teaching Hospital, Lahore Pakistan. Consecutive sampling was used to enroll 165 adult patients. Diagnosis of active H. pylori infection was done with the help of a stool antigen test, and NAFLD was identified with the help of abdominal ultrasonography (exclusions of significant amounts of alcohol consumption and other reasons of chronic liver disease). A structured proforma was used to collect demographic, anthropometric, clinical, and biochemical information data.

Results: The mean age of the participants was 44.5 ± 11.6 years, and 89 (53.9%) were female. Patients with active H. pylori infection were 92 (55.8) and the ultrasonographic presence of NAFLD was found in 74 (44.8) patients. H. pylori infection was more prevalent in patients with NAFLD compared to those without NAFLD (68.9% vs 45.1; $p=0.002$). Also significantly correlated ($p<0.05$) with NAFLD were old age, overweight or obesity, bigger waist circumference, diabetes mellitus, high blood pressure, increased triglycerides levels, and higher levels of alanine aminotransferase. Moderate-to-severe ($p=0.011$) hepatic steatosis was found much more frequently in patients with H. pylori infection. In multivariate logistic regression analysis, H. pylori infection was found to be an independent predictor of NAFLD (adjusted OR=2.14, 95% CI: 1.084.21; $p=0.028$) which is accompanied with body mass index, diabetes mellitus, hypertriglyceridemia, and advancing age.

Conclusion: Active H. pylori infection was found to be correlated with NAFLD presence with established metabolic risk factors being adjusted. Moreover, hepatic steatosis was worse in patients who were H. pylori-positive. These results indicate that inflammatory and metabolic mechanisms might be involved in NAFLD pathogenesis by virtue of infection with H. pylori. To determine causality and whether H. pylori eradication can decrease the risk or progression of NAFLD, large prospective cohort studies and randomized clinical trials are required.

Keywords: Helicobacter pylori; nonalcoholic fatty liver disease; NAFLD; hepatic steatosis; cross-sectional study; metabolic syndrome.

INTRODUCTION

Nonalcoholic fatty liver disease (NAFLD) is now considered to be one of the major global health problems and has already become the most common cause of chronic liver disease. It has grown exponentially in the last several decades mostly owing to increase in obesity, lack of physical exercise and poor eating habits around the world. Recent epidemiological data show that 1 in 4 adults around the world has NAFLD, and this is the cause of NAFLD being a significant cause of liver-related morbidity, mortality and health care expenditure. In addition to liver complications, NAFLD is strongly linked to the heightened risk of cardiovascular disease, chronic renal disease, and general death, suggesting its significance as a multisystemic metabolic disorder and not a hepatic disease alone¹⁻². Metabolic abnormalities, such as obesity, insulin resistance, type 2 diabetes mellitus, dyslipidaemia, hypertension, and the other elements of the metabolic syndrome are closely associated with the development and progression of NAFLD. These metabolic imbalances encourage excessive body lipid deposition, chronic low-grade inflammation, oxidative stress and progressive liver damage that could further progress into nonalcoholic steatohepatitis (NASH), advanced fibrosis, cirrhosis and hepatocellular carcinoma. This has prompted international liver societies in recent years to coin the acronym metabolic dysfunction-associated steatotic liver disease (MASLD) as a more appropriate term to reflect the predominant role of metabolic dysfunction in the pathogenesis of fatty liver disease. However, the name nonalcoholic fatty liver disease (NAFLD) has been and still remains popular among the current scientific community and in the minds of clinicians and researchers across the globe. To maintain similarity with the prior published pieces of evidence, the nomenclature used in the current study, and the name of the study, the nomenclature NAFLD has been used throughout the present study¹⁻³.

One of the most widespread chronic bacteria in the world is *H. pylori*. In addition to the known association to peptic ulcer disease, chronic gastritis and gastric malignancy, emerging data indicate that *H. pylori* can also affect systemic inflammation, insulin resistance, lipid metabolism, and endothelial dysfunction. These extra-gastric metabolic impacts have led to increased interest over whether *H. pylori* is an episodic or persistent in development or progression of NAFLD⁴⁻⁶.

Uncontrolled studies assessing the relationship between *H. pylori* and NAFLD have had inconsistent results. A small number of cross-sectional studies in China, Egypt and Iran found a positive relationship between infection with *H. pylori* and NAFLD, but in much larger

studies, Japan and some other cohorts failed to remain significantly independent. The meta-analyses since 2018 all tend to indicate a small yet significant positive correlation, but the area, study design, and diagnostic methods of heterogeneity remain high⁶⁻¹².

The possible biological pathways by which *H. pylori* contribute towards NAFLD comprise chronic low-grade inflammation, secretion of tumour necrosis factor-alpha and interleukin-6, impairment of insulin signaling, altered adipokine profiles, alterations in gut permeability, and effects on hepatic lipid accumulation. Due to the inconsistency of the literature and the value of the region-specific evidence, the aim of the study was to determine the connection between *H. pylori* infection and NAFLD in a cross-sectional group of adult patients attending a tertiary care centre¹³⁻¹⁵.

MATERIAL & METHODS

Study Design and Setting

The study is a hospital-based cross-sectional study undertaken in the Department of Gastroenterology and Internal Medicine in Arif Memorial Teaching Hospital, Lahore, Pakistan in 12 months between January 2024 and December 2024. The research was to examine if there was a linkage between nonalcoholic fatty liver disease (NAFLD) and *Helicobacter pylori* infection in adult patients visiting the outpatient and inpatient units of the hospital. Arif Memorial Teaching Hospital is a teaching hospital of tertiary care and specializes in both medical and gastroenterology services to a wide variety of patients both of Lahore and well beyond.

Population and Size of the sample of the study

One hundred and sixty-five adult patients that were aged 18 and above were recruited through a consecutive non-probability sampling method. The inclusion criteria in the study were patients who had both an *H. pylori* stool antigen testing and an abdominal ultrasonography. The sample size was calculated as sufficient to obtain sufficient statistical power in assessing the relationship between the NAFLD and *H. pylori* infection.

Inclusion Criteria

Inclusion criteria were: Patients The patients were to meet the following criteria:

- Age ≥ 18 years.
- Has had stool antigen testing regarding active *H. pylori* infection.
- Received an assessment of hepatic steatosis by means of abdominal ultrasonography during a clinical evaluation.

- Perfect demographic, clinical and laboratory data.
- Gave a written informed consent to take part in the study.

Exclusion Criteria

Patients were to be excluded in case they had any of the following:

- Marked intake of alcohol
- Chronic hepatitis B or C virus infection.
- Liver injury.
- Wilson's disease or hereditary haemochromatosis.
- Hepatic liver cirrhosis or hepatocellular carcinoma.
- Pregnancy.
- Past H. pylori therapy within the six months before.
- Unfinished clinical or laboratory charts.

Data Collection

Structured pretested data collection proforma was used in the collection of data. The study used demographic and anthropometric variables such as age and sex and body mass index (BMI) and waist circumference. Medical records and clinical interviews provided clinical data on the and diabetes mellitus, hypertension, dyslipidaemia, and medication history of the patients, and other pertinent comorbid conditions. Laboratory tests were to be done. All lab tests were conducted at the central lab of the hospital, through standard lab tests and quality control measures.

Diagnosis of Helicobacter pylori Infection

The diagnosis of active H. pylori infection was initiated on a commercially available stool antigen test, which identifies antigens of H. pylori in the stool sample. Trained laboratory staff performed the test in measuring up to the instructions specified by the manufacturer. The positive stool antigen test was deemed to be evidence of active H. pylori infection.

Nonalcoholic Fatty Liver Disease Diagnosis

Abdominal ultrasonography done by the experienced consultant radiologists who were not told the H. pylori status of patients diagnosed NAFLD. There was an assessment of hepatic steatosis using the set of ultrasonographic criteria, which comprised of greater hepatic echogenicity, vascular blurring, and posterior beam attenuation. Fatty liver had been graded as mild, moderate or severe based on the standard radiological grading criteria after eliminating an influential alcohol consumption and other known causes of chronic liver disease.

Outcome Measures

The main outcome was the NAFLD that was confirmed with ultrasonography. The severity of hepatic steatosis

and the identification of independent predictors of NAFLD (e.g., H. pylori infection, age, BMI, diabetes mellitus, serum triglycerides, and liver enzymes levels) were additional secondary outcomes.

Ethical Considerations

The study protocol was submitted to Institutional Review Board/ Ethical Review Committee of Arif Memorial Teaching Hospital, Lahore, and approved prior to the commencement of the study. All the participants were enrolled with written informed consent. The research study followed the ethical standards of the declaration of Helsinki. There was strict confidentiality and anonymity to the participants in the study and no data collected was used in any other way other than research.

RESULTS

There were 165 adult patients in the study. The mean age of the study population was 44.5 ± 11.6 years, with 89 (53.9%) females and 76 (46.1%) males. Helicobacter pylori infection was identified as active in 92 (55.8) patients and 74 (44.8) patients was found to have ultrasonographic findings of nonalcoholic fatty liver disease (NAFLD). There were patients who had NAFLD and were categorized based on it. Patients with NAFLD were much older and had greater levels of body mass index (BMI), waist circumference, serum triglyceride levels and alanine aminotransferase (ALT) compared to patients without NAFLD. Diabetes mellitus, hypertension, and H. pylori infection were also significantly more common among patients with NAFLD (Table 1).

The patients with NAFLD were considerably older in comparison to the non-NAFLD patients (47.8 ± 10.6 vs. 41.9 ± 11.4 years, $p=0.001$) The level of BMI and waist circumference were also significantly greater in NAFLD ($p<0.001$), and it is possible to suggest that obesity and central adiposity are significant factors that develop the issue. Patients with NAFLD had significantly higher metabolism comorbidities, such as diabetes mellitus (33.8% vs. 15.4, $p=0.007$) and hypertension (33.8% vs. 18.7, $p=0.028$). Moreover, triglyceride levels and ALT levels were significantly higher in NAFLD (both, $p<0.001$), indicating metabolic with malfunction and hepatocellular damage. Notably, NAFLD in patients was found to be actively infected by H. pylori (68.9 versus 45.1) ($p=0.002$). This statistically significant difference indicates that there is a positive relationship between NAFLD and H. pylori infection.

There was a high prevalence of NAFLD between H. pylori-positive and H. pylori-negative patients (55.4% and 31.5% respectively). Furthermore, moderate and severe hepatic steatosis grades were found more commonly in

patients who had an active infection of *H. pylori*. This statistically significant chromichi-square ($p=0.011$) value shows that there exists a significant association between increasing severity of NAFLD and *H. pylori* infection.

The odds ratio of 2.70 when used as crude shows that the likelihood of patients developing NAFLD was 2.7

times when they were infected by *H. pylori* than when they were not. This is in line with the hypothesis that *H. pylori* infection can promote the presence, in addition to the development, of hepatic steatosis.

Table 1. Baseline Characteristics According to NAFLD Status

Variable	NAFLD (n=74)	No NAFLD (n=91)	p-value
Age (years)	47.8 ± 10.6	41.9 ± 11.4	0.001
Male sex, n (%)	36 (48.6)	40 (44.0)	0.560
BMI (kg/m ²)	30.1 ± 3.9	26.4 ± 3.7	<0.001
Waist circumference (cm)	101.6 ± 9.8	92.3 ± 8.6	<0.001
Diabetes mellitus, n (%)	25 (33.8)	14 (15.4)	0.007
Hypertension, n (%)	25 (33.8)	17 (18.7)	0.028
Triglycerides (mg/dL)	196.2 ± 58.4	153.1 ± 49.7	<0.001
ALT (U/L)	43.7 ± 18.5	31.2 ± 14.1	<0.001
<i>H. pylori</i> positive, n (%)	51 (68.9)	41 (45.1)	0.002

Table 2. Distribution of NAFLD Severity According to *H. pylori* Status

<i>H. pylori</i> Status	No NAFLD	Mild	Moderate	Severe	Any NAFLD n (%)
Positive (n=92)	41	28	16	7	51 (55.4)
Negative (n=73)	50	18	4	1	23 (31.5)

Table 3. Multivariable Logistic Regression for Predictors of NAFLD

Variable	Adjusted OR	95% CI	p-value
Age (per year)	1.03	1.00–1.06	0.041
Male sex	1.12	0.58–2.17	0.730
BMI (per kg/m ²)	1.26	1.13–1.41	<0.001
Diabetes mellitus	2.08	1.01–4.28	0.047
Triglycerides ≥150 mg/dL	1.94	1.01–3.76	0.046
ALT >40 U/L	1.71	0.88–3.33	0.111
<i>H. pylori</i> Positive	2.14	1.08–4.21	0.028

DISCUSSION

This cross-sectional research discovered that *H. pylori* infection was significantly related to NAFLD among 165 adult patients. There were significantly higher prevalence rates of NAFLD in *H. pylori*-positive participants in comparison to *H. pylori*-negative participants and it was still found significant even after controlling age, BMI, diabetes, triglycerides, and ALT. We also noted a severity gradient with moderate and severe steatosis being more frequent in the case of the infected persons. These results confirm the hypothesis that *H. pylori* can role in hepatic steatosis in addition to the impact of conventional metabolic risk factors alone^{6-8,10-12}.

Our findings coincide with a number of past finding studies. According to research by Jiang et al., NAFLD was significantly associated with *H. pylori* infection in a very large North Chinese cohort, Abo-Amer et al and Mohammadifard et al also reported positive associations between NAFLD and infection of *H. pylori* infection in

Egyptian and Iranian population respectively. It was later proposed by Wang et al. that this correlation can be stronger among women, which suggests that sex-specific metabolic or hormonal interactions may be contributing to disparities in studies to some extent^{6-8,14}.

We also found our results consistent with the trend of most recent meta-analyses. Heydari et al. established that NAFLD was more common in people who were infected with *H. pylori* and the 2024 meta-analysis study indicated a bi-directional association between the two diseases. Other previous pooled studies by Zhou and Mantovani identified a small yet significant excess rate of NAFLD in those who are infected^{10-12,20}.

This association is probably caused by a number of pathophysiological pathways. Systemic inflammation, insulin resistance, dyslipidaemia, disrupted adipokine signalling, malfunctioning gut barrier, and endotoxaemia have been associated with chronic *H. pylori* infection. Polyzos et al. reported the association between *H. pylori* and insulin resistance, and subsequent reviews have

explained how these metabolic derangements might accelerate fat accumulation in the liver and the development of simple steatosis to steatohepatitis¹³. These mechanisms are consistent with the higher levels of triglycerides, higher BMI, and burden of diabetes evident in our NAFLD group, and raise an additional question that *H. pylori* could exacerbate an already negative metabolic picture.

The gradient of severity that was observed in our study is particularly attractive. Moderate and severe NAFLD were more common in patients who were infected compared to patients who did not have the infection. This trend is consistent with reports that *H. pylori* might not simply correlate with the presence of NAFLD, but might in fact increase the more advanced steatosis or fibrosis markers in certain settings. *H. pylori* Since reported in Southwest China, it was found that *H. pylori* were associated with increased liver stiffness more than with steatosis alone¹⁵.

Meanwhile, we must view our findings with a grain of salt since the literature is not homogeneous. The study did not demonstrate an association in a large cross-sectional Japanese study, and a Guatemalan study by Alvarez et al. emphasized once again communication findings of the significance of differences in the population, the method of diagnosis, and the metabolic context^{9,16}. This heterogeneity implies that the impact of *H. pylori* on NAFLD is potentially real and small, and may depend on ethnicity, diagnostic test, background cardiometabolic burden, and whether it is active infection or previous exposure that is assessed.

The other question that is of significance is whether the eradication of *H. pylori* enhances the outcome of NAFLD. Data on interventions are scarce and rather sporadic. No clear independent effect of eradication on liver fat content was demonstrated in early studies like that of Jamali et al., but later pilot and randomized studies (e.g. Maharshi et al., Yu et al.) indicated that eradication might enhance steatosis or inflammatory markers in selected patients¹⁷⁻¹⁹. In general, these trials are too small and too heterogeneous to warrant universal eradication on treatment of NAFLD but do favor subsequent prospective studies.

This research has a number of strengths. It employed active *H. pylori* testing, systematically ruled out important major secondary causes of fatty liver, and multivariate adjustment of critical metabolic confounders. It too evaluated NAFLD severity as opposed to dichotomous presence as opposed to absence only. Nevertheless, certain limitations have to be taken into account. To begin with, the cross-sectional design does not allow any causal inference. Second, although convenient and more common, ultrasonography lacks the sensitivity of

magnetic resonance or liver biopsy in quantifying steatosis. Third, there is a possibility that residual confounding with diet, physical activity, socioeconomic factors, and the use of medicines is there. Lastly, this was one-centre study and so this may limit generalisability^{2,3}.

CONCLUSION

H. pylori infection had a significant positive association with NAFLD in this cross-sectional study of 165 adults, and was more prevalent in patients with moderate-severe hepatic steatosis. After controlling the age, obesity, diabetes, triglycerides, and liver enzymes, it was found that the relationship was not lost indicating that *H. pylori* might have a direct effect on fatty liver disease. Despite the lack of causation made possible by the cross-sectional nature, the results indicate that *H. pylori* can cause the disease through inflammatory and metabolic mechanisms that overlap with the NAFLD mechanisms. The bigger prospective studies and properly designed interventions trials should be done to find out whether *H. pylori* eradication can help in decreasing the incidence, the severity, or the progression of NAFLD.

DECLARATION

Conflict of Interest: The authors declare no conflict of interest.

Funding: This research did not receive any external funding.

Author's Contribution: All authors contributed equally in the complication of current study.

Acknowledgments: The authors express their sincere gratitude to all colleagues and participants for their valuable contributions to this study.

Data Availability Statement: The data that supports the findings of this research are available on request from Corresponding Author.

REFERENCES

1. Younossi ZM, Golabi P, Paik JM, Henry A, Van Dongen C, Henry L. The global epidemiology of nonalcoholic fatty liver disease (NAFLD) and nonalcoholic steatohepatitis (NASH): a systematic review. *Hepatology*. 2023;77:1335-1347.
2. Rinella ME, Neuschwander-Tetri BA, Siddiqui MS, Abdelmalek MF, Caldwell S, Barb D, et al. AASLD Practice Guidance on the clinical assessment and management of nonalcoholic fatty liver disease. *Hepatology*. 2023;77(5):1797-1835.
3. European Association for the Study of the Liver, European Association for the Study of Diabetes, European Association for the Study of Obesity. EASL-EASD-EASO Clinical Practice Guidelines on the management of metabolic dysfunction-associated steatotic liver disease (MASLD). *J Hepatol*. 2024;81:492-542.
4. Li Y, Choi H, Leung K, Jiang F, Graham DY, Leung WK. Global prevalence of *Helicobacter pylori* infection between 1980 and

- 2022: a systematic review and meta-analysis. *Lancet Gastroenterol Hepatol.* 2023;8:553-564.
5. Crowe SE. *Helicobacter pylori* infection. *N Engl J Med.* 2019;380(12):1158-1165.
 6. Jiang T, Chen X, Xia C, Liu H, Yan H, Wang G, et al. Association between *Helicobacter pylori* infection and non-alcoholic fatty liver disease in North Chinese: a cross-sectional study. *Sci Rep.* 2019;9:4874.
 7. Abo-Amer YE, Sabal A, Ahmed R, Hasan NFE, Refaie R, Mostafa SM, et al. Relationship between *Helicobacter pylori* infection and nonalcoholic fatty liver disease (NAFLD) in a developing country: a cross-sectional study. *Diabetes Metab Syndr Obes.* 2020;13:619-625.
 8. Mohammadifard M, Saremi Z, Rastgoo M, Akbari E. Relevance between *Helicobacter pylori* infection and non-alcoholic fatty liver disease in Birjand, Iran. *J Med Life.* 2019;12(2):168-172.
 9. Okushin K, Takahashi Y, Yamamichi N, Shimamoto T, Enooku K, Fujinaga H, et al. *Helicobacter pylori* infection is not associated with fatty liver disease including non-alcoholic fatty liver disease: a large-scale cross-sectional study in Japan. *BMC Gastroenterol.* 2015;15:25.
 10. Heydari K, Yousefi M, Alizadeh-Navaei R, Lotfi P, Sheydaee F, Raei M, et al. *Helicobacter pylori* infection and non-alcoholic fatty liver disease: a systematic review and meta-analysis. *Turk J Gastroenterol.* 2022;33(3):171-181.
 11. Zhang D, Wang K, Bai J. Bidirectional relationship between *Helicobacter pylori* infection and nonalcoholic fatty liver disease: insights from a comprehensive meta-analysis. *Front Nutr.* 2024;11:1410543.
 12. Zhou BG, Yang HJ, Xu W, Wang K, Guo P, Ai YW. Association between *Helicobacter pylori* infection and nonalcoholic fatty liver disease: a systematic review and meta-analysis of observational studies. *Helicobacter.* 2019;24:e12576.
 13. Polyzos SA, Kountouras J, Zavos C, Deretzi G. The association between *Helicobacter pylori* infection and insulin resistance: a systematic review. *Helicobacter.* 2011;16(2):79-88.
 14. Wang J, Dong F, Su H, Zhu L, Shao S, Wu J, et al. *H. pylori* is related to NAFLD but only in female: a cross-sectional study. *Int J Med Sci.* 2021;18(11):2303-2311.
 15. Liu Y, Li D, Liu Y, Shuai P. Association between *Helicobacter pylori* infection and non-alcoholic fatty liver disease, hepatic adipose deposition and stiffness in Southwest China. *Front Med (Lausanne).* 2021;8:764472.
 16. Alvarez CS, Florio AA, Butt J, Rivera-Andrade A, Kroker-Lobos MF, Waterboer T, et al. Associations between *Helicobacter pylori* with nonalcoholic fatty liver disease and other metabolic conditions in Guatemala. *Helicobacter.* 2020;25(6):e12756.
 17. Jamali R, Mofid A, Vahedi H, Farzaneh R, Dowlathshahi S. The effect of *Helicobacter pylori* eradication on liver fat content in subjects with non-alcoholic fatty liver disease: a randomized open-label clinical trial. *Hepat Mon.* 2013;13:e14679.
 18. Maharshi V, Gupta P, Kumar VL, Upadhyay AD, Das P, Yadav R, et al. Effect of *Helicobacter pylori*-eradication therapy on hepatic steatosis in patients with non-alcoholic fatty liver disease: a randomized-controlled pilot study. *Gastroenterol Rep (Oxf).* 2020;8(2):104-110.
 19. Yu YY, Tong YL, Wu LY, Yu XY, et al. *Helicobacter pylori* infection eradication for nonalcoholic fatty liver disease: a randomized controlled trial. *Sci Rep.* 2022;12:19530.
 20. Mantovani A, Turino T, Altomari A, Lonardo A, Zoppini G, Valenti L, et al. Association between *Helicobacter pylori* infection and risk of nonalcoholic fatty liver disease: an updated meta-analysis. *Metabolism.* 2019;96:56-65.

Publisher's Note:

Pakistan Journal of Medical & Health Sciences (Pak J Med Health Sci) remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.