EAS Journal of Parasitology and Infectious Diseases
Abbreviated Key Title: EAS J Parasitol Infect Dis
ISSN: 2663-0982 (Print) & ISSN: 2663-6727 (Online)
Published By East African Scholars Publisher, Kenya

OPEN ACCESS

Volume-6 | Issue-6 | Nov-Dec- 2024 | DOI: https://doi.org/10.36349/easjpid.2024.v06i06.001

Original Research Article

Prevalence and Risk Factors of *Helicobacter pylori* Infection among Patients with Peptic Ulcer Disease Undergoing Upper Gastrointestinal Endoscopy at Benjamin Mkapa Hospital, Dodoma, Tanzania

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Article History
Received: 24.09.2024
Accepted: 30.10.2024
Published: 11.11.2024

Journal homepage:
https://www.easpublisher.com

Quick Response Code

Abstract: ***Background*:** *Helicobacter pylori* is a bacterium infection that is a risk factor for Peptic Ulcer Disease, and affected individuals remain asymptomatic until they present with complications. This study aimed at determining the prevalence and risk factors of *Helicobacter pylori* infection among patients with Peptic Ulcer Disease undergoing upper gastrointestinal endoscopy at Benjamin Mkapa Hospital. ***Methods*:** The study was a cross-sectional analytical Hospital-based study where a quantitative approach was used. A total of 149 patients with a Peptic Ulcer Disease with the age of ≥ of 18years were recruited at Benjamin Mkapa Tertiary and Teaching Hospital between February and April 2020. Demographic and clinical characteristics were captured by using a standard questionnaire. The patients' association characteristics were tested by using the X^2 with the corresponding p-value and risk factors measured by logistic regression, a p-value of<0.05 considered significant for the study at a 95%CI. *Helicobacter pylori* infection was detected using a monoclonal antigen test. ***Results*:** The overall prevalence of *Helicobacter pylori* infection in the study was 49.66 %. *H. pylori* infection was associated with age ≥60years (AOR=5.46, 95%CI (1.77-16.86) *p*-value=0.0032), Use of unsafe drinking water (AOR=2.51, 95%CI (1.11-6.22) *p*-value 0.0255). NSAID user (AOR=3.16, 95%CI (1.10-9.11) *p*-value 0.0330). Relatives with PUD (AOR=2.46, 95%CI (1.08-5.62) *p*-value 0.0323). ***Conclusion*:** The prevalence of *Helicobacter pylori* infection was found to be relatively high in this study. *Helicobacter pylori* infection was significantly associated with an increase in age, low level of education, unsafe water, Relatives with PUD, and the use of NSAIDS.

Keywords: *Helicobacter pylori*, Peptic Ulcer Disease (PUD), Prevalence, Risk Factors, Upper Gastrointestinal Endoscopy.

BACKGROUND

Helicobacter pylori (H. pylori) is a bacteria first isolated in the human stomach in 1983 and is major risk factor for Peptic Ulcer Disease [1, 2]. It is a gram-negative, motile, multiple sheathed with four to six flagellated organisms [3]. Its size is about 0.3-5um and has a microaerophilic rod, which needs about the oxygen of 4%, carbon dioxide of 5%, and hydrogen of 5% for growth and survival [4]. *H. pylori* infection can change from the spiral shape to a coccoid form to live in a low pH gastric environment and a deep layer of gastric mucosa as a mode of facilitating its pathogenesis [5].

The risk factors for *H. pylori* infection acquisition include low SES, low level of education, poor standards of living, and poor hygiene [6–8]. Additionally, the increase in body mass index and the number of individuals in the family during childhood, chronic use of non-steroid anti-inflammatory drugs, and blood group O+ have also been reported to be the risk factors for *H. pylori* infection [6–9].

H.pylori infection colonizes the world population by more than 50%, 70% found in developing countries, 30 to 40% in developed countries, 21% among Caucasians, and 52% among African Americans [10]. Moreover, the prevalence of Infection in Sub-Sahara

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Africa ranges from 24.3% to 93.3% [3, 11]. In Tanzania, the prevalence of *H.pylori* infection ranges from 39.1% to 65% based on the studies conducted in Lake zone, Bugando Medical Centre (BMC) and Northern part of Tanzania, Kilimanjaro Christian Medical Centre (KCMC) [12, 13], with unknown magnitude in Dodoma and other regions in center Tanzania. Moreover, the prevalence of *H. pylori* infection varies from one country to another, region to region, and may differ in the same Country [14].

However, the observation seen in inpatients case reports in medical and surgical wards at DRRH patients presented with upper gastrointestinal bleeding and peritonitis, with evidently by the positivity of laboratory results by helicobacter infection most of the patients. Intraoperative findings reported peptic ulcer perforation related to Helicobacter infection. A study was done by Chalya *et al*., at BMC reported similar results among patients admitted in the surgical ward [15]. This study aimed to determine the prevalence and risk factors of *H. pylori* infection among patients with Peptic Ulcer Disease undergoing upper gastrointestinal endoscopy at Benjamin Mkapa Hospital; hence, prepare the preventive measures and help the clinician spot those with identifiable risk factors to be tested for *H.pylori* Infection.

METHOD AND MATERIALS

Study Design: A hospital-based analytical cross-sectional study was used and with a quantitative approach.

Study of Population: Involved 149 patients with PUD at the gastroenterology unit at Benjamin Mkapa Hospital.

Sampling Technique: Purposively sampling was used among 587 patients presented with dyspepsia admitted at BMH, and outpatients attended at the gastrointestinal unit at BMH consented to undergo upper gastrointestinal endoscopy. 177 patients were found with PUD, 28 patients who were using triple therapy medication (Clarithromycin, Tinidazole, and Lansoprazole) were excluded, and 149 patients were involved in the study.

Inclusion Criteria: All patients aged ≥18years agreed to participate with PUD confirmed by upper endoscopy at the Benjamin Mkapa Hospital

Exclusion Criteria: Patients on triple therapy in the previous four weeks for H. pylori infection and patients with *H. pylori* infection following triple therapy eradication**.**

Study procedure

Upper Endoscopy was performed among patients with dyspepsia. The patients were asked to fast between 6 to 8 hours before the procedure. Patients were sprayed with xylocaine 2% to allow them to swallow an endoscope through the mouthguard, and gradually, the scope was descending through the esophagus to the duodenum. A secondary video camera on the tip of the scope permitted the doctor to see on the monitor screen. The controls allowed the doctor to move them in different directions, blow air into the gut, and distend the gastric wall. Then, carefully while examining the esophagus, the endoscope was entered into the stomach. Air was added to distend the stomach for a better view and removed after the procedure as it was described in the study done by Lee *et al*., [16]. The endoscope was advanced through the pylorus to the second portion of the duodenum to allow careful examination of the pathologies based on what was done in the study done by Dayna *et al*., [17]. During the endoscopic examination, those patients with active bleeding and bleeding varices were managed by cryotherapy and banding ligation, respectively, to arrest bleeding.

Stool Sample Collection

Patients confirmed peptic ulcer diseases are requested to provide fresh stool. 50mg or 80ul of stool samples are collected using sterile stool containers. All samples transported in a cool box contained Ice Park to BMH clinical laboratory for processing following standard operating procedures. The stool samples stored at 2-8°C for up to three days, if not be processed within six hours of collection [18, 19].

Stool sample processing

The stool container was closed tightly, shake the specimen vigorously. A small portion of a stool sample was transferred into an extraction buffer. Two drops (100µl) of the extracted sample transferred to the pad of the test strip. After 5 to 20 minutes, the results were interpreted. Positive results show the presence of *H. pylori* antigen in feces with two distinct lines. Negative results show the absence of detection of *H. pylori* antigen in feces displays one red line, and the Invalid shows only one red line for the test band appeared or no red line [19].

Data analysis

Data were analyzed using Statistical Package for Social Sciences (SPSS) version 25. Associations between sociodemographic and clinical characteristics of patients tested by using the chi-square test with the corresponding p-value. A logistic regression model was used to determine the factors associated with *Helicobacter pylori* infection among patients with Peptic ulcer disease undergoing upper gastrointestinal endoscopy. A p-value of <0.05 is considered significant for the study at a 95% confidence interval (CI). A p-value of <0.1 in the Univariate analysis were included in the Multivariate analysis.

Ethical consideration

The Ethical clearance to conduct this research was approved by the Ethical Review Board, University of Dodoma. Permission was obtained from the Director of Benjamin Mkapa Hospital for data collection and written informed consent from each patient.

RESULTS

Flow chart for selection of the study participants

Figure 1 presents the flow chart involving the selection of the study participants in this study. Out of 587 patients who underwent upper GI endoscopy, 177 of them were confirmed with PUD. Of those confirmed with PUD, 149 were retained in the study, and 28 of them were excluded because found on triple therapy for treating *H. pylori* infection.

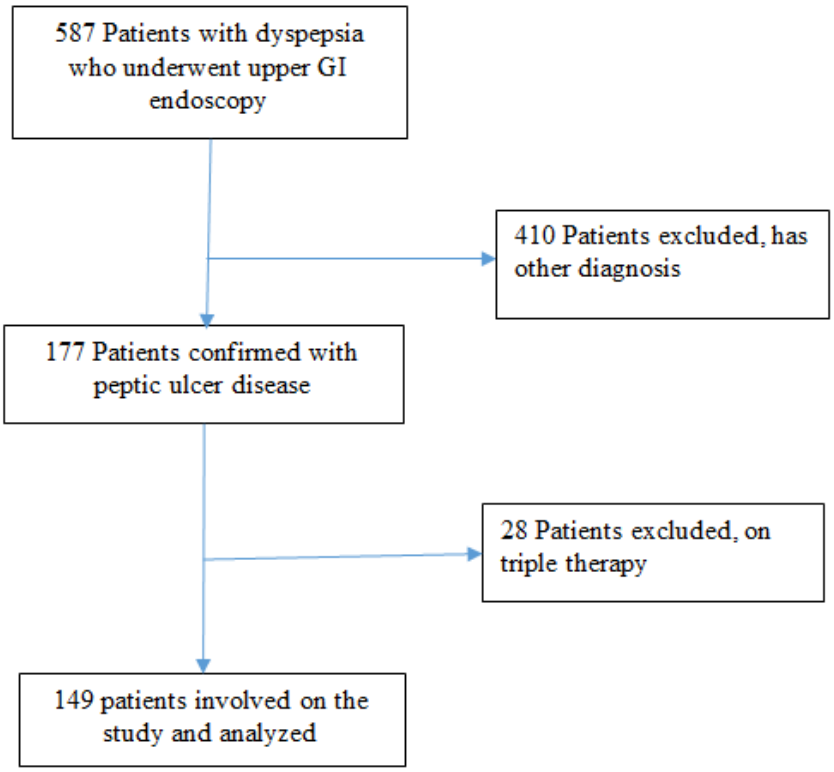

Figure 1: The flow chart of selection of the study participants

Demographic Characteristics of Respondents

One hundred seventy-seven were found with peptic ulcer disease, 28(female 17) patients met exclusion criteria, and 149 patients enrolled in the study. The respondents aged above 18 years, the mean age was 52.34, and SD 17.5. The majority of participants were aged more than 60years, 57(38.26%), Male was 77(51.68%). The majority of participants were from rural areas, 95(63.76%). Married respondents were 108(72.8%) (Table 1).

Table 1: Demographic and Clinical Characteristics of the Patients with Peptic Ulcers Disease (N=149)

Variable	**Frequency**	**Percent** Mean ±SD
Age categories (Years)		52.34±17.65
18-40	43	28.86
41-59	49	32.89
≥60+	57	38.26
Sex		
Male	77	51.68
Female	72	48.32
Place of residence		
Urban	54	36.24
Rural	95	63.76

Marital status		
Single	41	27.52
Married	108	72.48
Level of education		
Informal education	34	22.82
Primary	43	28.86
Secondary	31	20.81
Vocational training/diploma	20	13.42
University	21	14.09
Cigarette smoking		
No	130	87.25
Yes	19	12.75
Alcohol consumption		
No	107	71.81
Yes	42	28.19
A family member with PUD		
No	89	59.73
Yes	60	40.27
The use of NSAIDs		
No	115	85.91
Yes	34	14.09
Source of drinking water		
Safe water	59	39.60
Unsafe water	90	60.40
Have toilet		
No	8	5.37
Yes	141	94.63

Prevalence of *Helicobacter pylori* infection

Findings in Figure 2 show the prevalence of *H. pylori* infection among patients with 149 patients with Peptic ulcer diseases; 74 Patients have positive stool monoclonal antigen, which gives the overall prevalence of 49.66%.

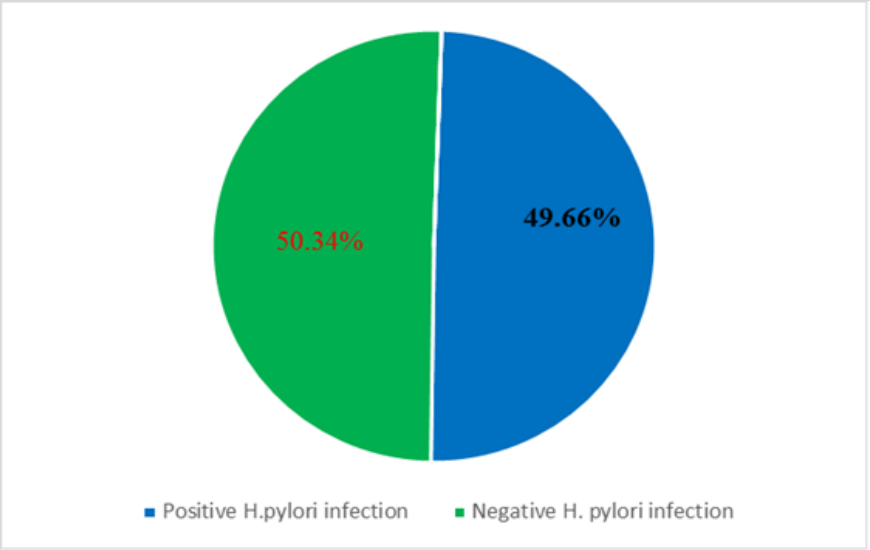

Figure 1: Prevalence of Helicobacter pylori Infection

Distribution of the Types of Peptic Ulcer Disease among study

The finding revealed the prevalence of the specific type of peptic ulcer, Gastric ulcer was 87(58.39%), while duodenal ulcer was 47(31.54%), and both gastric and duodenal were 15(10.07%).

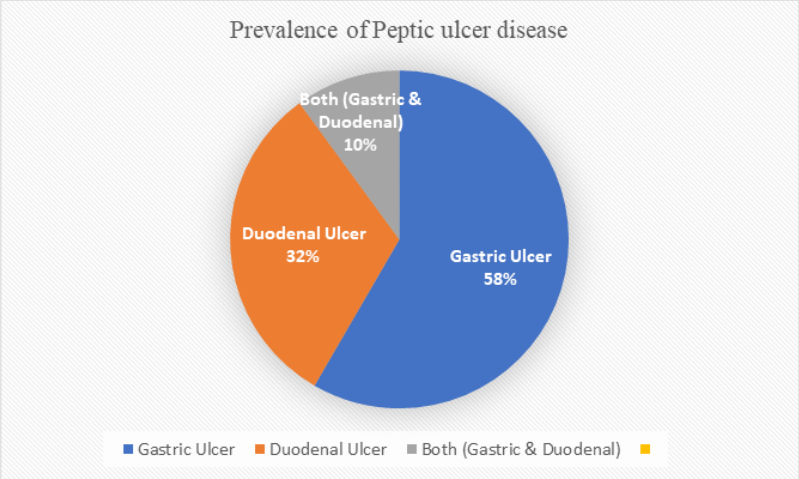

Figure 2: Distribution of the types of peptic ulcer disease

Association between Sociodemographic and Clinical characteristics of patients

Patients aged ≥60years were observed that the majority of the respondents found with *H. pylori* infection 67% followed by those with 41 to 59 years was 51%, and lastly, those aged 18-40 were 25%, and the difference was statistically significant (*p*-value =0.0002). With regard to the level of education attained by the patient, a large proportional of H. pylori infection was observed among patients with no formal education 76%, followed by respondents with primary education 53%, followed by those with secondary education 45%, followed by those with vocational training/ diploma 30%, the small proportion was observed among the participants with university level of education 24% and the difference was statistically significant (*p*-value= 0.001). A respondent with a family history of PUD in first-degree relatives, it was noted that the majority of the subject found with H.pylori infection 68% and those responded not were 38%, and the difference was statistically significant(*p*-value=0.001). Patients reported to use unsafe drinking water found to be associated with *H. pylori* infection 60% compared to those use safe drinking and difference was statistically significant (*p*-value=0.002), and large proportional of *H. pylori* infection of 68% were observed among patients with chronic use of NSAIDS compared to those were not using. The difference was statistically significant (*p*-value= 0.0170).

Table 2: **Association between Sociodemographic and Clinical characteristics of patients**

Variable	***Helicobacter pylori***		**Chi-square**	**p-value**
	Negative (N%)	**Positive (N%)**		
Age category			16.604	**0.0002**
18-40	32(74.42)	11(25.58)		
41-59	24(48.98)	25(51.02)		
60+	19(33.33)	38(66.67)		
Sex			0.540	0.4623
Male	41(53.25)	36(46.75)		
Female	34(47.22)	38(52.78)		
Place of resident			2.081	0.353
Urban	27(51.92)	25(48.08)		
Rural	48(49.48)	49(50.52)		
Marital status			0.018	0.894
Single	21(51.22)	20(48.78)		
Married	54(50.00)	54(50.00)		
Level of education			18.985	**0.001**
Never gone to education	8(23.53)	26(76.47)		
Primary school	20(46.51)	23(53.49)		
Secondary school	17(54.84)	14(45.16)		
Vocational training/diploma	14(70.00)	6(30.00)		
University	16(76.19)	5(23.81)		

Cigarette smoking			1.432	0.2314
No	63(48.46)	67(51.54)		
Yes	12(63.16)	7(36.84)		
Alcohol consumption			3.131	0.0768
No	49(45.79)	58(54.21)		
Yes	26(61.90)	16(38.10)		
Family members with PUD			11.616	**0.001**
No	55(61.80)	34(38.20)		
Yes	20(33.33)	40(66.67)		
Chronic use of NSAIDs			5.698	**0.0170**
No	64(55.65)	51(44.35)		
Yes	11(32.35)	23(67.65)		
Source of drinking water			9.712	**0.002**
Safe water	39(66.10)	20(33.90)		
Unsafe water	36(40.00)	54(60.00)		
Epigastric pain			1.208	0.2717
No	22(44.00)	28(56.00)		
Yes	53(53.54)	46(46.46)		
Heartburn			4.553	**0.0329**
No	48(44.86)	59(55.14)		
Yes	27(64.29)	15(35.71)		
Vomiting up blood			2.827	0.0927
No	68(53.13)	60(46.88)		
Yes	7(33.33)	14(66.67)		
Bilious vomiting and nausea			0.001	0.9790
No	68(50.37)	67(49.63)		
Yes	7(50.00)	7(50.00)		
Passing tarry black stool			2.949	0.0860
No	65(53.72)	56(46.28)		
Yes	10(35.71)	18(64.29)		

Logistic regression analysis for the factors associated with *Helicobacter pylori* infection

The age (p = 0.0113), source of drinking water (p = 0.0255), family history of PUD (p = 0.0323) and the use of non-steroid NSAIDS (p = 0.0330) were independent predictors of H. pylori infection. The results showed that for patients aged 60 years and above, the odds of H. pylori infection was five times more than the age group 41 to 59 years, and the association was statistically significant (p = 0.0032). Also, patients aged 41-59 years old had odds of H. pylori infection of three times more than those in the age group 18-40 years old, and the association was statistically significant (p = 0.0235).

The odds of acquiring *H. pylori* infection for patients using unsafe drinking water were 2.5-fold compared to those using safe drinking water, and the association was significant (p = 0.0255). Having a family history of PUD increased the likelihood of having *H. pylori* infection 2.5-fold compared to the patients from families without a history of *H. pylori* infection. The association was significant (p = 0.0323). Moreover, users of NSAIDs had 3-fold odds of being affected by *H. pylori* infection compared to non-user NSAIDS, and the association was statistically significant (p = 0.0323). Level education (p = 0.0999), drinking alcohol (p = 0.0758), heartburn (p = 0.276), vomiting up blood (p = 0.8193) and passing black tarry stool (p = 0.8133) were not risk factors of *H. pylori* infection (Table 3).

Table 3: Logistic regression analysis for the factors associated with Helicobacter Pylori infection

Variable	Univariate analysis		Multivariate analysis	
	OR(95%IC)	**P-Value**	**AOR(95%IC)**	**P-Value**
Age				
18-40	Reference		Reference	
41-59	3.03[1.25,7.34]	**0.0141**	3.61[1.19,10.95]	**0.0235**
60+	5.84[2.42,14.01]	**<0.0001**	5.46[1.77,16.86]	**0.0032**
Sex				
Male	Reference			

Female	1.27[0.67,2.42]	0.4631	-	-
Place residence				
Urban	Reference			
Rural	0.98[0.5,1.91]	0.9507	-	-
Marital status				
Single	Reference			
Married	1.05[0.51,2.16]	0.8942	-	-
Level of education				
Informal education	10.40[2.894,37.374]	**0.0003**	3.46[0.789,15.210]	0.0999
Primary	3.68[1.143,11.85]	**0.029**	2.129[0.572,7.925]	0.2600
Secondary	2.635[0.772,9.001]	0.1221	2.334[0.608,8.964]	0.2170
Vocational training/diploma	1.371[0.343,5.488]	0.6553	0.835[0.163,4.264]	0.8282
University	Reference		Reference	
Cigarette smoking				
No	Reference			
Yes	0.55[0.20,1.48]	0.2362	-	-
Alcohol consumption				
No	Reference		Reference	
Yes	0.52[0.25,1.08]	**0.0789**	0.44[0.18,1.09]	0.0758
Family members suffering from PUD				
No	Reference		Reference	
Yes	3.24[1.63,6.43]	**0.0008**	2.46[1.08,5.62]	**0.0323**
Chronic use of NSAIDs				
No	Reference		Reference	
Yes	2.62[1.17,5.88]	**0.0192**	3.16[1.10,9.11]	**0.0330**
Source of drinking water				
Safe	Reference		Reference	
Unsafe	2.93[1.48,5.80]	**0.0021**	2.51[1.11,6.22]	**0.0255**
Presence of toilet				
No	Reference			
Yes	3.13[0.61,16.04]	0.1711	-	-
Epigastric pain				
No	Reference			
Yes	0.68[0.34,1.35]	0.2726	-	-
Heartburn				
No	Reference		Reference	
Yes	0.45[0.22,0.95]	**0.0348**	0.55[0.22,1.40]	0.276
Vomiting up blood				
No	Reference		Reference	
Yes	2.27[0.86,5.99]	**0.0988**	0.87[0.26,2.91]	0.8193
Bilious vomiting and nausea				
No	Reference			
Yes	1.02[0.34,3.05]	0.9790	-	-
Passing tarry black stool				
No	Reference		Reference	
Yes	2.09[0.89,4.90]	**0.0899**	0.88[0.31,2.48]	0.8133

DISCUSSION

Prevalence of *Helicobacter pylori* infection

The study was conducted to determine the prevalence and risk factors of *H. pylori* infection among patients with PUD after being confirmed by upper gastrointestinal endoscopy. The prevalence in this study was 49.66%.

The prevalence of *H. pylori* Infection in this study was 49.66% among patients with PUD, which is lower than 65% and higher than 39.1%, which was reported in two different studies which were done at KCMC and BMC in Tanzania respectively [12, 13]. Low prevalence rates have also been reported in the studies which were done at Makerere (29.9%) [20] and Mbarara (24.3%) [11]. Furthermore, high prevalence rates were also reported in In Ethiopia (52.2%) and Nigeria (52.2%)

[9]. In sub-Sahara Africa, Nigeria, and Benin, the prevalence of H. pylori infection was reported to be 93.3%, 81.7%, and 74%, respectively [1, 3, 21].

Inconsistence of the prevalence has also been reported in other areas. For example, the Kingdom of Saud Arabia (46.5%) [22], India (77.69%) [23]. Cuba (73.55%) and Nepal (8%) [24, 25]. The study was conducted in South Florida by Rahul *et al.*, reported a prevalence of 51%, which is close to the prevalence reported in the present study [26] also as the prevalence was reported in the study that was done in Yangzhong city, China (51.2%) [27].

The prevalence obtained in this study and previous studies shows that there is quite a variation in the prevalence of *H. pylori* infection globally. The variation may be due to the test used; this study used a monoclonal antigen to detect *H. pylori*, which is not comparable with standard gold histology [28]. Other studies also used different tests that differ insensitivity, and specificity may contribute to prevalence variation among studies [29, 30].

Patients included in this study only attended at the gastrointestinal unit and admitted at BMH with the ability to pay for upper endoscopy. This may have contributed significantly to selection bias hence leading to the relatively high prevalence observed. The rapid increase in industrialization and improved social services in developed countries has been shown to reduce *H. pylori* infection [31].

Factors influencing *Helicobacter pylori* Infection

Age of individuals

The results revealed that participants aged 60 years and above were associated with H. pylori infection, similar to the finding observed in Ethiopia [32–36]. Furthermore, middle age group (40-60 years) old was found to be associated with *H. pylori* infection, which differs from other studies [10, 14, 22, 24, 37].

The high prevalence of *H.pylori* Infection among old patients may be due to age cohort effects among geriatrics [34, 38, 39]. In developed countries, *H. pylori* infection in childhood has been decreasing due to improved social services and living standards, but the prevalence among adults is significantly high because of the cohort effect [7, 8, 29]. Additionally, stress, anxiety, and depression (SAD) has been hypothesized by changes of Brain-Derived Neurotrophic Factor in the hippocampus and amygdala, which are independent risk factors for H. pylori infections [29, 40–43]. Moreover, SAD has a subtle presentation that leads to late-diagnosed when it is already complicated [44].

The immune system's role among geriatric patients has been reported to be caused by age-related changes in the hippocampus with the increase in IL-1β and lower interleukin (IL-4) [45]. A low level of IL-4 has been studied to be associated with the reduction of activation of T cells, B cells, and differentiation of naïve helper cells (Th 0 cells) to Th2 cells [46]. As the immune system goes down, the ability to clear infection regardless of antibiotics becomes impaired, leading to the persistence of *H. pylori* infection among adults [36, 45, 47, 48].

The physiological changes that occur with an increase in age of an individual progressively induce gastric frailty, which leads to the reduction of the protective barriers such as bicarbonate, mucous layer, and the level of prostaglandins [49, 50]. Therefore, it will reduce mucosal blood flow and decrease gastric acid secretion and predispose to the development of PUD in geriatric groups in addition to *H. pylori* infection and co-morbidities [49, 50].

Chronic use of non-steroid anti-inflammatory drugs

This study points out that respondents who reported using NSAIDs were more likely to suffer from PUD. The effect may be higher when co-existing with *H. pylori* infection. The findings support results from other studies conducted in China and Saudi Arabia [51, 52]. However, related research in Spain and Lebanon revealed that NSAIDs and co-existing H. pylori infection have a significant synergy effect on PUD's causation [53–56]. Additionally, other findings from the meta-analysis show that individuals using NSAIDS, the possibility of causing PUD by 25%, and rises to 41% when it becomes co-existing with *H. pylori* infection. On the other hand, the use of NSAIDs has a strong association with gastrointestinal bleeding by 1.8 to 6 times when co-existing with *H. pylori* infection [57, 58].

Furthermore, inconsistent results have been reported by Laine in Southern California, and Konturek *et al.*, in Poland revealed to be protective among NSAIDS users [59, 60]. NSAIDs have been associated with PUD because of their properties and mode of action [29]. As its weak acid remains in the acidic stomach's lipophilic form as soon as it diffuses the lipid membrane, the epithelium becomes ionized, followed by damaging gastric epithelial [29]. Also, NSAIDs inhibition of cyclooxygenase enzymes predisposes the gastric epithelium for ulceration. The risk doubled in the presence of *H. pylori* infection [50, 59]. The chronic use of NSAIDs has a genetic predisposition in single nucleotide polymorphism [29]. The presence of NSAIDs and *H. pylori* infection doubles the risk [29].

Individuals with a first-degree relative with peptic ulcer disease

The study results revealed that participants with a history of peptic ulcer disease in the first-degree relative positively associate with *H. pylori* infection. Similar to the studies conducted in Mbarara by Atila *et al*., in 2019 and sub-Sahara Africa by Aguemon *et al*., in 2005 and Smith *et al*., in 2018 and the United Arab Emirates [62]. However, the study conducted by Herman *et al.,* 1998 in German shows that those with a family history of peptic ulcer disease were found to be associated by two times, which is closely similar results of the study performed by Brenner *et al.,* in 1998. Additionally, studies conducted in developed countries like the USA and German showed that the first-degree relative to be an independent risk factor for *H. pylori* infection [64], [65]. Also, inconsistent results were found in Denmark and South Florida, which reported a negative association between family members with PUD and H.pylori infection [19, 66].

Furthermore, this study's findings show that the odds of *H.pylori* infection among first-degree relative acquisition were an independent risk factor, which is as hypothesized by genetic inspiration [38, 67]. The genetic polymorphism shows a strong association with *H. pylori* infection in a family member with blood group O+, and twins have been reported among family members with PUD, which is an independent risk factor for *H. pylori* infection [68].

The use of unsafe drinking water

This study's results indicate that respondents who used to drink unsafe water were at high risk of developing *H. pylori* infection, similar to the findings observed in the studies done in BMC and Mbarara by Attila *et al.,* 2019 and Jaka *et al.,* 2016. A similar cross-sectional study in Kano, Nigeria, and Northwest Ethiopia reported the same results [1, 69]. The study done in German shows similar findings [70]. Additionally, the use of unsafe water is not only associated with H. pylori infection but also other diseases like Typhoid fever and Trachoma, which both of the illnesses reported in Dodoma which is primarily due to water insecurity [71–74].

Contrary to the results of this study is a study conducted in South Florida about drinking unsafe water. It found that unsafe water unlikely to be associated with H. pylori infection [26]. However, nearly half of the participants involved in this study are coming from rural areas. Meanwhile, the provision of safe water can be a problem due to geographical location and infrastructure for the government to supply water in these remote areas.

The use of unsafe drinking water is related to the shortage of water availability, of which an individual has no option apart from using it. The Dodoma region' s is a semi-arid place, and it experiences drought most of the months with an annual rainfall of around 570 mm per year. This situation causes more than 70% of the rural community to suffer from the water crisis, hence, becoming vulnerable to water-borne disease and water-washed diseases [71, 75, 76].

Cigarette smoking and alcohol consumption

The finding of the study reviewed that those patients reported cigarette smoking found to be protective against *H.pylori* infection; this is similar to the studies conducted in Ethiopia, Nigeria, China, and Japan [27, 51, 77, 78]. This study is inconsistent with studies conducted in the US and northern against cigarette smoking and *H. pylori* infection [79, 80] This study's inconsistent results study can be due to sampling techniques used to recruit at Benjamin Mkapa Hospital. Across-sectional study design not suitable for the disease of long duration, and cigarette smoking is dose dependant risk factor and genetic predisposition [29, 81].

The study shows that alcohol consumption is protective against *H.pylori* infection, with similar results reported in the Republic of Georgia and Brazil [79, 82]. Moreover, the duration and amount of alcohol may give different results; an individual consuming a moderate amount of alcohol per week is protective against *H.pylori* infection [83]. Average alcohol consumption acts as a bacteriocidal against H. pylori infection. It increases gastric acid secretion; both protect against *H. pylori* infection, dose-dependent, and type of alcohol, which they differ preparation methods [84].

Limitation of the Study; The study was a cross-section hospital study, which is not suitable for a disease of long duration and investigation of disease of causality [81], Histopathology was not done, at that time no pathologist in Dodoma would interpret tissue samples taken during OGD and give results, The study was conducted with low funds, which would involve many centers in Tanzania, transportation of taken tissue samples to the center where the pathologist is available for results interpretation, also purchasing equipment for PCR, as may help in the identification of H. pylori hovering pathogenic islands and antibiotic resistance and also time duration for data collection was among of limitation of this study.

CONCLUSION

The overall prevalence of *H. pylori* infection among patients with PUD was relatively high. However, *H. pylori* infection was significantly associated with an increase in age, low level of education, drinking unsafe water, chronic use of NSAIDS, and individuals with the first-degree relative with PUD were the independent risk factors for *H. pylori* infection in the study.

Acknowledgment

The author would like to convey a great appreciation to Dr. Abdallah R. Mlwati, Dr. Bonaventura C.T Mpondo, and Dr. Masumbuko Y. Mwashambwa, my research supervisors, for their esteemed input to this study and the Head of department Clinical Medicine Dr. John R. Meda, Head of department public health and epidemiology Dr. Leonard K. Katalamula, Dr.Secilia K. Ng'weshemi, Dr. Boaz M. Matobogolo. Moreover, I would like to express my gratitude to the Dodoma University and Ethical Review Committee for approval for this study. Dr. Alphonce Chandika, Benjamin Mkapa Hospital Director, for permitting the research to be carried out.

Abbreviation

AOR	Adjusted Odds Ratio
BabA	Blood-group Antigen-Binding Adhesion
BMC	Bugando Medical Centre
BMH	Benjamin Mkapa Hospital
BMI	Body Metabolic Index
CUHAS	Catholic University Health and Allied Science
DU	Duodenal ulcer
Dup A	Duodenal Ulcer-Promoting Gene
GU	Gastric Ulcer
H. Pylori	*Helicobacter pylori*
KCMC	Kilimanjaro Christian Medical Centre
MALT	Mucosal-Associated Lymphoid Tissue Lymphoma
NIMR	National Institute for Medical Research
NSAIDs	Nonsteroidal Anti-Inflammatory Disease Drugs
OGD	Oesophago-Gastroduodenoscopy
OPD	Outpatient Department
OR	Odds Ratio
PPI	Proton Pump Inhibitor
SPSS	Statistical Package for the Social Sciences
UDOM	University of Dodoma
UGIB	Upper Gastrointestinal Bleeding
USA	United States of America

Data Availability: The data is available on request from the corresponding author

Conflict of Interest: The authors haves no conflict

Funding statement: This study was no fund obtained.

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Cite This Article: John D. Calori, Nazir J. Temba, Peter M. Karoli, Secilia K. Ng'weshemi, Boaz M. Matobogolo, Masumbuko Y. Mwashambwa, Bonaventura C. T. Mpondo, Abdallah R. Mlwati (2024). Prevalence and Risk Factors of *Helicobacter pylori* Infection among Patients with Peptic Ulcer Disease Undergoing Upper Gastrointestinal Endoscopy at Benjamin Mkapa Hospital, Dodoma, Tanzania. *EAS J Parasitol Infect Dis*, *6*(6), 58-71.