**International Journal of Immunology**
2015; 3(1): 7-13
Published online March 3, 2015 (http://www.sciencepublishinggroup.com/j/iji)
doi: 10.11648/j.iji.20150301.12
ISSN: 2329-177X (Print); ISSN: 2329-1753 (Online)

# *H. Pylori* Prevalence and Its Effect on $CD_4^+$ Lymphocyte Count in Active Pulmonary Tuberculosis Patients at Hospitals in Jimma, Southwest Ethiopia

**Wakjira Kebede[1, 2, *], Biniam Mathewos[2], Gemeda Abebe[1]**

[1]Department of Laboratory sciences and Pathology, Collage of Public Health and medical sciences, Jimma University, Jimma, Ethiopia
[2]Department of Immunology and Molecular Biology, School of Biomedical and Laboratory sciences, Collage of Medical sciences, University of Gondar, Gondar, Ethiopia

**Email address:**
dandiwk2004@gmail.com (W. Kebede)

**To cite this article:**
Wakjira Kebede, Biniam Mathewos, Gemeda Abebe. *H. Pylori* Prevalence and Its Effect on $CD_4^+$ Lymphocyte Count in Active Pulmonary Tuberculosis Patients at Hospitals in Jimma, Southwest Ethiopia. *International Journal of Immunology.* Vol. 3, No. 1, 2015, pp. 7-13.
doi: 10.11648/j.iji.20150301.12

**Abstract:** *Background: Helicobacter pylori*, a lifelong and typically asymptomatic infection of the stomach, strongly alter gastric immune responses. The present study aimed to survey the prevalence and related risks of *H. pylori* infection among tuberculosis (TB) patients at hospitals in Jimma City, Southwest Ethiopia. *Methods:* Comparative cross sectional study was conducted from February to June, 2014. Fifty four PTB patients and an equal number of non TB controls were enrolled. Convenient sampling technique was used to select the study participants. Structured questionnaire was used to collect socio demographic and clinical data. The stool for *H. pylori* antigen detection and venous blood for $CD_4^+$ lymphocyte count was collected. *Results:* Among 108 study participants, 62 (57.4%) was females. Majority of the study participants, 48 (44.4%) were in the age group of 18-34 years and the mean age of the participants was 37.5 ± 10.7 SD. The prevalence of *H. pylori* infection among TB patients and non TB controls were 19 (35.2%) and 11 (20.4%), respectively. TB patients with $CD_4^+$ lymphocyte count of less than 200Cells/$mm^3$ was more likely to be infected. *Conclusion: H. pylori* infection among TB patients was significantly higher than non TB controls. Low $CD_4^+$ lymphocyte count was found to be associated with high *H. pylori* infection among TB patients. Further study should be undertaken to reveal the potential pathogenic mechanisms for underlying associations for *H. pylori* and TB infection.

**Keywords:** *H. pylori,* Pulmonary Tuberculosis, $CD_4^+$ Lymphocyte, Jimma

## 1. Introduction

*Helicobacter pylori* (*H. pylori*) and *Mycobacterium tuberculosis* (*M. tuberculosis*) is the most prevalent and highly contagious infectious microorganisms around the world [1]. Roughly, half of the world populations is infected with *H. pylori,* a gram negative, microaerophilic bacteria, that colonizes the gastric mucosa and the epithelial lining of the human stomach; infects approximately 80% of people living in TB endemic regions [2]. The prevalence of the infection varies from country to country with the largest difference being observed between developed and developing countries where 50-90% in developing countries and 20-50% in developed countries population are infected [3].

*H. pylori* infection is typically acquired in early life via oral-oral or fecal-oral pathways and chronic infection is strongly linked to the development of gastric cancer and peptic ulcer disease [4]. Approximately 17% of infected patients develops peptic ulcers, and one-fourth of those patients experience an ulcer complication [5]. Studies have demonstrated that *H. pylori* infection may involve in the development of adenocarcinoma of the stomach, moreover, it has been contributed to the development of respiratory tract disease [6].

Lower respiratory tract infection is mostly caused by *M. tuberculosis*. Immunity to TB could be disrupted by many conditions like Human immunodeficiency virus (HIV) infection. *H. pylori* infection is also able to manipulate certain arms of the immune responses and sometimes leads to development of chronic inflammation that activates and aid to release molecules that can modulate immunity to TB. A history of PTB might be associated with increased prevalence

of *H. pylori* infection. However, no studies examined the prevalence of *H. pylori* infection among TB patients at the study area. Therefore, this study was aimed to determine the prevalence and risk factors for *H. pylori* infection among newly diagnosed PTB patients visiting TB diagnosis and treatment facilities at Jimma University specialized hospital.

# 2. Methods and Materials

### *2.1. Study Design and Setting*

Hospital based comparative cross-sectional study was conducted from February to June, 2014 in Jimma City. The City is located in Oromia Regional State at 352 KMs Southwest direction of Addis Ababa, Capital City of Ethiopia. There are two public hospitals in Jimma City: Jimma University Specialized Hospital (JUSH) and Shanan Gibe Hospital (SGH). Both hospitals are organized into different departments, of which TB Clinic is one of the departments [7]. The hospitals provide the inpatient and outpatient services, including care and treatment for TB patients being evaluated for a routine medical examination, including screening for TB and other opportunistic infection. All Laboratory work was done at Mycobacteriology laboratory research center of Jimma University.

### *2.2. The Population*

The study population was all consecutive, consented new smears positive outpatients pulmonary tuberculosis (PTB) patients aged ≥18 years visiting the two hospitals in Jimma City during the study period. Patients with previous history of anti TB treatment for more than two weeks, relapses, smear negative and extra- pulmonary TB cases were excluded.

### *2.3. Sample Size*

The sample size was determined by taking the prevalence of *H.pylori* from the previous study [8]. The sample size was calculated based on the sampling method recommended for double proportion populations. Therefore, the final sample size calculated was included 54 PTB confirmed patients and an equal number of non TB controls, Age and sex much with the cases.

### *2.4. Selections of Study Participants*

Two public hospitals were purposively selected in order to obtain the required number of study subjects, to minimize the study period and for effective utilization of resources. The sample size was proportionally allocated on the total number of new smears positive PTB patients registered in each hospital in the previous three months in the year 2013 and it was 196 and 103 in JUSH and SGH, respectively. Thus, in total 299 new smear positives PTB patients were registered in the same three months and the sample size was allocated for each hospital was 36 and 18 for JUSH and SGH respectively.

### *2.5. Sample Collection and Processing*

Three consecutive sputum specimens (spot, morning and spot) were collected by a laboratory technologist from each study participant in pre-labeled sterile wide-mouthed container with a secure, tight-fitting cover from TB suspected cases for acid fast bacilli (AFB) by Ziehl-Neelsen (ZN) staining technique [9]. Those patients who were smear positive was included in the study while those with smear negative results were further transported to Mycobacteriology laboratory research center of Jimma University for TB culture. Structured questionnaire was prepared in English and then translated into local language (Amharic and Afan Oromo) and it was used to collect the information on all study participants. The content of the questionnaire included socio demographic information and clinical characteristics of the patients including their HIV status. HIV status of the participants was obtained from the patients' card.

### *2.6. Sample Processing, Mycobacterial Culture and Isolation*

A volume of 1 to 2 milliliters of the smear negative sputum samples was digested, homogenized and decontaminated using equal volume of standard *N* -acetyl-L-cysteine-sodium hydroxide (NALC-NaOH). Briefly, the organisms were concentrated on centrifugation at 3000rpm for 15 minutes at $4^0$C and the sediment was reconstituted using 1 milliliter of sterile phosphate-buffer saline (PBS) (pH =6.8). The sediment was inoculated into Mycobacteria Growth Inhibitor Tube (MGIT) 960 vials supplemented as described by the manufacturer. All inoculated MGIT vials were incubated at $37^0$C in the MGIT 960 instrument (BACTEC™ 960) and inspected for growth of Mycobacteria either until they were flagged positive by the instrument or for a maximum of 42 days for primary isolation of the organisms [10]. Then after, stool and venous blood were collected from all study subjects for *H. pylori* antigen detection using ELISA and counting $CD4^+$ lymphocytes using Fluorsent activated cells sorting (FACS) count flow cytometry.

### *2.7. ELISA for Stool Antigen Detection*

The stool antigen test was performed on frozen stool samples stored at $-20^0$C. The stool sample was approximately the size of a pea and it was added to 200µl of diluent buffers with pH =7.2. It was mixed thoroughly using a vortex. Then, the samples were centrifuged at 5000rpm for 10 minutes. Supernatant of faecal suspension and a peroxidase labeled monoclonal antibody was added and then the preparation was incubated for 1 hour of room temperature with shaking. Washing was done followed by addition of enzyme substrate and incubated for 10 minutes at room temperature. This was followed by addition of a stop solution and the results were read by ELISA reader. According to manufacturer's instruction, optical density value ≥ 0.190 and < 0.190 at 450 nm was taken as positive and negative for *H.pylori* infection respectively [11, 12].

### *2.8. $CD_4^+$ Lymphocyte Count by FACS Count Flow Cytometry*

Two milliliters of whole blood specimen was collected from all study participants by vein puncture using evacuated blood collection tubes with Ethylenediamine tetra-acetic acid (EDTA). The blood specimen container was labeled with the time of specimen collection of unique identification numbers and transported to the JUSH laboratory. Then the collected whole blood was analyzed for $CD_4^+$ T cell count before 48 hours of its collection using BD FACS Count (Version1.0, Germany)

### *2.9. Quality Controls*

A known positive and negative slides was included with each run of ZN staining procedure during confirmatory tests of AFB from culture media. MGIT media were inoculated with known strain of $H_{37}Rv$ as positive control and sterile PBS inoculated and incubated for each batch as negative controls. Calibration of FACS count flow cytometry and ELISA was done according to the instruments manual for both the reagent and the machine. For all tests carried out in the laboratory, standard operating procedure under aseptic condition was followed for better monitoring of contamination.

### *2.10. Statistical Analysis*

Data were edited, cleaned, coded, and entered into a computer and analyzed using Statistical Package for Social Sciences (SPSS) version 21.0. Descriptive analysis, and odds ratio (OR) with 95% confidence interval were calculated. Binary logistic regression was applied to see the association with independent variables and outcome variable. Bivariate analysis was used to screen those variables which are candidates for multivariate analysis and those variables having P value ≤ 0.2 were reanalysed by multivariable analysis to control confounding [13]. The strength of associations with *H. pylori* infection and independent variables were measured by Adjusted Odds Ratio (AOR) with 95% confidence interval. Variables with P value ≤ 0.05 were considered as statistically significant.

### *2.11. Ethical Considerations*

The study protocol was reviewed and approved by the ethical committee of School of Biomedical and Laboratory Science, college of Medicine and Health Sciences (SBLS), University of Gondar and Jimma University College of Public Health and Medical Sciences. An official support letter was written obtained from SBLS of University of Gondar and submitted to administration offices of both hospitals from which support letter was obtained to be submitted to TB Clinic departments and Institute of Biotechnology and Mycobacteriology Laboratory research center of Jimma University. After the objective of the study was explained to the study participants and written consent in the local language was obtained from each participant. Results of laboratory investigations were referred to their attending clinician for treatment according to national treatment guidelines.

## 3. Results

### *3.1. Socio Demographic Characteristics of the Study Participants*

Among 108 study participants included in this study, 62 (57.4%) was females and 46 (42.6%) was males. Majority of the study participants 48 (44.4%) was in the age group of 18 - 34 years. The mean age of the participants was 37.5 ± 10.7 SD (range: 18 to 65 years) and 81 (75.0%) of them was from rural residents and 27 (25.0%) were from urban (Table 1).

***Table 1.*** *Socio demographic characteristics of the study participants at hospitals in Jimma City, Southwest Ethiopia, from February to June, 2014 (n = 108).*

| Characteristics | TB patients (n= 54) | Non TB controls (n= 54) | Total n (%) |
|---|---|---|---|
| Sex | | | |
| male | 23 | 23 | 46 (42.6) |
| female | 31 | 31 | 62 (57.4) |
| Age groups (in years) | | | |
| 18-34 | 24 | 24 | 48 (44.4) |
| 35-44 | 16 | 16 | 32 (29.7) |
| ≥ 45 | 14 | 14 | 28 (25.9) |
| Marital status | | | |
| Single | 20 | 18 | 38 (35.2) |
| Married | 34 | 36 | 70 (64.8) |
| Educational level | | | |
| Illiterate | 24 | 24 | 48 (44.5) |
| Literate | 30 | 30 | 60 (55.5) |
| Occupational status | | | |
| Civil servant | 6 | 9 | 15 (13.9) |
| Housewife | 19 | 22 | 41 (37.9) |
| Merchant | 15 | 7 | 22 (20.4) |
| Others* | 14 | 16 | 30 (27.8) |
| Place of residence | | | |
| Urban | 15 | 12 | 27 (25.0) |
| Rural | 39 | 42 | 81(75.0) |

* = students, daily laborer & farmer

### *3.2. Prevalence of H. Pylori Infection among TB Patients and Non TB Controls*

The prevalence of *H. pylori* infection was among TB patients significantly higher (COR = 2.59, 95% CI: 1.892-5.047, P = 0.032) in TB patients 19/54 (35.2%) than non TB controls 11/54 (20.4%). Age specific prevalence for 18-34 years, 35-44 years and greater or equal to 45 years were 5/19 (26.3%), 5/19 (26.3%) and 9/19 (47.4%), respectively (Figure 1).

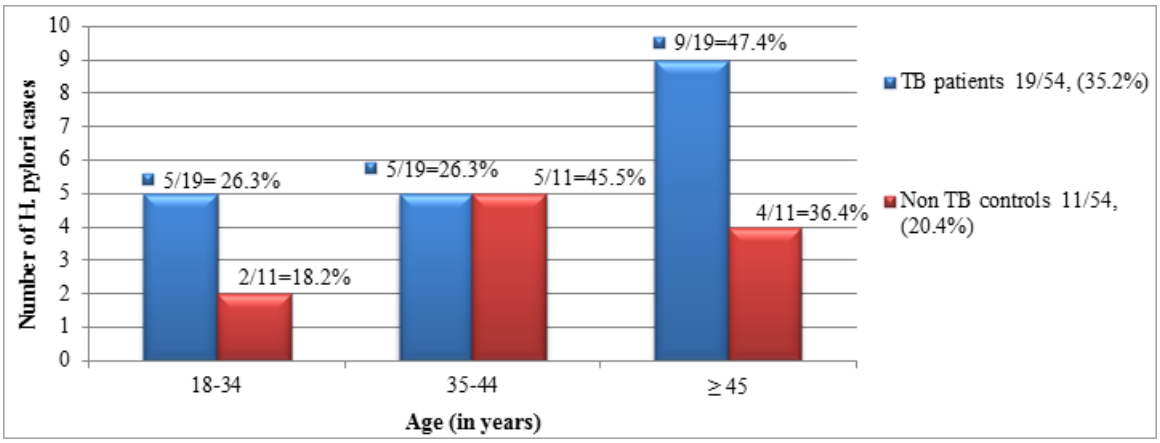


***Figure 1**. H. pylori prevalence among TB patients and non TB controls according to the age category.*

Among all of the TB patients 11/54 (20.4%) were HIV positive. Among these HIV positive individuals only 1/11 (9.1%) were infected with *H. pylori.* On the other hand, among HIV negative individuals 18/43 (41.9%) of them were infected with *H. pylori* infection. HIV status of TB patients had statistically significant associations with *H. pylori* infection (AOR= 0.09, 95% CI: 0.01 -0.97, P = 0.048).

### *3.3. Factors Associated with H. Pylori Infection among TB Patients*

There was no significant association with *H. pylori* infection and gender (COR = 2.63, 95% CI: 0.84 - 8.30, P = 0.098). However, significant higher prevalence of *H. pylori* infection was observed among TB patients with $CD_4^+$ lymphocyte count less than 200 cells/$mm^3$10/19 (52.6%) than with $CD_4^+$ lymphocyte count of greater or equal to 350 cells/$mm^3$, TB patients who had $CD_4^+$ lymphocyte count of less than 200 cells/$mm^3$ were almost ten times more likely to be infected by *H. pylori* than TB patients who had $CD_4^+$ lymphocyte count of greater than or equal to 350 cells/$mm^3$ (AOR = 9.96, 95% CI: 1.971 - 50.0, P = 0.005). Among the TB patients, the HIV positive individuals (all on ART treatment) was 0.09 times less likely to be infected by *H. pylori* than HIV negative individuals (AOR= 0.09, 95% CI: 0.01 – 0.97, P=0.048) (Table2).

***Table 2.** Logistic regression for selected risk factors for H. pylori infection among TB patients at hospitals in Jimma City, Southwest Ethiopia, from February to June, 2014 (n = 54).*

| Variables | *H. pylori* infection | | COR (95% CI) | P value | AOR (95% CI) | P value |
|---|---|---|---|---|---|---|
| | Negative n (%) | Positive n (%) | | | | |
| Sex | | | | | | |
| Male | 12 (34.3) | 11 (57.9) | 2.63 (0.83-8.4) | 0.098 | - | - |
| Female | 23 (65.7) | 8 (42.1) | 1 | | | |
| Age (in years) | | | | | | |
| 18-34 | 19 (54.3) | 5 (26.3) | 1 | | | |
| 35-44 | 11 (31.4) | 5 (26.3) | 1.72 (0.4-7.3) | 0.458 | - | - |
| ≥ 45 | 5 (14.3) | 9 (47.4) | 6.84 (1.6-29.79) | 0.010 | - | - |
| $CD_4^+$ T cell count | | | | | | |
| <200 Cells/mm3 | 5 (14.3) | 10 (52.6) | 7.6 (1.77 - 32.6) | 0.006 | 9.96 (1.97 – 50) | 0.005 |
| 200-349 Cells/mm3 | 11 (31.4) | 4 (21.1) | 1.4 (0.3 - 6.2) | 0.675 | 1.39 (0.29 – 6.5) | 0.677 |
| ≥350 Cells/mm3 | 19 (54.3) | 5 (26.3) | 1 | | 1 | |
| Place of residence | | | | | | |
| Urban | 10 (28.6) | 5 (26.3) | 1 | | | |
| Rural | 25 (71.4) | 14 (73.7) | 1.12 (0.3-3.94) | 0.860 | - | - |
| HIV serostatus | | | | | | |
| Positive | 10 (28.6) | 1 (5.3) | 0.14 (0.02-1.2) | 0.071 | 0.09 (0.01-0.97) | 0.048 |
| Negative | 25 (71.4) | 18 (94.7) | 1 | - | 1 | - |
| Occupational status | | | | | | |
| Civil servants | 4 (11.4) | 2 (10.5) | 1 | | | |
| House wife | 14 (40.0) | 5 (26.3) | 0.714 (0.01-5.2) | 0.739 | - | - |

| Variables | *H. pylori* infection | | COR (95% CI) | P value | AOR (95% CI) | P value |
|---|---|---|---|---|---|---|
| | Negative n (%) | Positive n (%) | | | | |
| Merchants | 9 (25.7) | 6 (31.6) | 1.3 (0.2-9.7) | 0.777 | - | - |
| Others | 8 (22.9) | 6 (31.6) | 1.5 (0.2-11.0) | 0.691 | - | - |
| Educational status | | | | | | |
| Illiterate | 12 (34.3) | 12 (63.2) | 3.3 (1.03-10.5) | 0.045 | 2.92 (0.77-11.1) | 0.06* |
| Literate | 23 (65.7) | 7 (36.8) | 1 | | | |
| Marital status | | | | | | |
| Single | 14 (40.0) | 6 (31.6) | 1 | | | |
| Married | 21 (60.0) | 13 (68.4) | 1.5 (0.5-4.7) | 0.541 | - | - |
| Family size | | | | | | |
| 2-4 | 12 (34.3) | 3 (15.8) | 1 | | | |
| ≥ 5 | 23 (65.7) | 16 (84.2) | 2.7 (0.7-11.4) | 0.157 | - | - |
| Smoking habits for the last six months | | | | | | |
| Yes | 5 (14.3) | 7 (36.8) | 3.5 (0.93-13.2) | 0.065 | - | - |
| No | 30 (85.7) | 12 (63.2) | 1 | | | |
| Alcohol consumption for last six months | | | | | | |
| Yes | 8 (22.9) | 6 (31.6) | 1.5 (0.5- 5.4) | 0.447 | - | - |
| No | 27 (77.1) | 13 (68.4) | 1 | | | |

Others = student, daily laborer and farmer, * = marginally insignificant in backward stepwise logistic regression

# 4. Discussion

In this study, the prevalence of *H. pylori* infection was 19/54 (35.2%) and 11/54 (20.4%) in TB infected patients and non TB controls, respectively. However, the prevalence of *H.pylori* in the general population was higher in other several studies [2, 3, 6]. Other studies conducted on the same type of study participants with the present study, showed different findings than the present study. For example, a study conducted in Athens reported *H. pylori* infection with prevalence of 87.5% and 61.4% among TB infected patients and control groups, respectively which is higher than the present study [8]. This is might be an active antigen method was used to investigate the infection of *H. pylori* and a positive tests are evidence of a current infection and not the possibility of a previous infection, which could have been the case had a serological tests been used.

When we see the prevalence of *H. pylori* with respect to age, the present study showed that it increased with the age from 5/19 (26.3%) in the age groups of 18-34 years and 9/19 (47.4%) in age of greater or equal to 45 years among TB patients. However there was no statistically significant association. Similarly a study conducted in Brazil revealed that prevalence of *H. pylori* infection increased from age group of 18-30 years to 46-60 years old with (84.7%) and (92%), respectively but with no statistically significant association [14].

Prevalence of *H.pylori* in respect of gender also did not show statistically significant associations with this study even though males were with higher prevalence (COR = 2.63, 95% CI: 0.83-8.4, P = 0.098). However, this was not in agreement with the findings reported from Peru which showed that there was a significant association [15]. This difference might be due to the small sample size used in this study.

TB diseases might be associated with increased prevalence of *H. pylori* infection. To date, different studies on association between *H. pylori* infection and respiratory disease have been investigated. However, these studies concluded differently. For example study from Peruvian has reported that there is no association with *H. pylori* and TB disease [15, 16]. In contrast, study among Pakistan and Gambian reported being *H. pylori* infected may contribute to being less to develop active TB disease [17]. However, in the present study the prevalence of *H. pylori* infection was higher among TB patients than non TB controls. Increased prevalence of *H. pylori* infection might be due to the immune response resulting from *H. pylori* infection is indeed predominantly leading to chronic inflammatory reaction and consequent damage to the mucosal tissue that resulting in unbalance between the pro-inflammatory and anti-inflammatory cytokines produced to activate macrophages and cytotoxic T cells that act during *M. tuberculosis* infection to reduce bacterial proliferation and also to reduce tissue damage [18, 19].

Moreover, *H. pylori* infection may also contribute to enhanced mycobacterial survival and replication in the host by decreasing $Th_1$ cell responses and delays proliferation and recruitment of $CD_4^+$ effector cells to the site of infection. On the other hand, the role of chronic *H. pylori* infection as a predisposing factor to development of pulmonary TB is still unknown [17, 20].

In the present study, $CD_4^+$ lymphocyte count and HIV status were found to be associated with *H. pylori* infection among TB patients. The $CD_4^+$ lymphocyte count is an indicator of immune status of the host [21, 22]. Those individuals who have $CD_4^+$ lymphocyte count less than 200 cells/$mm^3$ were significantly associated with the presence of *H. pylori* antigen in stools of TB patients. TB patients who had low $CD_4^+$ lymphocyte counts were more susceptible to *H. pylori* infection.

Of the total study subjects, 11/54 (20.4%) was HIV positive. This is in agreement with reports on Federal HIV/AIDS prevention and control office indicating the prevalence of HIV in TB cases to be 20%, but this is not comparable with study conducted in East Wollega Zone, Western Ethiopia, that showed prevalence of 33.7% [23, 24].

In the present study among TB patients who were positive for HIV were 0.09 times less likely to be infected with *H. pylori* than TB patients who had been not infected with HIV (AOR= 0.09, 95% CI: 0.01 – 0.97, P = 0.048). This might indicate that those individuals who had positive for HIV in this study have been treated with antibiotics as prophylaxis and/or anti-retroviral therapy drugs which may have effects against *H. pylori,* this is a possible explanation for the low *H. pylori* infection among this group[25].

Other variables such as Age, marital status, occupational status, place of residence and the number of person per household were not statistically associated with *H. pylori* infection in this study. This result was not comparable with the study done in Brazil and Addis Ababa [14, 26]. The difference may be due to the difference in study settings and design.

## 5. Conclusion

The overall prevalence of *H. pylori* infection in TB positive patients and healthy controls was 19 (35.2%) and 11 (20.4%), respectively. In this study, *H. pylori* among TB patients were higher compared with the data for healthy controls from the same area. This suggests that the two infections are interrelated; means that the variability of *H. pylori* infection in the two groups is significantly different. Factors like $CD_4^+$ lymphocyte level and HIV status were significantly associated with *H. pylori* infection. However, those who were infected by HIV were less likely infected by *H. pylori.* For controlling of the spread of TB and its associated problems, diagnosing and treating of TB infected patients for *H. pylori* infection is recommended. Generally, a cohort study will reveal details of the dynamics of *H. pylori* infection in the studied population.

## Authors' Contributions

GA conceived the study; WK, develops the proposal, performed all the laboratory activities and developed the draft of the manuscript and BM, involved in data analysis and interpretation of the results. All authors were read and approved the final version of the manuscript.

## Acknowledgements

Our acknowledgement would like to University of Gondar for financial support. Our thanks were also for all staff members of Mycobacteriology research center of Jimma University for their material and technical support during all laboratory activities. Finally we would acknowledge all study participants for their participation in the study.

## References

[1] Anastasios R, Nikiforos P, Konstantinos IG: Helicobacter pylori infection and respiratory diseases: a review. *WJG* 2003, 9(1):5-8.

[2] Dorji D, Dendup T, Malaty HM, Wangchuk K, Yangzom D, Richter JM: Epidemiology of Helicobacter pylori in Bhutan: The Role of Environment and Geographic Location. *Helicobacter* 2013(Electronic 1523):53-75.

[3] Hunt RH, Xiao SD, Megraud F, Leon-Barua R, Bazzoli F, van der Merwe S, al. e: Helicobacter pylori in developing countries. *World Gastroenterology Organization Global Guidelines* 2010.

[4] Neumann WL, Coss E, Rugge M, Genta RM: Autoimmune atrophic gastritis--pathogenesis, pathology and management. *Nat Rev Gastroenterol Hepatol* 2013, 10(9):529-541.

[5] Shrestha S, Paudel P, Pradhan GB, Shrestha L, Bhattachan CL: Prevalence study of H. pylori infection in dyspeptic patients coming to Nepal Medical College Teaching Hospital, Jorpati, Kathmandu. *Nepal Med coll J* 2012, 14(3):229-233.

[6] Katayoon N, Masoud S, Saeed F MS, Masoud J: Helicobacter pylori seroprevalence in patients with Bronchiectasis. *Tanaffos* 2006, 5(3):25-29.

[7] www.ju.edu.et. Jimma University: 2013.

[8] Philippou N, Roussos A, Tsimpoukas F, Anastasakou E, Mavrea S, Tsimogianni A: Helicobacter pylori infection in patients with active pulmonary tuberculosis before the initiation of anti-tuberculosis treatment. *Anals of Gastroenterology* 2003, 16(1):49-52.

[9] Cheesbrough M: District Laboratory Practice in Tropical countries. United States of America New York: Cambridge University press; 2006.

[10] Salman H, Sabine RG: MGITprocedure manual prepared for the foundation for Inovatives New diagnostics 2006.

[11] Veijola L, Myllyluom E, Korpela R, Rautelin H: Stool antigen tests in the diagnosis of Helicobacter pylori infection before and after eradication therapy. *WJG* 2005, 11(46):7340-7344.

[12] Ashgar SS: Helicobacter Pylori Diagnosis by Stool Antigen ELISA and Rapid Test. *J Applied Medical Sciences* 2013, 2(4):61-66.

[13] Hosmer DW, Lemesshow S, Strurdivant RX: Appliedlogestic regression USA: Johan wiley and sons 2013.

[14] Maria N, Dulciene M, Rodrigo T, Andreia MC, Manuel B, Lucia L: Helicobacter pylori Infection in Adults from a Poor Urban Community in Northeastern Brazil: Demographic, Lifestyle and Environmental Factors. *The Brazilian J of infectious disease* 2005, 9(5):405-410.

[15] Torres MA, Passaro DJ, Watanabe J, Parsonnet J, Small P, Miyagui J, Rodriquez C, Astete M, Gilman RH: No association between Helicobacter pylori and Mycobacterium tuberculosis infections among gastrointestinal clinic attendees in Peru *Epidemiol Infect* 2003(130):87-91.

[16] Buskin SE, Weiss NS, Gale JL, Nolan CM: Tuberculosis in relation to a history of peptic ulcer disease and treatment of gastric hyperacidity. *American journal of epidemiology* 1995, 141(3):218-224.

[17] Pawlowski A, Jansson M, Sko¨ld M, Rottenberg ME, Kallenius G: Tuberculosis and HIV Co-Infection. *Plos pathog* 2012, 8(2):e1002464.

[18] Altamann DM, Sansom D, Marshi SG: What is the basis for HLA-DQ association with Authoimmune disease. *Immunology Today* 1991, 12(8):267-270.

[19] Goldfled AE, Delgado JC, Thim S, Bozona V, Uglialoro AM, Turbay D ea: Association of an HLA-DQ allele with Clinical Tuberculosis. *JMA* 1998, 279(3):226-228.

[20] Brighenti S, Andersson J: Local Immune Responses in Human Tuberculosis: Learning From the Site of Infection. *JID* 2012, 205(2):316-324.

[21] Affusim C, Abah V, Kesieme EB, Anyanwu K, EifediyiR: TheEffectofLowCD4+LymphocyteCounton theRadiographicPatternsofHIVPatientswithPulmonary TuberculosisamongNigerians. *Tuber Rese and Treat*2013, 4:1-4.

[22] Shams-Hosseini SN, Javad Mousavi SA, Kadivar M, Ahmadipour E, Yazdani R, Moradians V: Helicobacter Pylori in Patients Suffering from Pulmonary Disease. *Tanaffos* 2011 10(1):31-36.

[23] Kebede W, Keno F, Ewunetu T: Acceptance of Provider Initiated HIV Testing and Counseling among Tuberculosis Patients in East Wollega Administrative Zone, Oromia Regional State, Western Ethiopia. *Tuber Rese and Treat* 2014, 5:1-5.

[24] World health organization: Global tuberculosis report. 2012.

[25] Hestvik E, Tylleskar T, Ndeezi G, Grahnquist L, Olafsdottir E, Tumwine JK, et al: Prevalence ofHelicobacter pyloriin HIV-infected, HAART-naïve Ugandan children: a hospital-based survey. *Journal of the International AIDS Society* 2011, 14(34):1-9.

[26] Taddesse G, Desta K, Esayas S, Bane A: Association of dyspepsia symptoms and Helicobacter pylori infections in private higher clinic, Addis Ababa, Ethiopia. *EMJ* 2011, 49(2):109-116.