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Sero - Prevalence of *Helicobacter Pylori* in HIV Positive Patients and HIV Negative Controls in St. Paul's General Specialized Hospital, Addis Ababa, Ethiopia

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Abstract: Background. *Helicobacter pylori (H. pylori)* is recognized as a major human pathogen. Clinical symptoms associated with *H. pylori* infection have been reported in patients with human immunodeficiency virus (HIV). A high, normal, and a lower sero - prevalence of *H. pylori* infection in HIV positive patients than negative controls was found in different studies. The aim of this study was to assess the sero - prevalence of *H. pylori* infection in HIV positive patients and negative controls and determine the impact of CD4 cell count in patients with *H. pylori* in St. Paul's General Specialized Hospital in Addis Ababa, Ethiopia. Methods. A comparative cross sectional study was conducted in HIV positive patients and controls with gastrointestinal symptoms using Serology test kit (ACON® *H. pylori*, USA). All individuals who came to Voluntary Counselling and Testing and Anti - Retroviral Therapy to the Out Patient Department of the hospital were examined for complaints of dyspepsia and those with the complaint were tested for *H. pylori.* Results. Of the 106 HIV positive subjects, 68 (64.2%) were positive for anti - *H. pylori* IgG antibodies; and of the 106 HIV negative controls, 52 (49.1%) were positive for anti - *H. pylori* IgG antibodies (p=0.037). There was no significant difference of *H. pylori* seroprevalence between relatively higher and lower CD4 cell counts in the HIV positive cases (p>0.05). Conclusion. A significantly higher seroprevalence of *H. pylori* was demonstrated in the HIV positive subjects. There was no significant difference in the prevalence of *H. pylori* between different CD4+ cell counts in the HIV positive study group.

Keywords: *Helicobacter Pylori*, Sero - Prevalence, HIV, CD4 Count, Dyspepsia

1. Introduction

Helicobacter pylori represents one of the most common and medically prominent infections worldwide. Infection with this micro aerobic, gram negative bacterium has been established as an etiologic factor in the development of peptic ulcer disease. In addition, *H. pylori* infection has been associated firmly with the development of gastric neoplasia, including gastric adenocarcinomas and gastric mucosa - associated lymphoid tissue lymphomas [1].

H. pylori is a spiral bacterium with flagellae and a potent producer of urease. It is through the production of bicarbonate, by metabolizing the urea in gastric fluid, that the organism is able to survive in the low pH environment of the stomach. There it colonizes the gastric epithelial cells passing into the extra - cellular, mucous layer with its more pH - neutral conditions. There infection induces a host response which results in mucosal damage and a chronic active gastritis. This occurs initially in the non - acid secreting areas of the stomach in the antrum [2]. More than half of all humans are colonized in their stomachs by *H. pylori*. That carriage is nearly universal among adults in developing countries suggests that in earlier times most humans carried these organisms. However, it is not known whether *H. pylori* has been with all humans for hundreds of years, or for millions of years. In either case, with the changing conditions associated with improved socioeconomic status, *H. pylori* is disappearing in

industrialized countries and declining in prevalence in some developing countries as well [3].

Infection with *H. pylori* has been recognized as a public health problem worldwide. Previous sero - epidemiologic studies indicated that about 50% of adults in the developed countries and nearly 90% of adults in developing countries were sero - positive for *H. pylori* [4].

Recent studies in Ethiopia showed that the overall prevalence of *H. pylori* infection in adult dyspeptic patients, as found by the different diagnostic methods, varied between 69 and 91% [4].

The risk factors for acquiring *H. pylori* and HIV infections are different: H. pylori is transmitted by gastro - or fecal - oral routes and is associated with low socioeconomic conditions, while HIV is transmitted through sexual intercourse, infected body fluids, and transplacentally [5]. If the host responses to these infections were independent, the prevalence of *H. pylori* should be similar in HIV - infected and non - infected patients. Yet, several epidemiologic studies have generally reported conflicting results while some identifiable patterns can be discerned. It does appear that the incidence of *H. pylori* infection is lower among patients with AIDS compared to matched HIV - infected and - uninfected controls [6].

The bacterium is consistently reported with high prevalence in HIV - negative patients with chronic gastritis and active ulcer disease [7].

Different studies showed that severly immunocompromezed patients with dyspeptic symptoms have much lower prevalence of *H. pylori* and active chronic gastritis in the gastric antrum than in HIV - negative patients [8, 9] Furthermore, a substantial but insignificant decrease of *H. pylori* infection prevalence was noted in HIV patients with an extensive decline of CD4 cell count (< 100/µl). [10, 11]. In another studies, the prevalenceof *H. pylori* infection is not significantly different between HIV positive and HIV negative subjects [12].

In our knowledge, there is no study about the prevalence of *H. pylori* in HIV positive patients in Ethiopia and generally not enough in the general population. Therefore the aim of this study was to assess the sero - prevalence of *H. pylori* infection in HIV positive patients and HIV negative controls with upper gastro intestinal symptoms in St. Paul's General Specialized Hospital, Addis Ababa, Ethiopia.

2. Materials and Methods

2.1. Study Design, Area and Period

A comparative cross - sectional study was conducted in St. Paul's General Specialized Hospital, Addis Ababa, Ethiopia, during December 2010 and February 2011.

2.2. Study Population and Sampling Technique

In the study, those HIV positive patients with dyspepsia and HIV negative subjects with similar symptoms who came to Voluntary Counselling and Testing (VCT) were considered as study population.

2.3. Sample Size Determination

The sample size of the study was determined by the following formula:

$$n1 = \frac{[Z\alpha/2\sqrt{\bar{p}\bar{q}}(1 + 1/\lambda) + Z\beta\sqrt{p1q1 + p2q2/\lambda}]^2}{\Delta^2}$$

Where n2=n1 λ , $\bar{p}$ =(p1+ λp2)/(1+ λ)

Where, p1=0.84, p2=0.73, from previous studies (Alimohamed *et al*, 2002)

95% confidence interval and at 80% power, n1=97, n2=97 and the total minimum sample size calculated is 194 subjects.

2.4. Eligibility Criteria

2.4.1. Inclusion Criteria

After informed consent, all volunteer HIV sero - positive patients with dyspepsia and HIV negative controls with similar symptoms were included in this study.

2.4.2. Exclusion Criteria

Study participants who had received any antibiotic for the treatment of *H. pylori* and/or any other gastro intestinal infection in the previous two weeks were excluded from the study.

2.5. Data Collection Tools and Procedures

After informed consent, all volunteer HIV sero - positive patients with dyspepsia and HIV negative controls with similar symptoms were included in this study. First, all individuals who came to the VCT and ART clinics in the OPD of the hospital were examined for complaints of dyspepsia by internist, Public Health Officers and experienced nurses. All individuals with complaints were included in the study and were interviewed to fill the questionnaire. Socio - demographic and other appropriate information were collected using the questionnaire. The major contents of the questionnaire include age group, sex, economic status and previous antibiotic history of the study subjects.

The principal investigator and one assigned laboratory technologist with prior experience were engaged in the laboratory procedures. Two counsellors with prior experience were assigned. Then, after appropriate pre - test counselling and providing information about the study in the VCT centre of the Hospital, the blood which was drawn for the purpose of HIV testing (about 5ml of venous blood) was used also to test *H. pylori* infection of the individuals for the purpose of this study when they were volunteer to provide their blood for other study other than HIV testing.

Participants who were HIV negative after testing but still dyspeptic were considered as controls and their serum was tested for *H. pylori*.

After the study participants were categorized in to HIV negative and HIV positive, the HIV positive patients were

again classified depending on their CD4 count from recorded data and then determined the prevalence of *H. pylori* in different CD4 counts.

HIV testing was performed using the national HIV - l test algorithm according to the guideline and every information was identified by a code number. For the purpose of this study, serological status of *H. pylori* was tested with commercial *H. pylori* serology test kit (ACON® H. PYLORI, USA) following instruction of the manufacturer. The kit was used for the detection of *H. pylori* IgG antibodies in serum and it can detect anti *H. pylori* antibodies with sensitivity (93%) and specificity (89.2%). Data quality control was ensured through: Careful selection and training of data collectors, recruiting experienced laboratory personnel and counsellors, Positive and negative controls of serological test kits were checked before the actual sample is tested and Supervision in every step of data collection.

2.6. Data Entry and Analysis

Data from the questionnaire and the laboratory test results were entered using SPSS statistical package version 16. Results were summarized by frequency, percentages and means. X^2 test was used to compare the prevalence of *H. pylori* between HIV positive and HIV negative groups and HIV positive patients with higher and lower CD4+ counts. 95% confidence interval was used to measure the strength of association. A value of $P<0.05$ was regarded as statistically significant.

2.7. Ethical Considerations

Ethical approval was obtained from ethical committee of the department of Microbiology, Immunology, and Parasitology of the School of Medicine, Addis Ababa University, before the study. Permission was obtained from the Medical director, Laboratory and ART clinic heads of St. Paul's General Specialized Hospital after explaining the purpose and the procedure of the study.

Pre - and post - test counselling was provided for the study participants. Informed written consent from study subjects was also obtained before sample collection. Information about the study was given to the participants, including purposes and procedures, potential risk and benefits. Identification of the study subject was done only through numerical codes. Patients were treated according to the Helsinki declaration. *H. pylori* positive patients were treated by clarithromycin - based triple therapy, (Omeprazole 20 mg BID + clarithromycin 500 mg BID + amoxicillin 1 g BID x 10 days).

3. Results

The mean age of the study subjects was 33years with the range of 18 - 60 years. Majority of the subjects (78.3%) were in the age range of 18 - 40 years. Females constituted 64 % of the total study participants. Majority of the participants (71.7%) reported that they lived with more than two people in the same house. Only 2.8% of the study subjects were with monthly income of 1500 to 2499 birr. Among the illiterate participants (n=35), 60% of them were positive for *H. pylori* antibody (Table 1). 79% (89 of 112) of the total study subjects were unemployed. Of these 52.8% were *H.pylori* positive (Table 1). Only 10.7% (12 of 112) of the study groups were college graduates and the *H. pylori* positivity in this group was 33.3%.

The gender specific prevalence of *H. pylori* infection is the same in the two groups (56.8 % in males and 56.6% in females (Table 1).

Table 1. *Socio demographic characteristics of study participants in relation to H. pylori serostatus, in St. Paul's Hospital, Addis Ababa, Ethiopia.*

		***Helicobacter pylori* sero - status**	
Variable	**N**	***H. pylori* Positive n (%)**	***H. pylori* Negative n (%)**
Age			
<18	6	4 (66.7)	2 (33.3)
18 - 40	165	94 (57)	71 (43)
>40	41	23 (56.1)	18 (43.9)
Sex			
Male	74	42 (56.8)	32 (43.2)
Female	136	77 (56.6)	59 (43.4)
Marital status			
Single	58	38 (65.5)	20 (34.5)
Married	107	58 (54.2)	49 (45.8)
Divorced	32	18 (56.2)	14 (43.8)
Widowed	15	7 (46.7)	8 (53.3)
Occupation			
Government employed	22	12 (54.5)	10 (45.5)
Private employed	73	52 (71.2)	21 (28.8)
Unemployed	89	47 (52.8)	42 (47.2)
Others	28	10 (35.7)	18 (64.3)
Level of education			
Illiterate	35	21 (60)	14 (40)
Elementary	61	34 (55.7)	27 (44.3)
High school	104	62 (59.6)	42 (40.4)
College	12	4 (33.3)	8 (66.7)

		Helicobacter pylori sero - status	
Variable	**N**	***H. pylori* Positive n (%)**	***H. pylori* Negative n (%)**
Monthly income			
No income	18	10 (55.6)	8 (44.4)
Less than 500 birr	99	54 (54.5)	45 (45.5)
500 to 1499 birr	85	53 (62.4)	32 (37.6)
1500 to 2499	6	2 (33.3)	4 (66.7)
No of people living together			
None	14	4 (28.6)	10 (71.4)
One	20	12 (60)	8 (40)
Two	26	14 (53.8)	12 (46.2)
>two	152	91 (59.9)	61 (40.1)

Table 2. *Demographic data in HIV positive and HIV negative study groups, St Paul's Hospital, Addis Ababa.*

	HIV positive (n=106) (%)	**HIV negative (106) n (%)**
Age (years) [range]	**18 - 55**	**18 - 60**
Sex		
Male	32 (30.8)	42 (39.6)
Female	72 (69.2)	64 (60.4)
Marital status		
Single	24 (22.6)	34 (32.1)
Married	60 (56.6)	47 (44.3)
Divorced	14 (13.2)	18 (17)
Widowed	8 (7.5)	7 (6.6)
Occupation		
Government employed	16 (15.1)	6 (5.7)
Private employed	42 (39.6)	31 (29.2)
Unemployed	42 (39.6)	47 (44.3)
Others	6 (5.7)	22 (20.8)
Level of Education		
Illiterate	16 (15.1)	19 (17.9)
Elementary school	30 (28.3)	31 (29.2)
High school	56 (52.8)	48 (45.3)
College	4 (3.8)	8 (7.5)
No of people living together		
None	4 (3.8)	10 (9.4)
One	2 (1.9)	18 (17)
Two	20 (18.9)	6 (5.7)
More than two	80 (75.5)	72 (67.9)

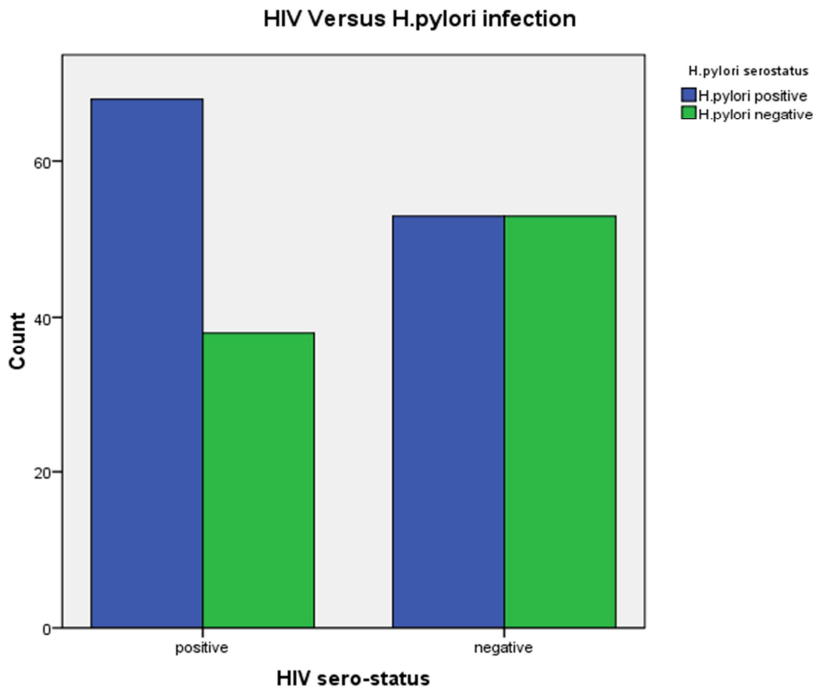

Figure 1. *H. pylori serostatus in HIV - infected and uninfected individuals, St. Paul's Hospital, Addis Ababa, Ethiopia.*

The overall prevalence of *H. pylori* infection in this study population was 57.08 % (120 / 212)

The mean age of the HIV - positive group was 34 years (range 18 - 55 versus 18 - 60) which was not statistically different from that of HIV - negative group. There were less male patients in the HIV positive group than in the HIV negative group (30% versus39.6%) (Table 2). Regarding marital status 56.6% of the HIV positive group was married while 44.3% were married in the HIV negative group. Government employed subjects were more in the HIV positive group than in the HIV negative group (15.1%, 5.7% respectively) (Table 2).

The prevalence of *H. pylori* infection in HIV negative group is significantly lower than in the HIV positive group. (68/106; 64.2% Vs 52/106; 49.1%, P= 0.037) (Table 3). Sixty eight of the 106 (64.2%) HIV positive subjects were *H. pylori* positive while fifty two of the 106 (49.1%) HIV negative were positive for *H. pylori*.

The HIV positive patients was further sub classified in to two groups according to their CD4+ lymphocyte count (mean=393.57/mm^3; median=406/mm^3 and range=693/mm^3).

Among the HIV positive patients there was no significant difference in the prevalence of *H. pylori* infection between CD4+ count less than 200 cells/ µl and CD4+ count greater than 200 cells/ µl. (p=0.493) (Table 4).

Table 3. *The relationship between Helicobacter pylori infection and HIV infection, St Paul's Hospital, Addis Ababa, Ethiopia.*

	HIV positive (n=106) %	HIV Negative (n=106) %	P - value
HP positive	68 (64.2)	52 (49.1)	0.037
HP Negative	38 (35.8)	54 (50.9)	

Table 4. *H. pylori infection and CD4+ count in HIV positive patients.*

		***H. pylori* sero-status**		**P-value**
		Positive	**Negative**	
$CD4^+$ count	$CD4^+ \leq 200/\mu l$ (n=38)	26 (68.4%)	12 (31.6%)	0.493
	$CD4^+ > 200/\mu l$ (n=68)	42 (61.8%)	26 (38.2%)	

4. Discussion

The aim of this study was to assess the sero - prevalence of *Helicobacter pylori* infection in HIV positive patients and HIV negative controls with dyspepsia and determine the impact of CD4 cell count in patients with *H. pylori* infection in St. Paul's General Specialized Hospital in Addis Ababa.

In the present study, it is found that the overall prevalence of *H. pylori* infection was 57%. This result is in agreement with previous studies which showed that the prevalence of *H. pylori* in dyspeptic patients was approximately 50 % [13] in the general population. But it also contradicted with some studies in the developing countries which showed high prevalence up to 90% [4]. This disagreement might be due to use of different methods for the diagnosis of *H.pylori* between the present study and the previous studies.

This study aimed to see if any difference was found in the sero - prevalence *H. pylori* between HIV positive patients and HIV negative controls.

The prevalence of anti - *H. pylori* IgG antibody in HIV positive patients in this study was significantly higher ($p < 0.05$) than that in the HIV negative control group (64.2% Vs 49.1%). Thus, it is indicated that HIV positive patients and HIV negative individuals have different susceptibility for the colonization *H.pylori* in gut. This contradicts with different previous studies in overseas [10, 11, 14, 15] that showed lower prevalence of *H. pylori* infection in HIV positive patients as compared to HIV negative controls. The reasons for such lower prevalence might be due to lack of CD4 cells, use of antibiotics and proton pump inhibitors and competitive inhibition by other pathogens. According to these studies $CD4^+$ lymphocytes were involved in the pathogenesis of *H. pylori* related gastritis and this gastritis might be a mechanism by which *H. pylori* colonization is enhanced. Therefore, fully functional $CD4^+$ lymphocytes might be required for *H. pylori* infection and *H. pylori* related peptic ulcer disease. However, our finding was in agreement with some previous studies [6]. The reason of higher prevalence of *H. pylori* in the HIV positive group in this study may be due to (i) hypochlorhydria sometimes associated with HIV infection.

Therefore the increased prevalence of *H. pylori* in HIV positive group of this study as compared to the HIV negative control group could be due to hypergammaglobulinemia since the sole diagnostic tool in this study was serological test to determine anti - *H. pylori* IgG. This may be due the fact that most HIV infected patients had IgG antibodies against other frequently encountered pathogens. (iii) Cell mediated immune deficiency in the HIV positive patients may also contribute to the high prevalence of *H. pylori* infection in this group. It is reported that Th1 and Th2 cells appear to predominantly present in the gastric mucosa of *H. pylori* positive subjects. Hence Th1 and Th2 cells play a central role in the immune regulation during *H. pylori* infection [17]. In HIV positive patients, there is a depletion of T helper cells and this may create favourable condition for the colonization and pathogenesis of *H.pylori* in the HIV positive group.

(iv)The mucosal surface of the gastro intestinal tract serves as a predominant structural and immunological barrier against microorganisms [17]. Thus, loss of the integrity of the mucosal surface such as, disruption of the tight epithelial

junctions may be the possible explanation for the high prevalence of *H. pylori* infection in HIV positive patients than in HIV negative controls in the present study.

In our study, the HIV positive patients were sub classified in to two groups based on their $CD4^+$ lymphocyte count to analyse the difference in sero - prevalence of *H. pylori* between the two groups. This sub analysis showed that no significant difference (p=0.493) in the prevalence of *H. pylori* infection in the two groups ($CD4^+$ < 200/µl and $CD4^+$ >200/µl). This result may be limited by the small number of subjects in each group. The result of this study on the impact of CD4 cell count in the prevalence of *H. pylori* infection in HIV positive patients was agreed with other previous studies in Italy and Greek [9, 11, 18]. Our study and the previous studies showed insignificant difference of *H. pylori* infection between low ($CD4^+$ < 200/µl) and high ($CD4^+$ >200/µl) CD4 cell counts. But this study was against studies conducted in Kenya, China and Brazil (12, 15, 19) which showed the prevalence of *H. pylori* was less in $CD4^+$ cell count less than 200/µl as compared to the prevalence of *H. pylori* in patients with CD4 cell count greater than 200/µl.

The low prevalence of *H. pylori* in patients with $CD4^+$ cell count less than 200/µl in the previous studies might be due to: in HIV positive patients with advanced AIDS stage, the frequent exposure to antimicrobials or histamine - 2 receptor antagonists may reduce *H. pylori* infection. But most of the HIV positive subjects in this study were not with advanced AIDS with mean CD4 count of 393.57/mm^3 and relatively free of frequent use of different antibiotics. Therefore according to results of this study decrease in absolute $CD4^+$ lymphocytes itself may not correlate with reduced *H. pylori* infection but may be due to the frequent exposure to antibiotics in HIV patients with advanced AIDS. This needs further study by considering the different clinical stages of AIDS.

5. Conclusions and Recommendations

From this study it is demonstrated that a higher prevalence of *H. pylori* infection in HIV sero positive patients than HIV negative control group with similar upper gastro intestinal symptoms. The HIV positive group showed a statistically significant higher sero - prevalence of *H. pylori* as compared to the HIV negative control group. Hypochlorhydria and depressed mucosal immune function may lead HIV positive patients to *H. pylori* colonization in the gut. It is also revealed insignificant difference of *H. pylori* prevalence in HIV positive patients with higher and lower CD4+ lymphocyte counts.

Based on our study the following recommendations were given.

- Consideration should be given for diagnostic evaluation of *H. pylori* in HIV patients.
- Large and well - designed studies must be conducted to determine the association between decreased acid secretion and *H. pylori* colonization in the gut.
- Researchers in future studies should control the potential confounder of antibiotic treatment in HIV positive patients beyond two week exclusion period.

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