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Helicobacter pylori Infection among Dyspeptic and Non-Dyspeptic HIV Patients at Yeka Health Center Addis Ababa, Ethiopia; Case Control Study

Getachew Seid[1*], **Kassu Desta**[2] **and Aster Tsegaye**[2]

[1]*Ethiopian Public Health Institute, Ethiopia*

[2]*Department of Medical Laboratory Science, Addis Ababa University, Ethiopia*

[*]**Corresponding author:** Getachew Seid, Ethiopian Public Health Institute, Ethiopia, Tel: +251912131707; E-mail: gech1365@gmail.com

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Abstract

Background: The exact role of *H. pylori* infection among HIV infected patients in causing gastrointestinal symptoms such as dyspepsia remains obscure. There are circumscribed data regarding *H. pylori* infection in HIV positive patients in cognation to $CD4^+$ T cell count in our city and country. Our study aims to assess the prevalence of *H. pylori* infection among dyspeptic and non -dyspeptic HIV patient and its association with $CD4^+$ T cell count at Yeka health center.

Methods: Institution based matched case control study was conducted from January to June 2017 on dyspeptic and non-dyspeptic HIV patients in Yeka health center. Cases were dyspeptic HIV patients who have symptom of dyspepsia and controls were non-dyspeptic HIV patients who have not symptom of dyspepsia. Stool antigen test was used to determine *H. pylori*. Data were managed using SPSS version 20.

Results: A total of 185 cases and 185 controls were participated in the study. In dyspeptic group gender distribution was 35.1% male: 64.9% female and 33% male: 67.0% female in the non-dyspeptic group. *H. pylori* antigen was detected in 117 (31.62%) of the total participants. The prevalence of *H. pylori* infection among cases and control was 60 (32.43%) and 57 (30.81%) respectively. The mean and median $CD4^+$ T cell count of dyspeptic study participants was (mean 370, median 364 cell/dl) while in the control group it was (mean 329,median312 cell/dl). In both case and control groups, no significant association was observed in the prevalence of *H. pylori* infection and $CD4^+$ T cell count (p-value 0.18).

Conclusion: The prevalence of *H. pylori* infection was not significantly different among dyspeptic and non-dyspeptic HIV patients. The infection was not significantly associated with $CD4^+$ T cell count both in the dyspeptic and non-dyspeptic HIV patients. Thus, screening and treatment of *H. pylori* infection should be done independent of $CD4^+$ T cell count level and symptom of dyspepsia.

Keywords: Dyspeptic; Non-dyspeptic; *H pylori*; Prevalence; Stool antigen test

Background

H. pylori is a spiral bacterium with flagella and a potent producer of urease. There the infection induces a host immune 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 [1].

It is clear that, among the several types of *H pylori*, only the carriers of the *Cag A* gene can induce the disease. So, only *CagA* and *VaC A* gene carriers could develop disease including ulcers. All the others genes combinations will not develop any disease and are asymptomatic [2].

The prevalence of *H. pylori* varies among continents and countries around the world, and its prevalence varies from 9% to 82% 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] however; persistent colonization depends on the host immune responses to the bacterium [4]. The infection is mainly acquired in childhood in both industrialized and developing countries, and persists throughout life unless treated [5].

HIV infected patients experience many forms of opportunistic infections, including gastrointestinal symptoms [6]. The exact role of *H. pylori* infection among HIV infected patients with gastro duodenal lesions might be different from the general population. It remains unclear if upper gastrointestinal symptoms such as dyspepsia are Highly Active Antiretroviral Therapy (HAART) related adverse effects as a result of *H. pylori* infection [7,8].

A previous study done on prevalence of *H. pylori* in association with HIV infection has suggested that cell mediated immune deficiency does not appear to increase the risk of infection with *H. pylori* [9]. Studies have shown that the prevalence of *H. pylori* infection in HIV positive patients is remarkably low compared with the general population [10]. The use of the stool antigen test is recommended for targeted high risk populations [11].

Some studies have indicated that the CD4/CD8 ratio in the gastric mucosa is different in subjects with and without *H. pylori* infection [12,13]. There for we aimed to assess the prevalence of *H. pylori*

infection utilizing stool antigen among dyspeptic and non -dyspeptic HIV patients and its relation with $CD4^+$ T cell count.

Materials and Methods

Institutional based case control study was conducted to assess the prevalence of *H. pylori* predicated on stool antigen detection among dyspeptic and non-dyspeptic HIV patients at Yeka health center, Addis Ababa, Ethiopia from January to June, 2017. All, dyspeptic and non-dyspeptic HIV patients, who were attending the ART clinic of Yeka health center within the study period and consummate the inclusion criteria were included in the study.

The sample size of the study was determined by two population proportion using the following formula:

$$n1 = \frac{(Z\alpha/2\sqrt{pq}\left(1 + 1/\lambda + Z\beta\sqrt{pq1q1} + \frac{p2q2}{\lambda}\right)2}{\Delta^2}$$

Where $n_2=n_1\,\lambda$, $\bar{p}=(p_1{}^+\,\lambda p_2)/(1^+\lambda)$

N1 for cases and n2 for controls

Where, p1=0.642, from previous studies [13] p2=0.50 (at the time of the study we cannot get any literature that show its prevalence so we use 50% prevalence of *H. pylori* in non-dyspeptic HIV patient) 95% confidence interval and at 80% power, n1=168, n2=168 so 168 cases and 168 control groups will be selected and including 10% contingency the total sample size will be 370 subjects. Convenience sampling technique was employed until the required sample size meet. We recruited them when they come to the clinic for CD4 count follow up by asking whether they had complain of dyspepsia or not. We use those who complain symptom of dyspepsia as case and those who do not complain symptom of dyspepsia as control.

Demographic data and potential risk factor of *H. pylori* infection were recorded.

We have collected 3 ml of blood venous blood for $CD4^+$ T cell count which was done by FACScount machine [14]; additionally we collected stool specimen on the same day; then the stool was processed for *H. pylori* stool antigen test.

All data quality control tools (pre-analytical, analytical and post-analytical stages) of quality assurance that were incorporated in to standard operating procedures (SOPs) of the serology and immune hematology laboratory were strictly followed.

Information from the laboratory analysis was cleaned, coded; data entry and analysis were done utilizing SPSS statistical software version 20. Descriptive statistics were employed to describe socio-demographic and clinical characteristics; adjusted odds ratio (ORs) with 95% confidence interval (CI) of positive replications to the different variables. Comparison between groups was done with Chi-square and P-value of <0.05 was considered consequential.

The study was conducted after it was ethically reviewed and approved by the Department of Research and Ethical Review Committee (DRERC) of Addis Ababa University and Addis Ababa Health Bureau. Apprised indicted consent was obtained from participants before data collected.

Results

Socio-demographic and clinical characteristics

A total of 370, study participants were enrolled in this study, 185 dyspeptic (cases) and 185 non-dyspeptics (controls), of which 126 (34.0%) were males and 244 (66.0%) females. Gender distribution was 35.1% male: 64.9% female in case group and 33% male: 67.0% female in the control group (p>0.05) (Table 1).

Variable	Dyspeptic n=185 (%)	Non-dyspeptic n=185 (%)	p-value
Age (y)			0.0631
18-30	51 (27.6)	68 (3.7)	
31-50	109 (58.9)	105 (56.8)	
>50	25 (13.5)	12 (6.5)	
Gender			0.898
Male	65 (35.1)	61 (33.0)	
Female	120 (64.9)	124 (67.0)	
Marital status			0.143
Single	35 (18.9)	45 (24.3)	
Married	124 (67.0)	127 (68.6)	
Divorced	11 (5.9)	9 (4.8)	
Widowed	15 (8.1)	4 (2.2)	
Occupation			0.177
Unemployed	61 (33.0)	56 (30.2)	
Government employed	38 (20.5)	40 (21.6)	
Private employed	84 (45.4)	79 (42.7)	
Other	2 (1.0)	10 (5.4)	
Education			0.322
Illiterate	16 (8.6)	12 (6.5)	
Elementary	78 (42.1)	85 (45.9)	
High school	63 (34.1)	51 (27.6)	
College and above	28 (15.1)	37 (20.0)	
Monthly income			0.272
No	3 (1.6)	7 (3.7)	
<1000 birr	79 (42.7)	82 (44.3)	
1000 to 2000 birr	92 (49.7)	83 (44.8)	
2001 to 3000 birr	9 (4.8)	4 (2.1)	
>3000 birr	2 (1.0)	9 (4.8)	

Table 1: Demographic characteristics of *H. pylori* infection among HIV-1 Patients attending at Yeka health center, Addis Ababa, 2017

Prevalence of *H. pylori* infection

The overall prevalence of *H. pylori* infection was 31.62 % (n=117); the gender categorical prevalence of *H. pylori* infection was 37.3% (n=47) in males and 28.7% (n=70) in Females. The *H. pylori* prevalence was high at the age group of above 50 years (35.1% and n=13).

The prevalence of *H. pylori* in the dyspeptic study group was 32.4% (n=60). Among *H. pylori* positives, the majority were in the females (66.7%, n=40), espoused (68.3%, n=41), private employed (48.3, n=29), and at elementary education level (46.7%, n=28).

The prevalence of *H. pylori* among the non-dyspeptic HIV patient was 30.81% (n=57). Among the *H. pylori* positives, the majority were in the females (52.6%, n=30), married (70.2%, n=40), private employed (47.4%, n=27), and at elementary education level (42.1%, n=24).

The prevalence of *H. pylori* infection in control HIV patient group and case HIV positive patient group is not statistically different (57/185; 30.8% *vs.* 60/185; 32.4%, P>0.05) respectively (Table 2).

	Dyspepsia		**COR (95%CI)**	***P* value**
	Yes	No		
***H. pylori* status**	n (%)	n (%)		
Positive	60 (16.2)	57 (15.4)	0.952 (0.745-1.217)	0.697
Negative	125 (33.8)	128 (34.6)	1.044 (0.838-1.302)	0.276
COR: Crude Odd Ratio, CI: Confidence Interval				

Table 2: Associations of *H. pylori* prevalence among dyspeptic and non-dyspeptic HIV patients at Yeka public health center, Addis Ababa, 2017

As displayed in Table 3, potential risk factors for *H. pylori* infection in the case group and the control group were analysed. However, none of them showed a significant association (Table 3).

Variable	**N**	**Dyspeptic (n=185)**				**Non-dyspeptic n=185 (%)**				
		H. pylori positive (n=60)	*H. pylori* negative (n=125)	X2	p-value	N	*H. pylori* positive n=57 (%)	*H. pylori* negative n=128(%)	X2	p-value
		n (%)	n (%)							
Siblings				2.037	0.916				10.435	0.107
None	8	4 (6.6)	4 (3.2)			16	5 (8.7)	11 (8.6)		
One	22	7 (11.6)	15 (12.0)			31	8 (14.1)	23 (18.0)		
Two	57	18 (30.0)	39 (31.2)			50	12 (21.1)	38 (29.7)		
Greater than two	98	31 (51.6)	67 (53.6)			88	32 (56.1)	56 (43.7)		
Alcohol consumption				0.777	0.378				2.488	0.115
Yes	57	16 (26.6)	41 (32.8)			43	18 (31.6)	25 (19.5)		
No	128	44 (74.4)	84 (67.2)			142	39 (68.4)	103 (80.5)		
Source of drinking water				-	-				-	-
Tap	185	60 (100)	125 (100)			185	57 (100)	128 (100)		
Well	0	0	0			0	0	0		
Cigarette smoking				0.984	0.321				1.85	0.173
Yes	3	1 (1.7)	2 (1.6)			3	1 (1.7)	2 (1.5)		
No	182	59 (98.3)	123 (98.4)			182	56 (98.3)	126 (98.5)		

Table 3: The association of risk factors and *H. pylori* status among dyspeptic and non –dyspeptic HIV patients at Yeka health center, Addis Ababa, 2017

H. pylori infection and CD4$^+$ T cell count

CD4$^+$ T-cells count had a median level of 364 cells/μl among Dyspeptic study participants (case) and 312 cells/μl in non-dyspeptic study group (control). Among the study participants, there was no significant difference in the prevalence of *H. pylori* infection between CD4$^+$ T cell count less than 200 cells/μl, 200 cells/μl to 500 cells/μl and greater than 500 cells/ μl (n=40; 40%, n=56; 29.62% and n=21; 25.92%, p-value 0.129 and COR 0.001) (Table 4).

CD4 T cell count	Dyspeptic (N=185)				Non-dyspeptic (N=185)			
	N	*H. pylori* positive n=60 (%)	*H. pylori* negative n=125(%)	p=0.320 X2 =14.00	N	*H. pylori* positive n=57(%)	*H. pylori* negative n=128	p=0.47 X2 = 6.0559
<200	46 (24.9)	16 (8.6)	30 (16.21)		54 (29.2)	24 (12.97)	30(16.21)	
200-500	89 (48.1)	30 (16.21)	59 (31.89)		100 (54.0)	26 (14.05)	74(40.0)	
>500	50 (27.0)	14 (7.56)	36 (19.45)		31 (16.8)	7 (3.78)	24(12.97)	

Table 4: $CD4^+$ T cell count and *H. pylori* status among dyspeptic and non-dyspeptic HIV patients at Yeka health center 2017

Discussion

Our primary group of study consisted of 185 dyspeptic HIV-infected patients (cases), to which we paired a second group composed by 185 non-dyspeptic HIV-infected subjects (controls). In our study, *H. pylori* infection registered a frequency of 30.8% among control group which was remotely lower than case group, 32.4%, but not enough to provide a statistically significant sodality between having dyspepsia and the presence of *H. pylori* (p=0.679, COR=0.81). This was akin to the conclusion of Ankouane et al. [15], but different from others [16,17]. But it dissents with antecedent study, in the general population *H. pylori* prevalence was (65%) patients with NUD and in (56%) asymptomatic controls [18]. The ground for such lower prevalence in this study might be due to the method difference that the studies were done; our study was stool antigen test and the anterior study was predicated on endoscopy.

The absence of a statistically consequential dispute regarding the frequency of *H. pylori* infection in dyspeptic HIV patients and non-dyspeptic HIV positive patients designate the desideratum for identifying other causes for dyspepsia in HIV-infected persons including fungal and viral digestive infections [1] to medication unpropitious effects [13], as some author Ether et al. state that more than a majority of all HIV-positive individuals repine of gastro-intestinal symptoms [19].

Our results showed low prevalence of *H. pylori* infection (31.6%) in HIV patients which differs from that anteriorly reported study for HIV-positive adults in Nigeria (46.8%) by Anejo-Okopi et al. [6]. This difference may be due to geographical and life habit difference of the study subjects. We observed homogeneous prevalence against the earlier reported study in Brazil 36.7% [20]. Tadege et al. [4] designated that the lower prevalence of *H. pylori* infection in HIV-positive patients is due to the suppressed immune response. Medications such as protein pump inhibiters and great attention to eradicate *H. pylori* infection by the medicals may result in decremented infection rate of *H. pylori* [4]. Other opportunistic infections such as cytomegalovirus may emerge to compete with *H. pylori* when secretion of gastric acids decreases. This may additionally cause inopportune environment for colonization of *H. pylori* [4]. Similarly Birhanu et al. have reported different prevalent rate utilizing other method like *H. pylori* IgG antibodies in St Paul's Hospital, Ethiopia (57.08%) [21,22].

Our study showed that high proportion (35.1%) of participants aged>50 y were infected with *H. pylori* as compared to younger age categories, and this was commensurable with earlier study findings in Nigeria [6], and Iran [4]. Albeit more immensely colossal percentage of women had participated compared to men; the study observed high proportions of *H. pylori* infection among women with no consequential risk of sodality.

In both case and control group we did not found a significant association with age group, marital status ,income, education, sibling, alcohol consumption, cigarettes smoking, khat chewing, house accommodations and water source (p>0.05) and a similar finding was reported from Malaty and Graham [5].

In our report in both case and control group, an incrimination in stool antigen prevalence of *H. pylori* is observed by incrementing the $CD4^+$ T cell count which did not reach statistical significant. The study concurred with other investigations [23]. This might be caused by the extortionate administration of antibiotics in HIV-positive patients to control the infectious complications. The cause, for the sodality between immune competence and *H. pylori* prevalence are still obscure, albeit several hypotheses have been offered [4]. The most popular hypotheses is that, in HIV patients with advanced disease stages there were more frequent bacterial infections, lead to antibiotic treatment courses which lead to unintended *H. pylori* eradication [4].

Conclusion

The overall stool antigen prevalence of *H. pylori* among HIV patients at the Yeka Health center was 31.63%. There was no significant difference in the detection of *H. pylori* stool antigen in dyspeptic patients and in the non-dyspeptics i.e.32.4 % versus 30.8%. This suggests the desideratum to investigate alternative etiologies for dyspepsia in HIV-positive patients, besides *H. pylori* infection.

List of Abbreviation

AIDS: Acquired Immune Deficiency Syndrome; ART: Anti-Retroviral Therapy; CDC: Center for Disease Control; CD3: Cluster of Differentiation 3; CD4: Cluster of Differentiation 4; CD8: Cluster of Differentiation 8; CI: Confidence Interval; CLSI: Clinical Laboratory Standard Institution; HIV: Human Immune Deficiency Virus; *H pylori*: *Helicobacter pylori*; HpSA: *H. pylori* Stool Antigen Test.

Ethics Approval and Consent to Participate

Ethical approval has taken from Addis Ababa University and Addis Ababa health bureau as well as written consent has taken from the participant.

Availability of Data Materials

The data will avail in the journal repository.

Competing Interests

The authors declare that there is no conflict of interest with regard to this research work and writing the article.

Consent for Publication

Not applicable

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