ORIGINAL ARTICLE

Seroprevalence of helicobacter pylori in human immunodeficiency virus-positive Patients and it's correlation with CD4+ Lymphocyte Count

Alireza Abdollahi[1], Saeed Shoar[1,2], Siroos Jafari[3], Hamid Emadi-Kochak[3]

[1]Associate Professor of Pathology, Central Laboratory, [2]Laboratory Assistant, Student Scientific Research Center, Central Laboratory, [3]Associate Professor of Infectious Diseases, Infectious Diseases Department, Imam Hospital Complex, Tehran University of Medical Sciences, Tehran, Iran

Address for correspondence:
Dr. Alireza Abdollahi, Associate Professor of Pathology, Division of Pathology, Imam Hospital Complex, Tehran University of Medical Sciences, Tehran, Iran.
E-mail: dr_p_abdollahi@yahoo.com

ABSTRACT

Background: This study assessed the seroprevalence of *Helicobacter pylori* antibodies among Iranian patients with human immunodeficiency virus (HIV) infection. It also examines whether anti *H. pylori* seroprevalence was associated with the severity of the HIV infection or the antiretroviral treatment. **Material and Methods:** A total of 114 HIV-infected patients and 114 age and sex-matched controls, without symptoms referable to upper gastrointestinal tract were recruited. Blood samples were obtained from all subjects. Serum IgG and IgA against *H. pylori* measured using the enzyme-linked immunosorbent assay (ELISA). **Results:** The rate of anti *H. pylori* IgG seropositivity was 57.9% in HIV-infected patients and 28.95% in controls ($P < 0.001$), while the rate of IgA seropositivity was 2.64% in HIV patients and 31.57% in controls ($P < 0.001$). Although there was an increasing trend of higher IgG and IgA titre by increasing CD4 cell count in HIV-positive patients, it was not reach statistical significance. There was no statistical difference in the serology of anti *H. pylori* IgG and IgA between patients receiving antiretroviral therapy comparing untreated HIV patients. **Conclusions:** This study showed higher seroprevalence of *H. pylori* IgG along with lower seroprevalence of *H. pylori* IgA in HIV-positive patients compared matched controls.

Key words: *H. pylori*, HIV, Seroprevalence, IgG, IgA

INTRODUCTION

Patients with human immunodeficiency virus (HIV) are susceptible to many different gastrointestinal (GI) opportunistic infections. HIV infection increase the colonisation of pathogens in GI tract.[1-3]

Helicobacter pylori, a gastric flagellate Gram-negative rod bacterium, is considered as the major aetiology of chronic gastritis and peptic ulcer disease.[4] Over half of the worlds' population is estimated to be infected with *H. pylori*.[5-7] The prevalence of *H. pylori* infection vary across different geographical regions with the range of 32% and 65%.[8-12] The infection has been seen in more than 90% of the patients with gastritis[13-15] and 70% to 100% of those with peptic ulcer diseases.[6,10,11] *H. pylori* is also known to have carcinogenic effects[16], and National Institute of Health Consensus Development Conference Statement recommends to eradicate this bacterium (if confirmed) in any cases of peptic ulcer.[17]

Access this article online	
Quick Response Code:	Website: www.nigeriamedj.com
	DOI: 10.4103/0300-1652.128176

The overall prevalence of *H. pylori* is suggested to be correlated with socioeconomic conditions. Gender, occupation, low socioeconomic status, educational level, and alcohol consumption are known risk factors for *H. pylori* infection.[15] However; persistent colonisation depends on the host immune responses to the bacterium.

Although some studies have shown that *H. pylori* infection is less common among HIV-positive individuals with GI symptoms[12,18-22], other investigations[23-25] suggested a higher prevalence of *H. pylori* infection in HIV-positive patients as a result of immune suppression. Hence, the relationship between *H. pylori* infection and HIV remain controversial.[26-27]

Moreover, there are limited data regarding the seroprevalence of *H. pylori* in HIV-positive patients, particularly in our region. This study aims to assess the

seroprevalence of *H. pylori* infection among HIV-positive patients and its correlation with $CD4^+$ cell count and some of hematological parameters. It also examines whether *H. pylori* seroprevalence is related with severity of HIV infection, or advanced stage of the disease and the administered antiretroviral regimens.

MATERIALS AND METHODS

A case- control study was carried out at the center of high-risk behavioural disease in Imam Hospital Complex, a major referral hospital in Tehran, capital of Iran, affiliated to Tehran University of Medical Sciences (TUMS). One-hundred and fourteen patients already diagnosed as HIV-positive cases attended our center, between January 2010 and June 2011, were consecutively enrolled in this study. Controls were subjects with negative test result for HIV, and recruited from the same centre. Controls were individually matched to cases with respect to sex and age (± 2 years) and where possible socio-economic status. None of the HIV-positive patients and controls had symptoms referable to upper GI tract. Subjects with a history of autoimmune diseases, malignancies such as lymphoma, peptic ulcer disease, documented diagnosis of viral infections within the past month, those who had received corticosteroid, antibiotics within the past 4 weeks, and subjects were previously treated for *H. pylori* infection were excluded.

The study was approved by Research Ethic Committee of TUMS and informed consent was obtained from each patient before enrollment in this study. Diagnosis of HIV infection confirmed with serology, polymerase chain reaction (PCR) or Western blot following the recommendation of National AIDS Control Organization (NACO 2007).

Five millilitres clotted blood and 3 cc anticoagulated blood with EDTA were obtained from subjects. The clotted blood was centrifuged in 3000 g for 15 minutes. Extracted serum was then stored in a -70 centigrade Celsius freezer. Ig A and Ig G anti-*H. pylori* antibody titretitre was measured by ELISA techniques in room temperature using Mono bind Inc, Lake Forest, CA, USA kit. Following the instruction provided by the manufacturer, values upper than 20 µ/ml were considered positive. Anticoagulated blood was also assessed in terms of $CD4^+$ and $CD8^+$ lymphocytes cell count by flow cytometry device (FCM) (PARTEC, Japan).

Determination of AIDS status was done according to the guidelines of World Health Organization (WHO). HIV patients with CD4 count below 200 cells/ml or specific clinical conditions suggestive of advanced immunodeficiency infection were categorised as AIDS stage.

Data were analyzed using Statistical Package for Social Sciences (SPSS version 18, Chicago, Inc). Data were presented as mean ± standard deviation (SD). Anti-*H. pylori* Immunoglobulin titres were expressed as Median and interquartile ranges (25^{th}–75^{th} centile). Chi-square test was employed to compare the seroprevalence of *H. pylori* infection between groups. Students't test and one-way ANOVA test were employed to declare the trend in each parameter between different groups. The distribution of anti-*H. pylori* IgG was shown using boxplot graph. *P*-value less than 0.05 was considered statistically significant.

RESULTS

A total of 114 HIV-positive patients and 114 controls were included in this study. Values of anti-*H. pylori* IgG and IgA anti bodies higher than the cut-off level of 20 u/ml were considered seropositive. The demographics and seroprevalence of anti-*H. pylori* Ig subclasses among HIV patients and controls were shown in Table 1. There were no significant difference regarding the age, gender, residence, and educational status between HIV patients and controls.

IgG antibody titre was positive in 66 HIV-positive patients (57.9%) which revealed a statistically significant difference ($P<0.0001$) when compared with 33 HIV-negative patients (28.95%) [Table 1]. On the other hand, IgA titre was positive in 3 HIV-positive patients (2.64%) compared to 36 HIV-negative patients (31.57%) which revealed a significant difference statistically ($P<0.0001$).

In HIV-positive patients, mean corpuscular volume (MCV), mean corpuscular haemoglobin (MCH), and IgG titre had a significant relationship with *H. pylori* seropositivity ($P<0.05$). In contrast, other haematological parameters did not reveal a statistically significant difference [Table 2].

None of the HIV-positive patients showed clinical symptoms suggestive of advanced immunodeficiency infection. Hence,

Table 1: Demographic characteristics and seroprevalence of anti-*H. pylori* Immunoglobulin subclasses HIV patients and controls

	HIV positive (n = 114) %	HIV negative (n = 114) %	*P* value
Age	37.5±3.3	36.8±2.7	0.081
Male/ Female	99/15	101/13	0.687
Urban/ Rural	86/28	92/22	0.337
Educational status (%)			
Illiterate	6 (5.3)	4 (3.5)	
Up to high school	25 (21.9)	18 (15.8)	
Diploma	48 (42.1)	53 (46.5)	0.617
Higher levels	24 (21.1)	30 (26.3)	
Unknown	11 (9.6)	9 (7.9)	
CD_4+ Count (cells/µl)	293.9 ± 187.6	981.4±261.2	<0.001
Anti-H. *pylori* IgG titre	25.0 (9.6-84.0)	17.0 (14.0-24.0)	<0.001
IgG seropositivity	66 (57.9)	32 (28.1)	<0.001
Anti-H. pylori Ig A titre	4.7 (3.5-8.2)	17.0 (14.7-29.0)	<0.001
IgA seropositivity	3 (2.64)	36 (31.58)	<0.001
IgG and IgA seropositivity	2 (1.75)	9 (7.89)	0.031

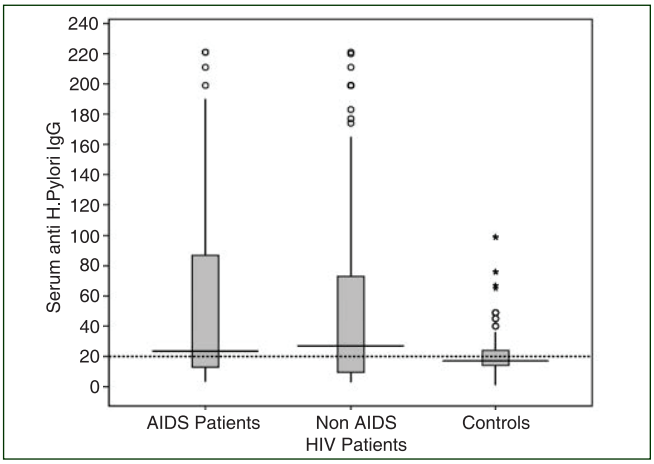

Figure1: Distribution of serum anti-*H. pylori* IgG in AIDS, non-AIDS HIV patients, and controls. The boxplot shows the median, interqaurtile range, minimum, maximum and outliers of serum anti-*H. pylori* IgG

staging of AIDS was done according to the values of $CD4^+$ count <200 cells/µl. Among 114 HIV patients, 37 were classified in AIDS stage. Figure 1 represents the distribution of serum anti-*H. pylori* IgG in AIDS, non-AIDS HIV patients and controls.

Of 114 HIV-positive patients, 79 (69.3%) were receiving antiretroviral therapy (ART). There were no statistical differences in the positive serology of anti-*H. pylori* IgG and IgA between patients undergone ART comparing untreated HIV patients (54.4% vs. 65.7%, $P = 0.26$ and 1.3% vs. 5.7%, $P = 0.17$).

To examine whether the severity of the HIV infection was associated with anti-*H. pylori* seroprevalence, HIV patients were classified into three groups based on their $CD4^+$ cell count accordingly; $CD4^+ < 200$, $200 \geq CD4^+ < 500$, and $CD4^+ \geq 500$. Serum anti-*H. pylori* IgG and IgA seroprevalence were compared between these groups [Table 3]. Although there was an increasing trend of higher IgG and IgA titre by increasing CD4 cell count in HIV-positive patients, it was not reach statistical significance.

DISCUSSION

HIV infection is associated with different GI opportunistic infections, including cytomegalovirus (CMV), cryptosporidium, microsporidia and fungal oesophagitis.[1,3] Whether HIV/AIDS infection has an impact on altering *H. pylori* prevalence remained controversial. Prevalence of *H. pylori* in HIV-positive patients has been reported in several studies[18,19,21,22,25,28-31], either in eastern world with higher prevalence[22,28,29] of *H. pylori* infection or in the tropical countries with lower prevalence.[23-25] This variation would apparently be due to the differences in sanitation, educational level, age, life styles, and socioeconomic status of different cultures which have been shown to be of major impacts in colonisation of *H. pylori* and subsequent infection.[32,33]

Table 2: Relationship of *H. pylori* seropositivity with $CD4^+$ cell count, hematological parameters and some other biochemistry in HIV-positive patients

Hematological parameter	IgG seropositivity	*N*	Mean ± SD	*P*-value
CD_4	No	48	270.06±167.14	0.281
	Yes	66	308.98±204.02	
CD8	No	48	766.62±377.50	0.885
	Yes	66	777.15±385.46	
WBC	No	48	5.03±1.65	0.061
	Yes	66	5.74±2.18	
Haemoglobin	No	48	14.44±1.86	0.131
	Yes	66	13.86±2.12	
Haematocrit	No	48	40.89±4.68	0.374
	Yes	66	40.01±5.45	
MCV	No	48	98.00±12.98	0.023
	Yes	66	92.43±12.48	
MCH	No	48	34.57±5.44	0.017
	Yes	66	32.16±5.14	
MCHC	No	48	35.18±1.41	0.094
	Yes	66	34.69±1.58	
Platelet	No	48	199.83±75.13	0.932
	Yes	66	201.12±82.84	
Eosinophil	No	48	193.33±155.25	0.582
	Yes	66	177.27±151.81	
Basophil	No	48	24.58±11.48	0.618
	Yes	66	26.06±17.96	
MG	No	48	2.89±1.05	0.155
	Yes	66	2.64±0.86	
ZN	No	48	147.42±44.47	0.721
	Yes	66	150.71±51.16	

Present study found higher seroprevalence of anti-*H. pylori* IgG in HIV patients compared to controls. The presence of anti-*H. pylori*-specific IgG is considered as a marker of chronic infection with this pathogen. In this study, we tried to match the cases and controls regarding the gender, age, residence and educational level status to minimise the effect of socioeconomic status on our results. This study also categorised HIV patients into AIDS and non-AIDS stages based on the CD4 cell count, and found no significant difference in serum anti-*H. pylori* IgG distribution between two groups.

In contrast, Moges *et al.*[28] was reported lower prevalence of *H. pylori* infection (19.6%) in Ethiopian HIV-positive population compared to HIV-negative dyspeptic patients (80.4%).

Studies by Hong-bin *et al*. and Lv *et al* were[34] reported a strong relationship between *H. pylori* infection and the stage of HIV from asymptomatic to the AIDS stage and indicated that the lower prevalence of *H. pylori* infection in HIV-positive patients is due to the suppressed immune response. They used upper endoscopy and gastric mucosa biopsy to confirm *H. pylori* infection. This is of great importance to remember that *H. pylori* infection

Table 3: Anti-*H. pylori* seropositivity according different categories of CD4 cell count in HIV-positive patients

	CD4+ count (cells/μl)			Trend *P*-value
	< 200 (cells/μl) (*n* = 37) %	200- 500 (cells/μl) (*n* = 59) %	≥ 500(cells/μl) (*n* = 18) %	
Anti-*H. pylori* IgG titre	23.60 (11.65-96.5)	23.00 (6.95-68.00)	30.50 (11.62-83.75)	0.581
IgG seropositivity	22 (59.5)	31 (52.5)	13 (72.2)	0.325
Anti-*H. pylori* IgA titre	4.50 (3.40-7.70)	4.60 (2.65-4.60)	6.85 (3.52-8.90)	0.181
IgA seropositivity	0 (0)	1 (1.7)	2 (11.1)	0.042

is confirmed by endoscopic studies and urease breathe test. Fialho *et al*.[30] was evaluated the *H. pylori* status of HIV patients with dyspepsia by urease test and histology. They demonstrate lower prevalence of *H. pylori* in these patients compared with symptomatic controls. As the symptoms of dyspepsia are more common among HIV-positive population, taking medications such as PPIs and more attempts to eradicate *H. pylori* infection by the physicians may result in decreased infection rate of such microorganism.[29] Decreased secretion of gastric acids has been accounted as another explanation[34]; hence, other opportunistic infections such as CMV may emerge to compete with *H. pylori*. This in addition to the decreased acid secretion may lead to inappropriate environment for colonisation of *H. pylori*.[22-25,35-37] Other studies proposed a different role of *H. pylori* in peptic ulcerogenesis, and chronic active gastritis in HIV-positive patients.[29]

Interestingly, in this study there was a significant difference in the seroprevalence of different subclasses of Ig between HIV-positive and negative patients. HIV-positive patients had a higher rate of IgG seropositivity and a lower rate of IgA seropositivity compared to controls. It seemed like HIV infection had increased the level of IgG titre while it had decreased the level of IgA titre. Decreased gastric colonisation of *H. pylori* may be an explanation for this.[30] However, it is not clear why level of IgG titre does not decrease in proportion to the decreased IgA level. Dysregulation of humoral and cellular immunity in HIV-positive patients may be a possible explanation for this pattern.[38] While alteration in activity of humoral response may lead to a decrease in secretion of anti-*H. pylori* antibodies such as IgA, abnormal production of nonspecific polyclonal antibodies may explain the increased level of anti-*H. pylori* IgG antibodies. Previous studies have indicated that HIV infection by compromising the status of cellular immunity may result in decreased serum level of antibodies. However, this finding has been only shown in one study and in IgG subclass.[28] Although it seems rational that HIV infection suppresses the ability of antibody production, our results demonstrated a different pattern. In contrast to the previous studies reporting lower seroprevalence of anti-*H. pylori* IgG, our study showed that IgG titre is higher in HIV-positive patients while the level of serum IgA is lower. It has been stated that serum levels of IgG and IgA are sensitive tests for *H. pylori* infection.[39] Although it has been suggested that the positive cut off should be adjusted with age, this factor does not account in our results as controls were matched with cases regarding sex and age.

Haematological parameters were also compared between *H. pylori* seropositive and seronegative cases in HIV-positive group. Except MCV, MCH, no significant differences in other parameters were observed between *H. pylori* seronegative and seropositive HIV patients. There was no single study in the literature to be compared with this finding.

In the present study, an increase in seroprevalence of *H. pylori* is observed by increasing the CD4+ cell count which did not reach statistical significance. In contrast, Some investigations have shown a significant relationship between $CD4^+$ cell count and *H. pylori* infection.[28,29,31,34] This could be explained due to the excessive administration of antibiotics in HIV-positive patients to control the infectious complications.[31]

The diagnosis of *H. pylori* infection in our study was not possible due to lack of endoscopic studies and other gold standard methods. Other studies have performed endoscopic biopsy or urease breath test to confirm the diagnosis of this infection.[9,15,25,29-31] This is one of the limitations of our study as Fabris *et al.* have strongly alarmed about interpretation of anti-*H. pylori* IgG titre in HIV-positive patients.[40]

Future studies should evaluate the prevalence of *H. pylori* in HIV-positive patients using endoscopic studies and with special attention to the stages of HIV, administration of anti retroviral regimens and histological findings.

CONCLUSIONS

This study showed higher seroprevalence of *H. pylori* IgG along with lower seroprevalence of *H. pylori* IgA in HIV-positive patients compared to HIV-negative subjects.

ACKNOWLEDGMENT

This study was financially supported by Research Deputy of Tehran University of Medical Science. The authors declare that there is no conflict of interests.

REFERENCES

1. Francis ND, Boylston AW, Roberts AH, Parkin JM, Pinching AJ. Cytomegalovirus infection in gastrointestinal tracts of patients infected with HIV-1 or AIDS. J Clin Pathol 1989;42:1055-64.
2. Dieterich DT, Wilcox CM. Diagnosis and treatment of esophageal diseases associated with HIV infection. Practice Parameters Committee of the American College of Gastroenterology. Am J Gastroenterol 1996;91:2265-9.
3. Romanelli F, Smith KM, Murphy BS. Does HIV infection alter the incidence or pathology of Helicobacter pylori infection? AIDS Patient Care STDS 2007;21:908-19.
4. Howden CW. Clinical expressions of Helicobacter pylori infection. Am J Med 1996;100:27S-32S; discussion 32S-34S.
5. Montecucco C, Rappuoli R. Living dangerously: How Helicobacter pylori survives in the human stomach. Nat Rev Mol Cell Biol 2001;2:457-66.
6. Carrick J, Lee A, Hazell S, Ralston M, Daskalopoulos G. Campylobacter pylori, duodenal ulcer, and gastric metaplasia: Possible role of functional heterotopic tissue in ulcerogenesis. Gut 1989;30:790-7.
7. Suzuki H, Hibi T, Marshall BJ. Helicobacter pylori: Present status and future prospects in Japan. J Gastroenterol 2007;42:1-15.
8. Li YY, Hu PJ, Du GG, Hazell SL. The prevalence of Helicobacter pylori infection in the Peoples Republic of China. Am J Gastroenterol 1991;86:446-9.
9. Dooley CP, Cohen H, Fitzgibbons PL, Bauer M, Appleman MD, Perez-Perez GI, *et al*. Prevalence of Helicobacter pylori infection and histologic gastritis in asymptomatic persons. N Engl J Med 1989;321:1562-6.
10. Peterson WL. Helicobacter pylori and peptic ulcer disease. N Engl J Med 1991;324:1043-8.
11. Taylor DE, Hargreaves JA, Ng LK, Sherbaniuk RW, Jewell LD. Isolation and characterization of Campylobacter pyloridis from gastric biopsies. Am J Clin Pathol 1987;87:49-54.
12. Francis ND, Logan RP, Walker MM, Polson RJ, Boylston AW, Pinching AJ, *et al*. Campylobacter pylori in the upper gastrointestinal tract of patients with HIV-1 infection. J Clin Pathol 1990;43:60-2.
13. Megraud F. Resistance of Helicobacter pylori to antibiotics. Aliment Pharmacol Ther 1997;11 Suppl 1:43-53.
14. Marshall BJ. Helicobacter pylori. Am J Gastroenterol 1994;89 (8 Suppl):S116-28.
15. Malaty HM, Kim JG, Kim SD, Graham DY. Prevalence of Helicobacter pylori infection in Korean children: Inverse relation to socioeconomic status despite a uniformly high prevalence in adults. Am J Epidemiol 1996;143:257-62.
16. Schistosomiasis, liver flukes and helicobacter pylori. IARC Working Group on the Evaluation of Carcinogenic Risk to Humans, Lyon, 7-14 June 1994. IARC Monogr Eval Carcinog Risks Hum 1994;61:1-241.
17. NIH Consensus Conference. Helicobacter pylori in peptic ulcer disease. NIH Consensus Development Panel on Helicobacter pylori in Peptic Ulcer Disease. JAMA 1994;272:65-9.
18. Lichterfeld M, Lorenz C, Nischalke HD, Scheurlen C, Sauerbruch T, Rockstroh JK. Decreased prevalence of Helicobacter pylori infection in HIV patients with AIDS defining diseases. Z Gastroenterol 2002;40:11-4.
19. Edwards PD, Carrick J, Turner J, Lee A, Mitchell H, Cooper DA. Helicobacter pylori-associated gastritis is rare in AIDS: Antibiotic effect or a consequence of immunodeficiency? Am J Gastroenterol 1991;86:1761-4.
20. Panos GZ, Xirouchakis E, Tzias V, Charatsis G, Bliziotis IA, Doulgeroglou V, *et al.* Helicobacter pylori infection in symptomatic HIV-seropositive and -seronegative patients: A case-control study. AIDS Res Hum Retroviruses 2007;23:709-12.
21. Cacciarelli AG, Marano BJ Jr., Gualtieri NM, Zuretti AR, Torres RA, Starpoli AA, *et al.* Lower Helicobacter pylori infection and peptic ulcer disease prevalence in patients with AIDS and suppressed CD4 counts. Am J Gastroenterol 1996;91:1783-4.
22. Chiu HM, Wu MS, Hung CC, Shun CT, Lin JT. Low prevalence of Helicobacter pylori but high prevalence of cytomegalovirus-associated peptic ulcer disease in AIDS patients: Comparative study of symptomatic subjects evaluated by endoscopy and CD4 counts. J Gastroenterol Hepatol 2004;19:423-8.
23. Olmos M, Araya V, Pskorz E, Quesada EC, Concetti H, Perez H, *et al.* Coinfection: Helicobacter pylori/human immunodeficiency virus. Dig Dis Sci 2004;49:1836-9.
24. Sud A, Ray P, Bhasin DK, Wanchu A, Bambery P, Singh S. Helicobacter pylori in Indian HIV infected patients. Trop Gastroenterol 2002;23:79-81.
25. AliMohamed F, Lule GN, Nyong'o A, Bwayo J, Rana FS. Prevalence of Helicobacter pylori and endoscopic findings in HIV seropositive patients with upper gastrointestinal tract symptoms at Kenyatta National Hospital, Nairobi. East Afr Med J 2002;79:226-31.
26. Bamford KB, Fan X, Crowe SE, Leary JF, Gourley WK, Luthra GK*, et al.* Lymphocytes in the human gastric mucosa during Helicobacter pylori have a T helper cell 1 phenotype. Gastroenterology 1998;114:482-92.
27. Moran AP, Svennerholm AM, Penn CW. Pathogenesis and host response of Helicobacter pylori. Trends Microbiol 2002;10:545-7.
28. Moges F, Kassu A, Mengistu G, Adugna S, Andualem B, Nishikawa T, *et al*. Seroprevalence of Helicobacter pylori in dyspeptic patients and its relationship with HIV infection, ABO blood groups and life style in a university hospital, Northwest Ethiopia. World J Gastroenterol 2006;12:1957-61.
29. Lv FJ, Luo XL, Meng X, Jin R, Ding HG, Zhang ST. A low prevalence of and endoscopic findings in HIV-positive Chinese patients with gastrointestinal symptoms. World J Gastroenterol 2007;41:5492-6.
30. Fialho AB, Braga-Neto MB, Guerra EJ, Fialho AM, Fernandes KC, Sun JL, *et al.* Low prevalence of H. pylori infection in HIV-positive patients in the northeast of Brazil. BMC Gastroenterol 2011;11:13.
31. Hestvik E, Tylleskar T, Ndeezi G, Grahnquist L, Olafsdottir E, Tumwine JK, *et al.* Prevalence of Helicobacter pylori in HIV-infected, HAART-naive Ugandan children: A hospital-based survey. J Int AIDS Soc 2011;14:34.
32. Klein PD, Graham DY, Gaillour A, Opekun AR, Smith EO. Water source as risk factor for Helicobacter pylori infection in Peruvian children. Gastrointestinal Physiology Working Group. Lancet 1991;337:1503-6.
33. Epidemiology of, and risk factors for, Helicobacter pylori infection among 3194 asymptomatic subjects in 17 populations. The EUROGAST Study Group. Gut 1993;34:1672-6.
34. Luo HB, Hu ZW, Guo JW. [Helicobacter pylori infection in the gastric mucosa of patients with HIV/AIDS in different clinical stages]. Nan Fang Yi Ke Da Xue Xue Bao 2009;29:1397-9.
35. Welage LS, Carver PL, Revankar S, Pierson C, Kauffman CA. Alterations in gastric acidity in patients infected with human immunodeficiency virus. Clin Infect Dis 1995;21:1431-8.
36. Fong TL, Dooley CP, Dehesa M, Cohen H, Carmel R, Fitzgibbons PL, *et al.* Helicobacter pylori infection in pernicious anemia: A prospective controlled study. Gastroenterology 1991;100:328-32.
37. Lake-Bakaar G, Quandros E, Beidas S, Beidas S, Elsakr M, Tom W, *et al*. Gastric secretory failure in patients with

acquired immunodeciency syndrome (AIDS). Ann Intern Med 1988;109:502-4.
38. Benz J, Hasbach H, Brenden M, Eidt S, Fatkenheuer G, Schrappe M, *et al*. Humoral and cellular immunity in HIV positive and HIV negative Helicobacter pylori infected patients. Zentralbl Bakteriol 1993;280:186-96.
39. She RC, Wilson AR, Litwin CM. Evaluation of Helicobacter pylori Immunoglobulin G (IgG), IgA, and IgM serologic testing compared to stool antigen testing. Clin Vaccine Immunol 2009;16:1253-5.
40. Fabris P, Bozzola L, Benedetti P, Scagnelli M, Nicolin R, Manfrin V, *et al.* H. pylori infection in HIV-positive patients. A serohistological study. Dig Dis Sci 1997;42:289-92.

How to cite this article: Abdollahi A, Shoar S, Jafari S, Emadi-Kochak H. Seroprevalence of helicobacter pylori in human immunodeficiency virus-positive Patients and it's correlation with CD4 + Lymphocyte Count. Niger Med J 2014;55:67-72.

Source of Support: Nil, **Conflict of Interest:** None declared.