RAPID COMMUNICATION

A low prevalence of *H pylori* and endoscopic findings in HIV-positive Chinese patients with gastrointestinal symptoms

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patients may be different from the general population that is closely related to *H pylori* infection.

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Abstract

AIM: To compare the prevalence of *H pylori* infection, peptic ulcer, cytomegalovirus (CMV) infection and Candida esophagitis in human immunodeficiency virus (HIV)-positive and HIV-negative patients, and evaluate the impact of CD4 lymphocyte on *H pylori* and opportunistic infections.

METHODS: A total of 151 patients (122 HIV-positive and 29 HIV-negative) with gastrointestinal symptoms were examined by upper endoscopy and biopsy. Samples were assessed to determine the prevalence of *H pylori* infection, CMV, candida esophagitis and histologic chronic gastritis.

RESULTS: The prevalence of *H pylori* was less common in HIV-positive patients (22.1%) than in HIV-negative controls (44.8%; $P < 0.05$), and the prevalence of *H pylori* displayed a direct correlation with CD4 count stratification in HIV-positive patients. In comparison with HIV-negative group, HIV-positive patients had a lower incidence of peptic ulcer (20.7% vs 4.1%; $P < 0.01$), but a higher prevalence of chronic atrophy gastritis (6.9% vs 24.6%; $P < 0.05$), Candida esophagitis and CMV infection. Unlike HIV-negative group, *H pylori* infection had a close relationship to chronic active gastritis ($P < 0.05$). In HIV-positive patients, chronic active gastritis was not significantly different between those with *H pylori* infection and those without.

CONCLUSION: The lower prevalence of *H pylori* infection and peptic ulcer in HIV-positive patients with gastrointestinal symptoms suggests a different mechanism of peptic ulcerogenesis and a different role of *H pylori* infection in chronic active gastritis and peptic ulcer. The pathogen of chronic active gastritis in HIV-positive

INTRODUCTION

H pylori has been extensively studied and proven to be the main cause of chronic gastritis and peptic ulcer in the HIV-negative population^[1,2]. The reported prevalence of *H pylori* in unselected populations ranges from 32% to 65%^[3-6]. Over 90% patients with chronic active gastritis showed an evidence of *H pylori* infection^[3,4,7], and 70%-100% of those patients had peptic ulcer disease^[1,5,7].

In contrast, the prevalence of *H pylori* infection in patients infected with HIV has been reported to be remarkably low^[8-11]. Reasons for these lower rates of *H pylori* infection remain unclear. Other studies showed that *H pylori* infection is similar in both HIV-positive and HIV-negative patients^[12,13]. Patients infected with HIV, with or without acquired immune deficiency syndrome (AIDS), have a high incidence (50%-90%) of upper gastrointestinal symptoms^[14]. The immune deficiencies caused by HIV give rise to many different gastrointestinal opportunistic infections, such as cytomegalovirus (CMV) infection and fungal esophagitis^[15,16].

The aims of our study are to assess the prevalence of *H pylori* infection and the association with histological chronic active gastritis in HIV-positive patients with gastrointestinal symptoms. The impact of CD4⁺ count on the prevalence of *H pylori*, gastric CMV infection and Candida esophagitis was also evaluated.

MATERIALS AND METHODS

Patients

The study was carried out at Beijing You'an Hospital,

Capital Medical University, Beijing, the largest referral center for management of HIV infection and HIV-related complications in China, from January 2003 to March 2006. Endoscopy was performed in 151 patients for gastrointestinal symptoms such as abdominal pain, dyspepsia, diarrhea, nausea, vomiting, gastrointestinal bleeding, and odynophagia or dysphagia.

The study groups consisted of 122 HIV-positive patients (49 males and 73 females; mean age 40.8 ± 7.9 , range 26–60 years) and 29 age-matched HIV-negative patients (15 males and 14 females; mean age 49.5 ± 12.7 , range 28–77 years) as control groups. The absolute CD4⁺ lymphocyte count of HIV-positive patients at the time of endoscopic examination was measured with FACS Count Reagents (BD Company, USA). Patients all gave their consent before undergoing endoscopy, and the symptoms, consumption of medications within one month, including antibiotics, proton pump inhibitors were also recorded.

Endoscopy, diagnosis and histology

Video-endoscopes (Olympus XQ240, Tokyo, Japan) were used for the procedure. All patients underwent three biopsies from the lesser and greater curvature of the gastric antrum and lesser curvature of lower body, one for Rapid Urease Test (RUT) and two for histology. Additional biopsies were obtained from endoscopic lesions such as ulceration. The biopsy specimens were placed in 10% formaldehyde at the time of endoscopy and stained with hematoxylin-eosin, Warthin-Starry stains for histologic chronic gastritis and *H. pylori* infection, and immunocytochemical techniques were performed for CMV infection (Monoclonal Mouse Anti-Human Cytomegalovirus, Dako). The *H. pylori* infection was diagnosed by positive identification of both the organism on histology (Warthin-Starry) and RUT. The histologic gastritis was diagnosed according to the Sydney criteria^[17]. Specimens were reviewed by only one pathologist who was blind to the status of those patients in present study.

The Candida esophagitis was diagnosed by sheathed brush cytology from endoscopic lesions, and gross appearance of mucosal presented with white plaques. Specimens obtained by sheathed brush should be smeared onto slides for fungi.

Statistical analysis

Chi-square test or Fisher exact probability tests were used to compare the prevalence of *H. pylori*, CMV infection, Candida esophagitis, and peptic ulcer between HIV-positive patients, control groups, and HIV-infected patients with higher and lower CD4⁺ counts and the use of antibiotics and proton pump inhibitors. Independent sample *t* test was used to compare the age and sex between the HIV-positive and control groups. A value of $P < 0.05$ was regarded as statistically significant.

RESULTS

The patient data and the prevalence of *H. pylori* and endoscopic findings in HIV-positive patients and HIV-negative patients are shown in Table 1. The gastrointestinal

Table 1 Patient data and clinicopathology

	HIV-positive (<i>n</i> = 122) (%)	HIV-negative (<i>n</i> = 29) (%)	<i>P</i>
Age (yr)	40.8 ± 7.9	49.5 ± 12.7	NS
Male	49	15	NS
Female	73	14	NS
Gastrointestinal symptoms			NS
Abdominal pain and distention	38	14	NS
Dyspepsia	42	6	NS
Diarrhea	27	1	0.02
Nausea and vomiting	47	8	NS
Odynophagia and dysphagia	21	0	0.035
Others	16	6	NS
Consumption of medications within one month, <i>n</i> (%)			
Antibiotics	47 (38.5)	3 (10.3)	0.004
Proton pump inhibitor	3 (2.5)	4 (13.8)	0.034
<i>H. pylori</i> infection	27 (22.1)	13 (44.8)	0.013
Candida esophagitis	19 (15.6)	0 (0)	0.05
Peptic ulcer	5 (4.1)	6 (20.7)	0.007
Chronic atrophy gastritis	30 (24.6)	2 (6.9)	0.036
CMV infection	6 (4.9)	0 (0)	0.49

CMV: Cytomegalovirus; NS: Not significant.

Table 2 *H. pylori* infection and previous use of antibiotics related to CD4⁺ count in HIV-positive patients *n* (%)

CD4 ⁺ count	<i>H. pylori</i> infection	<i>P</i>	Antibiotic therapy	<i>P</i>
CD4 ⁺ ≥ 200/μL (<i>n</i> = 65)	19 (29.2)	0.044	19 (29.3)	0.024
CD4 ⁺ < 200/μL (<i>n</i> = 57)	8 (14.0)		28 (49.1)	
CD4 ⁺ ≥ 100/μL (<i>n</i> = 85)	24 (28.2)	0.014	29 (34.1)	0.13
CD4 ⁺ < 100/μL (<i>n</i> = 37)	3 (8.1)		18 (48.6)	

symptoms of HIV-positive patients were mostly nonspecific, such as diarrhea, dyspepsia, abdominal pain, nausea, vomiting, and odynophagia or dysphagia. Only the occurrence of symptoms of diarrhea, odynophagia, and dysphagia in HIV-positive patients was significantly higher than that of control group ($P < 0.05$). The prevalence of *H. pylori* infection was significantly lower in the HIV-positive group than that of HIV-negative control group (27/122; 22.1% *vs* 13/29; 44.8%, $P < 0.05$). Endoscopic examination revealed more patients with peptic ulcer in HIV-negative group than in HIV-positive group (6/29; 20.7% *vs* 5/122; 4.1%, $P < 0.01$). More histologic chronic atrophy gastritis was found in HIV-positive patients than in HIV-negative group (30/122; 24.6% *vs* 2/29; 6.9%, $P < 0.05$). Opportunistic infection by CMV was noted in 4.9% (6/122) HIV-positive patients but none in the HIV-negative group ($P = 0.49$). The incidence of Candida esophagitis in HIV-positive patients (19/122; 15.6%) was significantly higher than that of HIV-negative patients ($P < 0.05$).

H. pylori infection was less common in those with CD4⁺ counts < 200/μL than those with CD4⁺ counts > 200/μL in HIV-positive patients (8/57; 14.0% *vs* 19/65; 29.2%, $P < 0.05$). Interestingly, the prevalence of *H. Pylori* infection displayed a direct correlation with the CD4⁺ lymphocyte count stratification in HIV-positive patients (Table 2). The Candida esophagitis was significantly more common in

Table 3 Relationship of CD4⁺ count to *H. pylori* infection and Endoscopic Findings in HIV-positive patients *n* (%)

	CD4 ⁺ ≥ 200/μL (<i>n</i> = 65)	CD4 ⁺ < 200/μL (<i>n</i> = 57)	<i>P</i>
<i>H. pylori</i> infection	19 (29.2)	8 (14)	0.044
Candida esophagitis	4 (6.2)	15 (26.3)	0.002
Peptic ulcer	2 (3.1)	3 (5.3)	0.881
Chronic atrophy gastritis	13 (20)	17 (29.8)	0.209
CMV infection	1 (1.5)	5 (8.8)	0.155

HIV-positive patients with CD4⁺ count < 200/μL than those with CD4⁺ count > 200/μL (15/57; 26.3% *vs* 4/65; 6.2%, *P* < 0.01), and the average CD4⁺ counts of patients with Candida esophagitis was 116.47 ± 133.08/μL. The CMV infection was more common in HIV-positive patients with CD4⁺ count < 200/μL than those with CD4⁺ count > 200/μL, but it was not statistically significant (5/57; 8.8% *vs* 1/65; 1.5%, *P* = 0.155) (Table 3).

Histological examination revealed less chronic active gastritis in HIV-positive patients than in HIV-negative control group (24/122; 19.7% *vs* 9/29; 31%, *P* = NS), but the difference was not statistically significant. The relationship of *H. pylori* infection with chronic active gastritis was evaluated in HIV-positive and HIV-negative patients (Table 4). In HIV-negative group, the incidence of chronic active gastritis was significantly higher in those with *H. pylori* infection (61.5%) than those without (6.3%; *P* < 0.01). In HIV-positive group, the rate of chronic active gastritis was not significantly different between those with *H. pylori* infection (29.6%) and those without (16.8%; *P* = 0.14).

The relationship in *H. pylori* and CMV infection and peptic ulcer was evaluated between the two groups of patients. Peptic ulcer was detected in five HIV-positive patients, one of whom (20%) was positive for *H. pylori* infection and one (20%) for CMV infection. In HIV-negative group, six patients were diagnosed as having peptic ulcer, four (67%) as *H. pylori* infection and none as CMV infection.

The previous use of antibiotics and proton pump inhibitor was also evaluated between HIV-positive and HIV-negative patients (47/122; 38.5% *vs* 3/29; 10.3%, *P* < 0.01 and 3/122; 2.5% *vs* 4/29; 13.8%, *P* < 0.05, respectively) (Table 1). If CD4⁺ count was taken into consideration, the use of all kinds of antibiotics in HIV-positive patients with CD4⁺ counts < 100/μL was not significantly different in those with CD4⁺ counts > 100/μL (18/37; 48.6% *vs* 29/85; 34.1%, *P* = 0.13) (Table 3). Those antibiotics mainly included Sulfonamides, penicillins and quinolones. None of the patients took NSAIDs, aspirin or steroid before endoscopic examination.

The HIV-positive patients in this study were usually concomitant with HCV and/or HBV infection which was more significantly frequent than in HIV-negative patients (102/122; 83.6% *vs* 2/29; 6.9%, *P* < 0.01). Nine patients with esophagogastric varices (7.4%) and 3 patients with portal hypertensive gastropathy (2.5%) in HIV-positive group were also found by endoscopic examination.

Table 4 Relationship of chronic active gastritis to *H. pylori* infection

	HIV-positive group (<i>n</i> = 122)		HIV-negative group (<i>n</i> = 29)	
	<i>H. pylori</i> ⁺ (<i>n</i> = 27)	<i>H. pylori</i> ⁻ (<i>n</i> = 95)	<i>H. pylori</i> ⁺ (<i>n</i> = 13)	<i>H. pylori</i> ⁻ (<i>n</i> = 16)
Chronic active gastritis, <i>n</i> (%)	8 (29.6)	16 (16.8)	8 (61.5)	1 (6.3)
<i>P</i>	0.140		0.005	

DISCUSSION

In the present study, we found that the majority of gastrointestinal symptoms of HIV-positive patients at our hospital were similar to that of HIV-negative group. In comparison with HIV-negative group, the symptoms of diarrhea, odynophagia, and dysphagia were significantly more in HIV-positive patients (*P* < 0.05). Several previous studies^[16,18-19] revealed that more than 71% of AIDS patients who present with dysphagia and odynophagia have endoscopic evidence of esophageal candidiasis. Our result showed a high infection rate of Candida esophagitis in HIV-positive patients (19/122; 15.6%), which may be a possible explanation. Studies showed that the incidence of Cryptosporidium infection has been estimated to be 16%-33% in the AIDS patients in north America with chronic diarrhea^[20,21]. In developing countries, the infection of Cryptosporidium was 55% among AIDS patients^[22]. The etiological factor of diarrhea in HIV-positive patients in our study was not evaluated.

The prevalence of *H. pylori* infection in HIV-positive patients at our hospital was significantly lower than that in HIV-negative control group. Our results were in agreement with some previous reports^[8-11]. The reason of lower prevalence of *H. pylori* infection may be lack of CD4⁺ cells, use of antibiotics and proton pump inhibitor, decreased acid secretion, or competitive inhibition by other pathogens in HIV-positive patients.

According to previous reports, CD4⁺ lymphocytes were reported to be involved^[23-25] in the pathogenesis of *H. pylori*-related gastritis or ulcer. It is well known that CD4⁺ cells play a role in inducing gastritis and this gastritis might be a mechanism by which *H. pylori* colonization is enhanced^[26]. Patients with HIV infection and a low CD4⁺ count would then lose this mechanism by which *H. pylori* colonization is sustained, and infection intensity would diminish. In addition, the T-cell response to the organism could serve to induce tissue and epithelial damage. In AIDS patients, the decreased T-cell would induce a decreased incidence of *H. pylori* gastritis^[27]. In our results, a stratification of cases on the basis of CD4⁺ count has shown a decrease of *H. pylori* infection with the progression of HIV-related disease, and histological examination revealed less chronic active gastritis in HIV-positive patients than in HIV-negative control group (19.7% *vs* 31%). *H. pylori* infection was closely related to chronic active gastritis in HIV-negative group (*P* < 0.05), but not in HIV-positive patients, indicating that other pathogens might exist, such as CMV and Cryptosporidium infection.

An impairment of *H. pylori* colonization environment

might result from a progressive atrophic involution of the gastric mucosa with secondary decreased acid secretion in HIV-positive patients, which represents an altered intragastric environment^[28,29]. In our results, histologic chronic atrophy gastritis in HIV-positive patients was significantly higher than in HIV-negative group (30/122; 24.6% *vs* 2/29; 6.9%, $P < 0.05$), which might be result of gastric secretory failure in HIV infection patients. The impaired acid secretion may allow subsequent gastric bacterial overgrowth and provide a less suitable environment or competitive inhibition for *H pylori* colonization.

An altered intragastric environment might also result from frequent use of antibiotics against opportunistic infections in patients at an advanced stage of HIV infection^[30]. In comparison with HIV-negative group, HIV-positive group had a more frequent use of antibiotics. In HIV-positive patients, previous use of antibiotics with $CD4^+$ counts $< 100/\mu\text{L}$ was not significantly different from that of $CD4^+$ counts $> 100/\mu\text{L}$ ($P = 0.13$), but the prevalence of *H pylori* infection showed significant difference. In our patients, the antibiotics most frequently used was trimetoprim-sulfa, usually for treatment or prophylaxis against pneumocystis in HIV-positive patients, and monotherapy of antibiotics has been proven to inhibit rather than eradicate *H pylori*^[31]. Therefore, current prophylaxis has been excluded from evaluation of eradicating the microorganism. Previous use of proton pump inhibitors might alter intragastric environment and therefore influence the prevalence of *H pylori* infection. In the present study, the HIV-negative control group took proton pump inhibitors more frequently than HIV-positive group (13.8% *vs* 2.5%, $P < 0.05$), which further proved the lower prevalence of *H pylori* infection in HIV-positive patients.

In this study, all 122 HIV-positive patients with gastrointestinal symptoms, only 4.1% had peptic ulcer, but 20.7% in HIV-negative group. This might explain that the low prevalence of *H pylori* infection result in the lower incidence of ulcers among HIV-positive patients. On the other hand, decreased acid secretion in HIV-positive patients plays a role in the lower incidence of peptic ulcer. According to previous reports, CMV-associated peptic ulcer disease was highly prevalent and CMV was the only organism significantly associated with gastroduodenal ulcers in HIV-positive patients, and *H pylori* was an uncommon cause of peptic ulcer^[11,32]. In our study, among the 5 patients with peptic ulcer in HIV-positive group, only one proved to have CMV infection, which was lower according to previous studies^[11,32]. The inadequate biopsies in the present study may be a possible explanation. According to literature, histological changes of CMV infection are patchy in distribution, however, and the single biopsy sensitivity for ulcerative lesions has been reported to be as low as 13%. Therefore, at least 8-10 biopsies of suspicious lesions are recommended^[33].

Candida esophagitis is one of the most common opportunistic infections in patients with AIDS^[34]. Our study showed that the Candida esophagitis was significantly higher in HIV-positive patients with $CD4^+$ count below $200/\mu\text{L}$, and the average $CD4^+$ counts with

Candida esophagitis was $116.47 \pm 133.08/\mu\text{L}$. According to previous studies, the CMV infection is also a common opportunistic pathogen in HIV-positive patients with a low $CD4^+$ count and is one of the main causes of gastrointestinal ulcer in AIDS patients^[11,33]. The incidence of CMV infection in HIV-positive patients (4.9%) in our study was lower than previous reports^[32]. The incorrect location of biopsy may be another possible reason. According to literature review, gastric CMV infections are usually seen in the fundus with contiguous involvement of the esophagus and gastroesophageal junction, and the distal stomach and antrum are less commonly involved^[22,35]. In the present study, the biopsy specimens were usually obtained from gastric antrum and lower body, therefore might lower the incidence of CMV infection in our patients.

The HIV-positive patients in the present study, mainly from Henan Province of China, infected through illegal blood plasma collection, and usually coinfectd with HCV and/or HBV infection (83.6%). Endoscopic examination also revealed findings such as esophagogastric varices and portal hypertensive gastropathy, which were significantly different from previous reports.

In summary, we have found that a lower prevalence of *H pylori* infection and peptic ulcer in HIV-positive patients with gastrointestinal symptoms than that of HIV-negative patients with similar symptoms. The mechanism of chronic active gastritis in HIV-positive patients may be different from HIV-negative group that was closely related to *H pylori* infection. Various opportunistic infections (especially Candida esophagitis) of upper gastrointestinal tract likely occur in HIV-positive patients with a $CD4^+$ count less than $200/\mu\text{L}$.

COMMENTS

Background

Helicobacter pylori has been proven to be the main cause of chronic gastritis and peptic ulcer in the HIV-negative population. The role and prevalence of *H pylori* infection might be different in the HIV infected patients.

Research frontiers

The immune deficiencies caused by HIV give rise to many different gastrointestinal opportunistic infections, and the prevalence of *H pylori* infection in patients infected with HIV is remarkably low.

Innovations and breakthroughs

It is the first report to characterize the prevalence and role of *H pylori* infection in chronic active gastritis and peptic ulcer in HIV-positive patients infected through illegal blood plasma collection in China, who are usually coinfectd with HCV and/or HBV. The pathogen of chronic active gastritis in HIV-positive patients may be different from the general population that was closely related to *H pylori* infection.

Applications

This observation might be of potential value in HIV-positive patients with gastrointestinal symptoms.

Peer review

The authors compared the prevalence of *H pylori* infection, peptic ulcer, cytomegalovirus (CMV) infection and Candida esophagitis in human immunodeficiency virus(HIV)-positive and HIV-negative patients. The lower prevalence of *H pylori* infection and peptic ulcer in HIV-positive patients with gastrointestinal symptoms suggests a different mechanism of peptic ulcerogenesis and a different role of *H pylori* infection in chronic active gastritis and peptic ulcer.

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