

Incidence and Treatment Outcomes of *Helicobacter pylori* Infection in HIV-infected Patients

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ABSTRACT

Helicobacter pylori is an established causative agent of peptic ulcer disease throughout the world. There is paucity of information regarding co-infection in HIV-infected individuals. We analyzed HIV-infected patients diagnosed with *H. pylori* infection in our institution over a six year period.

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Key words: *Helicobacter pylori*; HIV infection; Co-infection

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DEAR EDITOR

Several studies have indicated *Helicobacter pylori* infections to be common in HIV-infected patients and prevalence of the disease to be higher in patients with CD4+ T-cell counts greater than 200/ μ L^[1-2]. Nkuize *et al*, reviewed the characteristics of 48 HIV-infected patients with *H. pylori* infection who underwent upper endoscopic evaluations. They demonstrated co-infection to be more common in patients of Black African origin, in heterosexual individuals, in patients with a high BMI, high CD4+ T-cell counts, and low HIV viral loads^[3]. There is little data on *H. pylori* treatment choices and outcomes of

treatment in HIV-infected patients. Therefore, we retrospectively analyzed our HIV-infected patients that were diagnosed with *H. pylori* infection from 1/1/2004-12/31/2010. From 4918 HIV-infected patients seen in our clinic during the study period, 169 were diagnosed with *H. pylori* infection. All 169 patients had symptoms: Reflux esophagitis-like symptoms or dyspepsia, or dysphagia/epigastric discomfort. 150 patients were diagnosed by serology alone, 12 patients had also positive stool antigen, and 7 were diagnosed incidentally by a positive histology after undergoing diagnostic upper endoscopy and gastric biopsy. We analyzed demographic data, HIV risk factors, CD4+ T cell counts, HIV viral loads, birth place/travel histories, incarceration history, upper endoscopies, and stool antigen studies. We reviewed the treatment choices used and their outcomes.

The prevalence of *H. pylori* infection was 169/4918, 3.4%. Most were men, 106 (62.7%). See Table 1. The median age was 46 years. The median CD4+ T-cell count was 429/ μ L (range 2-1486). Only 22 patients had CD4<200. 104 patients had undetectable HIV viral load. 77% of patients were on antiretroviral therapy. The majority of patients were African American (70/169, 41.4%) and Hispanic (85/169, 50.2%); only 13/169 (7.69%) were Caucasian, and 1/169 was Asian. The HIV-risk factors were 44% Heterosexual, 38% were men who have sex with men, 17% were IV drug users, and 1% acquired HIV via blood transfusion. 64 patients were born outside the United States (78% from Latin America/Caribbean), 16% Africa, 3% Asia, see figure 1, and 15 had travelled to known

Table 1 169 HIV-infected patients co-infected with *H. pylori*.

Diagnosis (# of patients)	150, Serology Alone
	12, Stool Antigen positive
	7, Positive gastric biopsy
HIV Risk Factors %	Heterosexual 44%
	MSM 38%
	IVDU 17%
	Blood transfusion 1%
Race/Ethnicity (%)	African American 70 (41.4%)
	Hispanic 85 (50.3%)
	Caucasian 13 (7.7%)
	Asian 1 (0.6%)
Gender	Men 106 (62.7%)
	Women 63 (37.3%)
On HAART (%)	130 patients (77%)
Undetectable HIV Viral load (%)	104 patients (61.5%)
Median CD4+ T cell(range)	429/ μ L (2-1486)
# of patients received Treatment	129

MSM: men who have sex with men; IVDU: Intravenous drug use; HAART: Highly active antiretroviral therapy.

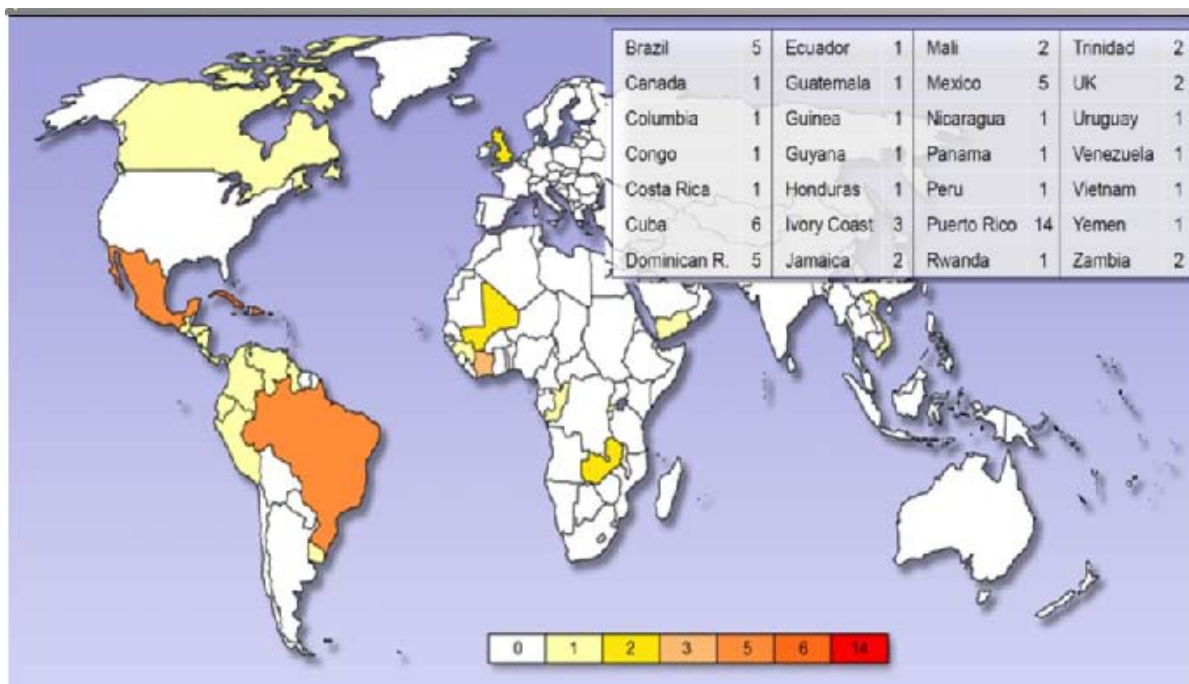


Figure 1 Patients born Outside the United States.

H. pylori endemic regions prior to the HIV diagnosis. 46 patients had history of incarceration in United States. 48 patients underwent upper endoscopic evaluation, 15 of them had upper endoscopy after treatment that confirmed eradication of infection.

Treatment was offered in 129 patients. 40 patients either did not have stool antigen testing or were lost to follow up. The most common regimen used (in 113 patients) was a 14-days course of Amoxicillin-Clarithromycin and Proton Pump Inhibitor (PPI) known as the standard therapy. 8 patients used Bismuth-Metronidazole-Tetracycline regimen; 3 Clarithromycin-Metronidazole-PPI; 2 Clarithromycin-Tetracycline-PPI and 2 a levofloxacin based-PPI regimen. See figure 2.

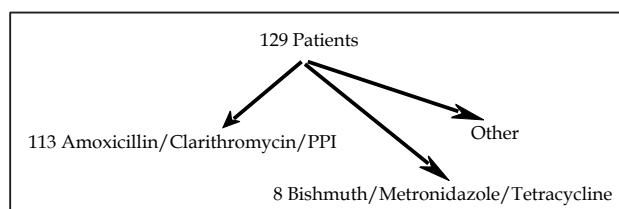


Figure 2 Choice of Treatment Regimens for *H. pylori* eradication. PPI: Proton Pump Inhibitor; Other treatments include: Clarithromycin/Metronidazole/PPI; Clarithromycin/Tetracycline/PPI.

From the 129 patients, eight patients had evidence of treatment failure (persistent positive stool antigen). All had failed amoxicillin-based therapies, and were receiving HAART; their mean CD4+ T cell count was 535 (218-1103), median was 472. One patient was retreated with the same regimen (amoxicillin/clarithromycin/PPI) and the stool antigen was again positive; he was then treated with sequential therapy, amoxicillin/PPI for five days, followed by five days of tinidazole/clarithromycin/PPI and the stool antigen became negative. Two patients were retreated with tinidazole/amoxicillin/PPI but were lost to follow up. Four patients were retreated with Bismuth-Metronidazole-Tetracycline, one had resolution of symptoms, the other developed negative stool antigen, and the other two patients were lost to follow up. 1/129 patients died from unknown etiology on the 3rd day of *H. pylori* treatment. The (13)C -Urea breath test (UBT) was not available, and the stool antigen test was obtained 2

months after treatment and after a chronic PPI was held for at least two weeks before obtaining the specimen. 15 patients underwent upper endoscopy and biopsy after treatment, and histological analysis confirmed eradication of the organisms.

Successful eradication of *H. pylori* in HIV-infected patients had been documented^[4]. Magen *et al.*, showed 11 HIV-infected patients treated with amoxicillin based therapy to have a negative UBT eight weeks after completion of treatment. Persistent infection in non-HIV infected patients is also well documented^[5]. Recent clinical trials and meta-analyses have also shown that the eradication rate of the standard triple therapy has declined to less than 80% and this is attributed to the increasing incidence of clarithromycin-resistant strains of *H. pylori*^[6]. The respected European Helicobacter Study Group has recently released its guidelines for management of *H. pylori* infection; regional knowledge of clarithromycin resistance rates now dictates initial treatment strategies^[7]. (For further details, the authors highly recommend reviewing the referenced guidelines).

From our review of the literature we didn't find other reports of HIV-infected patients who had failed standard triple therapy for *H. pylori* infection. In our experience, use of bismuth or sequential therapy was successful in achieving eradication where the standard triple therapy had failed. Our study has many limitations stemming from its retrospective nature and from the fact that many patients were diagnosed on positive serology and symptoms alone. This fact points to the difficulties and challenges that physicians face in clinical practice as many patients didn't provide stool specimen, and UBT was not easily available.

H. pylori co-infection in our large urban HIV-infected population was not uncommon. It was found in patients that grew up or had travelled to endemic regions. Treatments were in general well tolerated, however, treatment failures to the standard triple therapy was noted. Physicians who treat *H. pylori* infection in HIV-infected patients should be aware of increasing incidence of clarithromycin resistant *H. pylori* stains. We strongly recommend pursuing test of cure in all HIV-infected patients who received the standard triple therapy, and if failure is detected, to follow appropriate retreatment strategies as outlined by recently released guidelines.

REFERENCES

- 1 Nkuize M, De Wit S, Muls V, Arvanitakis M, Buset M. Upper gastrointestinal endoscopic findings in the era of highly active antiretroviral therapy. *HIV Med* 2010; **11**: 412-417
- 2 Fialho ABC, Braga-Neto MB, Guerra AJC, et al. Low prevalence of *H. pylori* infection in HIV-positive patients in the Northeast of Brazil. *BMC Gastroenterol* 2011; **11**: 13
- 3 Nkuize M, De Wit S, Muls V, Ntounda R, Gomez-Galdon M, Buset M. Comparison of demographic characteristics and upper gastrointestinal endoscopy findings in HIV-positive, antiretroviral-treated patients with and without *Helicobacter pylori* infection. *Helicobacter* 2012; **17**: 153-159
- 4 Magen E, Elbirt D, Agmon-Levin N, Mishal J, Stoege Z. Eradication of *Helicobacter pylori* can facilitate immune re-constitution in HIV-1 infected immunological non-responders. *Int J Infect Dis* 2010; **14**: e322-e327
- 5 McColl KE. *Helicobacter pylori* Infection. *N Engl J Med* 2010; **362**: 1597-1604
- 6 Seng-Kee C, Feng-Woei T, Ping-I H, Deng-Chyang Wu. A new look at Anti-*Helicobacter pylori* therapy. *World J Gastroenterol* 2011; **17**: 3971-3975
- 7 Malfertheiner P, Megraud F, O'Morain CA, et al Management of *Helicobacter pylori* infection – the Maastricht IV/ Florence Consensus Report. *Gut* 2012; **61**: 646-664

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