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PREVALENCE OF *HELICOBACTER PYLORI* AND ENDOSCOPIC FINDINGS IN HIV SEROPOSITIVE PATIENTS WITH UPPER GASTROINTESTINAL TRACT SYMPTOMS AT KENYATTA NATIONAL HOSPITAL, NAIROBI

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# PREVALENCE OF *HELICOBACTER PYLORI* AND ENDOSCOPIC FINDINGS IN HIV SEROPOSITIVE PATIENTS WITH UPPER GASTROINTESTINAL TRACT SYMPTOMS AT KENYATTA NATIONAL HOSPITAL, NAIROBI

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## ABSTRACT

***Background:*** **Human immunodeficiency virus (HIV) seropositive patients frequently experience upper gastrointestinal tract (GIT) symptoms that cause considerable morbidity and are due to multiple aetiologies. The role of *Helicobacter pylori* gastric mucosal infection in HIV related upper GIT morbidity is unclear. No data exist on the prevalence of *H.pylori* gastric mucosal infection and upper gastrointestinal endoscopic findings in HIV seropositive patients at the Kenyatta National Hospital.**

***Objectives:*** **The aim of the study was to determine the prevalence of *H. pylori* gastric mucosal infection and the pattern of upper gastrointestinal endoscopic findings in HIV seropositive patients.**

***Design:*** **A hospital-based prospective case-control study.**

***Setting:*** **Kenyatta National Hospital, Endoscopy Unit.**

***Subjects:*** **Fifty two HIV seropositive patients with upper GIT symptoms were recruited (as well as 52 HIV seronegative age and gender matched controls).**

***Intervention:*** **Both cases and control subjects underwent upper GIT endoscopy and biopsies were taken according to a standard protocol. *H. pylori* detection was done by the rapid urease test and histology, and *H. pylori* gastric mucosal infection was considered to be present in the presence of a positive detection by both tests; biopsies were also taken for tissue diagnosis and CD4+ peripheral lymphocyte counts were determined using flow cytometry.**

***Results: H. pylori* prevalence was 73.1% [95% CI 59.9-83.8] in HIV positive subjects and 84.6% [95% CI 72.9-92.6] in HIV negative controls (p=0.230). Prevalence of *H. pylori* decreased with decreasing peripheral CD4+ lymphocyte counts. Median CD4+ lymphocyte count was 67 cells per cubic millimetre in HIV positive patients. On endoscopy, the most common lesion in HIV positive patients was oesophageal candidiasis (occurring in 51.9%), which was often associated with presence of oral candidiasis and, together with erosions, ulcers and nodules in the oesophagus, occurred exclusively in these patients. A few cases of cytomegalovirus and herpes simplex oesophagitis were seen, as were cases of upper GIT Kaposi's sarcoma, and one gastric lymphoma.**

***Conclusions: H. pylori* prevalence was not significantly different between HIV positive and HIV negative subjects, and decreased in HIV positive subjects with decreasing CD4+ cell counts. Oesophageal candidiasis was the most important endoscopic finding in HIV positive patients and was often associated with oral thrush.**

## INTRODUCTION

Human immunodeficiency virus (HIV) related morbidity is a major disease burden in Kenya currently and accounts for the highest number of hospital admissions at Kenyatta National Hospital (KNH)(1). The prevalence of HIV has been increasing in Kenya over the years. The national HIV prevalence has increased from 4.8% in 1990 to 13.5% in 2000. The numbers of in-patient admissions due to HIV related morbidity have also increased over the years at KNH.

Up to 90% of HIV seropositive patients experience gastrointestinal symptoms at some time of their illness(2) and these present important diagnostic and management challenges since clinical signs and symptoms rarely suggest a specific aetiology. Multiple pathogens are usually present during the terminal stages of HIV disease and therefore objective studies should be done to identify specific infections or neoplasms associated with advanced HIV infection. The aetiologies of GIT symptoms range from opportunistic infections to neoplasms. Nutritional status in AIDS patients is partly challenged by pathogens in the upper GIT(3). It is important therefore to identify these pathogens and treat them accordingly.

*H. pylori* has been associated with type B gastritis in 84.6%-100% of patients, peptic ulcer disease in 95%-100% and non-ulcer dyspepsia in 80.5%(4-5). The prevalence of *H. pylori* is higher in developing countries than in developed countries and has been shown to be inversely related to socio-economic status(6).

Very few studies have been done on *H. pylori* prevalence in HIV seropositive individuals. The reports have been on small series of cases and there appears to be no data from Africa as a whole on this subject. We therefore aimed to determine the prevalence of *H. pylori* gastric mucosal infection in HIV seropositive patients with upper gastrointestinal symptoms and describe upper gastrointestinal endoscopic findings in these patients.

## MATERIALS AND METHODS

*Study Population:* This was a case-control study carried out at the endoscopy unit at KNH. KNH is a national referral and teaching hospital situated in the city of Nairobi in Kenya. Recruitment into the study began on the 15th June 2000 and ended on 15th April 2001. Consecutive patients aged 12 years and above with upper GIT symptoms (dysphagia, odynophagia, nausea, vomiting, haematemesis, heartburn and dyspepsia) referred for endoscopy and satisfied the study criteria and were willing to participate in the study and undergo HIV antibody testing after appropriate pre-test counselling, were recruited. Cases were HIV seropositive patients with upper GIT symptoms, who satisfied the study criteria. Controls were HIV seronegative patients with upper GIT symptoms, matched for age and sex. Patients who had been on the following drugs for at least two weeks prior to endoscopic examination: proton pump inhibitors, histamine-2 receptor antagonists, bismuth salts, antibiotics, or antifungal drugs were excluded from the study as were those with a history or evidence of a bleeding diathesis. An informed consent was obtained from all patients. This study was undertaken after approval by the Ethical and Scientific Review Committee of KNH.

A total of 104 patients were studied, 52 of whom were HIV seropositive (20 males and 32 females, median age 32 years range 18 - 55 years) and 52 HIV seronegative subjects (20 males and 32 females, median age 29.5 years, range 18 - 50 years). The median CD4+ cell count in the HIV seropositive patients was 67 cells/mm$^3$ (range 2-1126) compared to 894 cells/mm$^3$ (range 341-1493) in HIV seronegative subjects. Most of the patients were of low socio-economic status having resided in rural areas before moving to the city.

*Laboratory methods:* Upper GIT endoscopy was done in the standard manner, after initial local pharyngeal anaesthesia by an endoscopist. After macroscopic examination, ten biopsies were taken, two from each of the following sites: terminal oesophagus, gastric body, incisura angularis, antrum and from the second part of the duodenum. Additional biopsies were taken from any suspicious lesions. One biopsy specimen from the antrum, incisura and body was placed into the CLO test slide for rapid urease test. The rest of the biopsies were fixed in 10% buffered formalin in separate labelled bottles before transport to the laboratory. Specimens were fixed for at least six hours before processing. Haematoxylin and eosin staining was used for tissue diagnosis and cold ZN stain for *H. pylori*. Modified Giemsa was used for quality control for *H. pylori* staining. Silver impregnated Grocott stain was used to identify *Candida* spp. A histopathologist who was blinded to the gross endoscopic findings and CLO test results read the histology slides. Patients were considered to be *H. pylori* positive only when both the CLO test and cold ZN histology were positive. HIV screening was done using the Elisa system (Organon Tecknika, Belgium) and CD4+ and CD8+ cell counts were analysed by a flow cytometer (Becton Dickinson, USA).

*Statistical analysis:* All data emanating from the study was entered into a computer database and analysis performed using the SPSS 10.0 statistical software. The prevalence of *H. pylori* in the HIV seropositive group was determined as a percentage (with 95% confidence interval) and compared to that in the control group. The prevalence of endoscopic macroscopic findings and histopathologic diagnoses were determined and compared between case and control subjects. The results were stratified by the CD4+ cell counts to see if there was any correlation between the CD4+ cell counts and upper GIT pathology and *H. pylori* prevalence. Kappa test was used for agreement. Significance of differences in the prevalence was tested using the Chi-square or Fisher's exact test. Statistical significance was defined as a p value less than 0.05.

## RESULTS

Overall, the prevalence of *H. pylori* in the study patients was 78.8% [95% CI 70.2-85.9]. Prevalence of *H. pylori* in HIV seropositive patients was 73.1% [95% CI 59.9-83.8] and in HIV seronegative patients was 84.6% [95% CI 72.9-92.6]. The prevalence of *H. pylori* in HIV seropositive patients was lower than in HIV seronegative patients but the difference was not statistically significant (p=0.230).

The prevalence of *H. pylori* was stratified according to CD4+ counts in HIV seropositive patients (Table 1). The prevalence of *H. pylori* decreased with decreasing CD4 cell counts, being significantly lower in those with CD4 cell counts <50/mm$^3$ (57.1%) as compared to those with a count ≥ 50/mm$^3$ (86.6%). Stratification of *H. pylori* according to CD4+ cell counts in HIV seropositive patients with dyspepsia revealed a decreasing prevalence with decreasing CD4+ cell counts, being significantly lower in those with CD4+ cells <50/mm$^3$ as compared to those with counts ≥50/mm$^3$ (57.8% vs. 89.2%, p=0.018). The prevalence of *H. pylori* in HIV seropositive patients without oesophageal candidiasis was stratified according to CD4+ cell counts. The prevalence of *H. pylori* decreased with decreasing CD4+ cell counts, being significantly lower at CD4+ cell counts of >50 as compared to CD4+ cell counts ≥50 cells mm$^3$ (42.8% vs. 88.2%, p=0.038)

**Table 1**

*H. pylori prevalence stratified and compared according to CD4+ cell counts in HIV seropositive patients (n=51)*

| Groups according to CD4 cell count cut-off value | *H. Pylori* prevalence (%) [95% CII] | p-value |
|---|---|---|
| CD4 cell count >200 cells/mm$^3$ (n=12) | 83.3 [54.9-97.1] | 0.706 |
| CD4 cell count <200 cells/mm$^3$ (n=39) | 71.1 [56.3-84.2] | |
| CD4 cell count >100 cells/mm$^3$ (n=19) | 84.2 [62.8-95.8] | 0.323 |
| CD4 cell count <100 cells/mm$^3$ (n=32) | 68.7 [51.3-82.9] | |
| CD4 cell count >50 cells/mm$^3$ (n=30) | 86.6 [70.9-95.6] | 0.024 |
| CD4 cell count <50 cells/mm$^3$ (n=21) | 57.1 [35.7-76.6] | |

The prevalence of H. pylori was high in patients with gastric ulcers (100%) and duodenal ulcers (88.2%). HIV positive patients with a normal stomach had a significantly lower prevalence of *H. pylori* (64.2%) compared to HIV negative patients (91.6%) (p=0.019).

Oral lesions were present in 35 (67.6%) HIV seropositive patients. Other oral lesions, oral hairy leukoplakia and oral ulcers, were present in seven (13.5%) of these patients (Table 2). Oesophageal lesions were significantly more common in HIV seropositive patients compared to HIV seronegative patients. Oesophageal candidiasis was present in 27 (over 50%) HIV seropositive study patients, while oesophageal ulcers and erosions were present in 13 (25%) patients.

**Table 2**

*Oral and oesophageal findings in study patients*

| Lesion | HIV positive (n=52) No. (%) | HIV negative (n=52) No. (%) | P-value |
|---|---|---|---|
| Oral thrush | 24 (46.2) | 0 (0) | <0.001 |
| Oral KS | 4 (7.7) | 0 (0) | 0.118 |
| Other oral lesions | 7 (13.5) | 0 (0) | 0.013 |
| Normal oesophagus | 16 (21.7) | 37 (71.1) | <0.001 |
| Candidiasis | 27 (51.9) | 0 (0.0) | <0.001 |
| Erosions | 5 (9.6) | 0 (0.0) | 0.057 |
| Ulcers | 8 (15.4) | 0 (0.0) | 0.060 |
| Nodules | 2 (3.8) | 0 (0.0) | 0.248 |

Diverse endoscopic appearances of *Candida* were observed. Severe candidiasis involving the entire oesophagus and significantly narrowing the lumen was present in six (11.5%) patients. *Candida* was associated with erosions in five (9.6%) HIV seropositive patients. Erosions were limited to the lower third of the oesophagus. Two patients with *Candida* also had linear oesophageal ulcers involving the lower third of the oesophagus. Twenty out of 27 (74%) HIV seropositive patients with oral thrush had oesophageal candidiasis. Oesophageal candidiasis occurred independently without oral thrush in seven (26%) patients. Oral thrush and oesophageal candidiasis were significantly associated (p<0.001) and also strongly correlated (phi statistic = 0.659).

Oesophageal ulcers were present in eight (15.4%) HIV seropositive subjects. The presentations were also diverse ranging from linear ulcers involving the lower third of the oesophagus, typical punched out ulcers of HSV to scattered large patches of ulcerations involving the entire oesophagus. Oesophageal nodules that were violaceous, raised and suspicious of Kaposi's sarcoma were present in two HIV positive patients.

Gastritis was the most frequent lesion seen in the stomach (Table 3). Gastric nodules were present in five (9.6%) HIV seropositive patients as compared to none in HIV seronegative patients. They were associated with ulcerations in two (3.8%) HIV seropositive subjects. Gastric nodules were suggestive of KS in four (7.8%) cases. Bile reflux was a frequent endoscopic finding in both groups. Duodenitis was the most common duodenal lesion seen, followed by duodenal ulcers.

**Table 3**

*Endoscopic gastric and duodenal findings in study patients (n=104)*

| Lesion | HIV positive (n=52) No. (%) | HIV negative (n=52) No. (%) | P-value |
|---|---|---|---|
| Normal stomach | 28 (53.8) | 24 (46.2) | 0.557 |
| Fundal gastritis | 1 (1.9) | 0 (0.0) | * |
| Antral gastritis | 7 (13.5) | 11 (21.1) | 0.300 |
| Haemorrhagic gastritis | 1 (1.9) | 4 (7.6) | 0.359 |
| Global gastritis | 5 (9.6) | 8 (15.3) | 0.374 |
| Gastric ulcer¶ | 4 (7.6) | 2 (3.8) | 0.678 |
| Gastric nodules | 5 (9.6) | 0 (0.0) | 0.057 |
| Bile reflux | 8 (15.3) | 7 (13.4) | 1.000 |
| Normal duodenum | 36 (69.2) | 29 (55.7) | 0.314 |
| Duodenitis | 8 (15.4) | 12 (23.0) | 0.456 |
| Duodenal ulcer | 7 (13.5) | 10 (19.2) | 0.597 |

*Numbers too small for statistical comparison
¶Four benign and two malignant (one Kaposi's sarcoma and one lymphoma, both in HIV seropositive patients)

All endoscopic abnormalities were stratified according to CD4+ cell counts. There were significantly more oesophageal abnormalities at CD4+ cell counts of <200/mm$^3$ as compared to CD4+ cell counts ≥200 cells/mm$^3$ (79% at CD4+ <200/mm vs. 31% at CD4+ ≥200 p>0.001). The difference in the prevalence of endoscopic gastric and duodenal abnormalities was not significant at CD4+ cell counts of <200/mm$^3$ as compared to CD4+ cell counts of ≥200/mm$^3$ (Figure 1).

The most frequent histologic diagnosis, chronic gastritis, was present in 98 (94.2%) patients. Oesophagitis, was present in 45 (86.5%) HIV seropositive patients as compared to 27 (51.9%) HIV seronegative patients (p<0.001). Viral aetiologies were identified in six (11.5%) and malignancies in four (7.6%) HIV seropositive patients.

*Candida* was identified on histology in 19 of the 27 (74%) subjects endoscopically thought to have oesophageal candidiasis (Table 4). The correlation between *Candida* on histology and endoscopic candidiasis was strong and statistically significant (p<0.001, kappa test= 0.754).

CMV was associated with three (37.5%), *Candida* with two (25%) and KS with one (12.5%) of the oesophageal ulcers. Two (25%) of oesophageal ulcers remained undiagnosed. Of the five patients with oesophageal erosions, three were associated with *Candida*, one with CMV and one with HSV. *Candida* and HSV were identified in two patients on histology despite normal oesophageal endoscopy. Histology confirmed KS in one patient with oesophageal nodules (n=2), while gastric KS was confirmed in two patients and gastric lymphoma in one patient with gastric nodules (n=5).

**Figure 1**

*Stratification of Upper GI endoscopic findings by CD4 cell counts and GI region (n=103)*

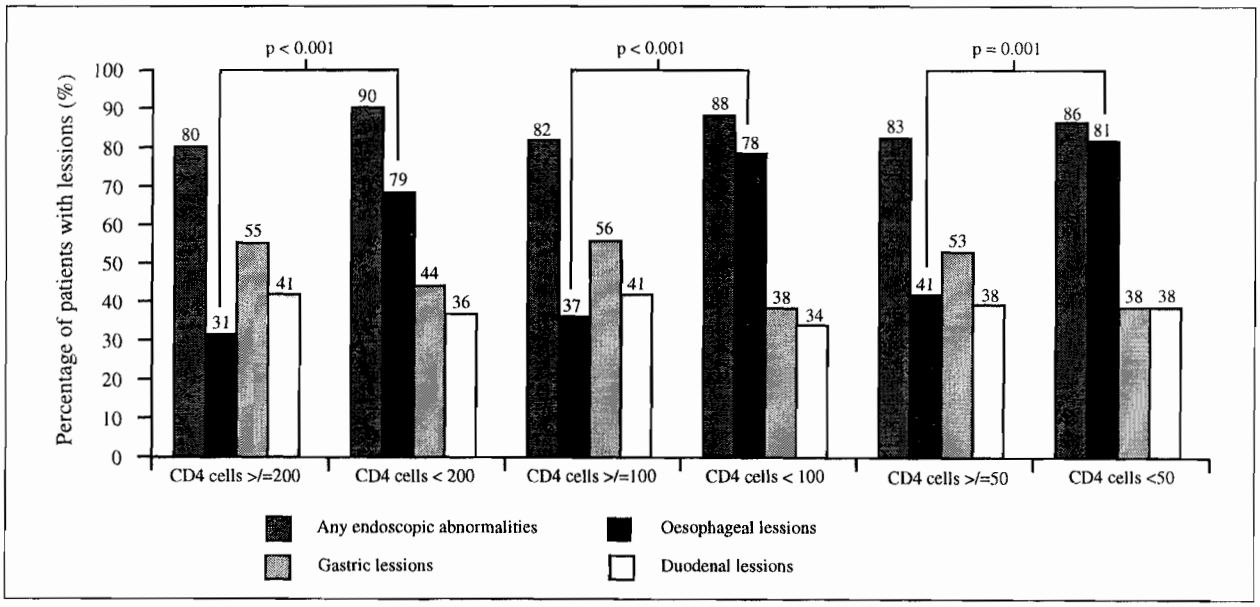


**Table 4**

*Histological findings in study patients (n=104)*

| Histology | | HIV positive (n=52) No. (%) | HIV negative (n=52) No. (%) | P-value |
|---|---|---|---|---|
| | Oesophagitis* | 45 (86.5) | 27 (51.9) | <0.001 |
| | *Candida* | 20 (38.5) | 0 (0.0) | <0.001 |
| | CMV oesophagitis | 4 (7.7) | 0 (0.0) | 0.118 |
| Oesophagus | HSV oesophagitis | 2 (3.8) | 0 (0.0) | 0.495 |
| | Bacterial oesophagitis | 3 (5.8) | 0 (0.0) | 0.241 |
| | Oesophageal KS | 1 (1.9) | 0 (0.0) | 1.000 |
| | Chronic gastritis | 46 (89.5) | 52 (100.0) | 0.067 |
| | Gastric KS | 2 (3.8) | 0 (0.0) | 0.495 |
| Stomach | Gastric lymphoma | 1 (1.9) | 0 (0.0) | 1.000 |
| | Bacterial gastritis | 1 (1.9) | 0 (0.0) | 1.000 |
| Duodenum | Duodenitis | 26 (50.0) | 24 (46.0) | 0.695 |

*Indicates presence of inflammatory changes on histology

## DISCUSSION

This was a prospective study that determined the prevalence of *H. pylori* and pattern of upper GIT endoscopic findings in HIV seropositive patients at KNH. Most of the current data on *H. pylori* prevalence and upper GIT endoscopic findings in HIV seropositive patients have been obtained from studies done in the western countries(7-11), with only one report on upper GIT endoscopic findings in AIDS patients from Uganda(12).

The overall prevalence of *H. pylori* in our study patients was 78.8%. The prevalence of *H. pylori* in HIV seropositive patients was 73.1% and in HIV seronegative patients was 84.6%. The prevalence of *H. pylori* was lower but not significantly slow in the HIV seropositive cases as compared to the HIV seronegative control group.

Stratification of *H. pylori* prevalence according to lower CD4+ cell counts in HIV seropositive patients revealed a decreasing prevalence with decreasing CD4+ cell counts and lowest prevalence of 57.1% was observed at CD4+ cell count of less than 50 cells/$mm^3$ (p=0.024). The trend in decreasing *H. pylori* prevalence with decreasing CD4+ cell counts remained on exclusion of patients with endoscopic candidiasis from the analysis, suggesting that oesophageal candidiasis was not a factor accounting for the lower *H. pylori* prevalence in HIV seropositive patients. Prevalence of *H. pylori* at CD4+ cell counts of<100 cells/$mm^3$ was 43% in the series by Fabris *et al* (11). Other series have also reported a lower prevalence at lower CD4+ cell counts, 13% and 33% in series by Cacciarelli *et al.*,(13) and Vaira *et al.*,(9 ) respectively.

The variability in overall prevalence rates might be related to the epidemiological distribution of *H. pylori* and the different inclusion criteria for patient enrolment. Most similar studies were carried out in the western countries where the population prevalence of *H. pylori* is lower than in developing countries(6).

Overall, all these studies including ours, report a lower *H. pylori* prevalence with decreasing CD4+ cell count. Eighty-two percent of the patients in the series by Fabris *et al*(11) had been on antibiotics in the previous six months and the *H. pylori* prevalence in their series varied inversely with previous antibiotic use. Patients who had been on antibiotics in the previous two weeks were not recruited in our study, but antibiotic use could have been under-reported in HIV seropositive patients. HIV seropositive patients who are in the advanced stages of their disease are usually on multiple medications and antibiotic use for intercurrent illnesses at some point in their disease could have resulted in the lower *H. pylori* prevalence in HIV seropositive patients in all these studies.

In a study on T-cell subsets in *H. pylori*-associated gastritis, Dusch and colleagues(14) reported that CD4+ gastric mucosal lymphocytes selectively accumulate in *H. pylori* associated chronic antral gastritis. It is possible that patients with low serum CD4+ cell counts are incapable of accumulating high levels of intra-epithelial gastric mucosal CD4+ cells required for *H. pylori* proliferation. It has also been noted that AIDS patients tend to develop hypochlorhydria(15). An acidic environment is important for *H. pylori* colonisation and impaired acid secretion has been implicated for the low *H. pylori* prevalence in HIV seropositive patient population. We did not determine the gastric pH in our study population, which would have assisted in confirming this hypothesis.

As found in other studies, (9,13) the prevalence of *H. pylori* in HIV seropositive patients with CD4+ cell counts >200 cells/mm$^3$ was essentially equal to that in the HIV seronegative control group.

The prevalence of peptic ulcer disease (PUD) was 21.6% in HIV seropositive patients compared to 23% in the HIV seronegative control group. The prevalence of *H. pylori* was very high in the patients with PUD. Fifty percent of the gastric ulcers in HIV seropositive patients were attributed to KS and GIT lymphoma in our series. All gastric ulcers and 88.2% of the duodenal ulcers were associated with *H. pylori* in our study. Studies in the western countries report a lower prevalence of PUD in HIV seropositive patients. Peptic ulcer disease was found in 38% of Kenyan patients with dyspepsia by Ogutu *et al* (5) study at KNH. The prevalence of peptic ulcer disease has generally been reported to be higher in situations of low socio-economic status and concrete life difficulties. This could have accounted for the higher prevalence of PUD in our study compared to the western studies(16).

Oral lesions were present in 66.9% of the HIV seropositive patients. Oral thrush was present in 46% in our series compared to 36-92% in similar studies. Varied prevalence of oral lesions has been observed in different studies. This difference in prevalence could have resulted from different inclusion criteria, severity of immunosuppresion and previous use of oral topical anti-fungal therapies.

Oesophageal candidiasis accounted for 51.9% of the oesophagitis and was confirmed using histology in 74% of the cases in our series. Our results are consistent with a previous published report by Bonacini *et al.*,(7). They, however, also used pre-biopsy brushings in the diagnosis, which confirmed 95% of the oesophageal candidiasis.

Oral thrush had a positive predictive value of 83.3%, and a negative predictive value of 91.3%, for diagnosis of oesophageal candidiasis. Oral thrush has been considered to be an excellent marker of oesophageal *Candidiasis* and our study confirms this(17). However oesophageal candidiasis can occur without oral thrush as it did in 26% of our patients. Therefore, consideration may be given for HIV seropositive patients presenting with upper GIT symptoms, particularly dysphagia and odynophagia, and found to have oral thrush to be offered empirical therapy for presumptive oesophageal candidiasis. Such a strategy could be prospectively evaluated for its efficacy and cost-effectiveness.

Erosions and ulcers were identified in 25% of our patients. In our study viral pathogens were identified in 11.5%. Despite the higher prevalence of erosions and ulcers in our study population the yield of viral pathogens was low compared to studies done in the west. Bonacini *et al.*,(7), using histology and viral cultures found viral infections in 36% of patients, and positive viral cultures resulted in their high yield of viral pathogens. Wilcox *et al.*,(18) identified viral pathogens in 50% of their cases of oesophageal ulcers in HIV seropositive patients. They took six or more biopsies from each ulcer and this increased their yield of viral pathogens. We were unable to take six or more biopsies from each ulcer or do viral cultures because of logistical constraints and this could have resulted in the lower yield of viral pathogens in our study.

Viral pathogens accounted for six cases of the erosions and ulcers in our series with CMV being the aetiological cause of three of the ulcers and one of the erosions. HSV, however, was not isolated from the typical punched out ulcers, but was found to be the aetiology of one of the erosions. *Candida* was associated with three of the erosions and two of the ulcers in our study population. Two cases of oesophageal ulcers remained undiagnosed. The yield of histology in identification of viral pathogens was 42% in our study compared to 46% in the series by Bonacini *et al* (7). Unlike previous studies from Africa our findings confirm the presence of viral pathogens as the aetiologic cause of oesophagitis in this region and reinforce the importance of endoscopy and biopsy in HIV seropositive patients.

Two of the patients had normal endoscopy but histological features of *Candida* and HSV were seen. It is possible that these represented early stages of fungal or viral infection in which organisms were present in

insufficient quantities to result in endoscopic abnormalities.

Two HIV seropositive patients had nodules in the oesophagus, five had nodules in the stomach and one in the duodenum. Oesophageal nodules were suspicious of oesophageal KS in both our patients and was confirmed using histology to be KS in one. Gastric nodules were associated with ulcers in two of the patients. Lymphoma was found in one case and KS confirmed in two of the five cases of gastric nodules using histology. The other two cases of gastric nodules were suspected to be KS but not confirmed using histology. Due to the submucous nature of KS, mucosal biopsy detects less than 50% of the suspected cases(19). This could have resulted for two of the suspected cases of KS being missed on mucosal biopsy in our study.

Overall, *H. pylori* prevalence was lower but not significantly lower in HIV seropositive patients as compared to the HIV seronegative control group and decreased with decreasing CD4+ cell counts in these patients. Oesophageal candidiasis was the most common endoscopic finding in HIV seropositive patients and was frequently associated with oral thrush

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