

Oropharyngeal candidiasis and *Candida* colonization in HIV positive patients in northern India

Vijeta Maurya^{1,2}, Ashutosh Srivastava¹, Jyoti Mishra³, Rajni Gaiind², Rungmei S. K. Marak⁴, Anil Kumar Tripathi⁵, Mastan Singh¹, Vimala Venkatesh¹

¹Department of Microbiology, Chhatrapati Shahuji Maharaj Medical University, Lucknow, UP, India

²Department of Microbiology, Vardhaman Mahavir Medical College and Safdarjung Hospital, New Delhi, India

³Department of Pathology, Chhatrapati Shahuji Maharaj Medical University, Lucknow, UP, India

⁴Department of Microbiology, Sanjay Gandhi Post Graduate Institute of Medical Sciences, Lucknow, UP, India

⁵Department of Medicine, Chhatrapati Shahuji Maharaj Medical University, Lucknow, UP, India

Abstract

Introduction: Oropharyngeal candidiasis (OPC) is the most common opportunistic fungal infection reported in human immunodeficiency virus (HIV) positive patients worldwide. This prospective study was undertaken to investigate OPC and *Candida* colonization (CC) and their correlation with CD4⁺ cell counts and antiretroviral therapy (ART) in HIV-positive patients.

Methodology: In total, 190 HIV-positive patients were enrolled for study in three groups as follows: Group A, 90 patients without ART; Group B, 100 patients undergoing treatment with ART; and Group C, 75 HIV-negative control patients. All HIV patients underwent clinical examination and were subjected to CD4⁺ cell counts. Swabs were collected from the oral cavity of all individuals and plated on Sabouraud's dextrose agar. Identification of *Candida* species was performed by conventional methods.

Results: *Candida* species were isolated in 84/190 (44.2%) and 20/75 (26.6%) of the HIV-positive subjects and controls respectively (p<0.01). OPC was noted in 21/190 (11%) of the HIV-positive patients. *Candida albicans* was the most frequently isolated species. Patients with CD4⁺ cell counts ≤ 200 cells/mm³ were significantly (p<0.001) more frequently colonized (37/63; 58.7%) and infected (18/21; 85.7 %) with *Candida* species. *Candida* species was seen in patients with CC and OPC with CD4⁺ cell counts between 201 and 500 (21/63; 33.4% vs 3/21; 14.3%) and > 500 cell/mm³ (5/63; 7.9% versus 0/21 0%) respectively.

Conclusion: OPC and *Candida* colonization occur more frequently in HIV-positive patients with CD4⁺ cell counts ≤200 cell/mm³. ART significantly reduces OPC. *C. albicans* is the most frequently isolated species in both OPC and colonization, suggesting endogenous infection.

Key words: Oropharyngeal candidiasis (OPC); *Candida* colonization; antiretroviral therapy; CD4⁺ cells; HIV

J Infect Dev Ctries 2013; 7(8):608-613. doi:10.3855/jidc.2801

(Received 18 June 2012 – Accepted 30 September 2012)

Copyright © 2013 Maurya *et al.* This is an open-access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

Introduction

The oral cavity is colonized by *Candida albicans* or other *Candida* species in 40% to 60% of healthy persons [1]. In immunocompetent individuals, *Candida* species are mostly transient flora. Many factors contribute to the development of oropharyngeal candidiasis (OPC) including malnutrition, poor oral hygiene, dental malocclusion, and immunosuppression [2]. The current epidemic of human immunodeficiency virus (HIV) is a major cause of immunosuppression. According to the Joint United Nations Programme (2009) on HIV/AIDS [3] and the National AIDS Control Organization (2008) [4], there are an estimated 2.4 million cases of HIV infection worldwide and 22.7 per 100,000 in India respectively. The World Health Organization (WHO) predicts that

India will have one of the largest populations of HIV/AIDS patients worldwide in coming decades. OPC has been described as the most frequent opportunistic fungal infection among HIV-positive patients and it has been estimated that more than 90% of HIV-positive patients develop this often debilitating infection at some time during the progression of their disease [5,6]. The presence of *Candida* species in the oral cavity of HIV-positive patients is an indicator of subsequent development of OPC [7]. HIV-positive patients experience recurrent episodes of OPC and oesophageal candidiasis. As immunodeficiency progresses, these patients may also receive multiple courses of antifungal drugs contributing to antifungal resistance. In these patients, antifungal agents are also

less efficacious and take longer to achieve a clinical response [8].

Until recently, *C. albicans* was reported as the predominant species in OPC but recent publications suggest that non-*albicans Candida* species are now emerging [1,9]. There is a paucity of microbiological studies on OPC and *Candida* colonization (CC) in HIV patients from India; therefore, the present study was conducted to determine the profiles of CC and OPC and their correlation with CD4⁺ cell counts and antiretroviral therapy (ART) in HIV patients.

Methodology

Study population

This hospital-based prospective observational study was conducted between August 2007 and July 2008. Patients attending the ART Centre and the Integrated Counseling and Testing Centre (ICTC) at Chhatrapati Shahuji Maharaja Medical University (CSMMU), Lucknow, were enrolled for the study. A total of 190 HIV-positive patients were enrolled after they provided informed written consent. Seventy-five HIV-negative subjects in the same age group and socioeconomic status were enrolled as study controls. The study was approved by the institutional ethical clearance committee. Individuals with diabetes mellitus or other systemic diseases, pregnant women, smokers, dentures/orthodontic device users, and those on antibiotics and/or on antifungal treatment in the preceding three months were excluded.

HIV serostatus of the study group was determined using a commercially available enzyme linked immunosorbent assay (ELISA) (HIV-1 Enzaids, Span Diagnostics Ltd, Udhna, Surat, India) and three rapid antibody tests manufactured by CombAids-RS Span Diagnostics Ltd, Surat, India; Retrocheck HIV, QUAL proDiagnostics, Goa, India and Acon Biotech ProLink, San Diego, CA, USA, following NACO recommended algorithms [10]. A CD4⁺ cell count using the Fluorescent Antibody Cell Sorter (FACS, Becton Dickinson, Singapore, BD) Count system was performed in all enrolled HIV-positive patients.

Clinical and microbiological assessment of subjects

An oral examination was performed and suspected lesions were clinically evaluated by the Faculty of Dental Sciences. *Candida* colonization was defined as isolation of *Candida* species from the oral cavity without the presence of oral lesions. All patients who had oral lesions and from whom *Candida* species were isolated were considered to have OPC. Recent CD4⁺ cell counts and a history of intake of ART were noted

for all HIV-positive patients. A single oral swab was collected from each study participant by passing a sterile swab firmly across the buccal mucosa, floor of mouth, dorsal surface of the tongue in cases of asymptomatic patients, and from the base of the oral lesion in cases of symptomatic patients. Swabs were cultured on Sabouraud's dextrose agar (SDA) with chloramphenicol 0.5g/l, then incubated at 37°C and observed daily for seven days. Pure growth of *Candida* species was considered for analysis. *Candida* was identified by conventional tests and species identification was performed using the germ tube test, growth on CHROMagar *Candida* Medium (HiMedia, Mumbai, India), and sugar assimilation tests [11,12].

Statistical analysis was conducted by using the chi-square test. Data were analyzed with SPSS software version 20 (IBM SPSS, Chicago, USA).

Results

In the present study, 190 HIV-positive patients were designated into two groups: group A (n = 90) without ART and group B (n = 100) with ART. The demographic data and clinical profile of these patients is shown in Table 1. The median duration of ART was two years (range, 1.5 months to 6 years). Overall, *Candida* species was isolated from the oral cavity in 84/190 (44.2%) HIV-positive patients and in 20/75 (26.6%) of HIV-negative patients. The difference was significant (p < 0.01). Of the 84 HIV-positive patients with *Candida* species, 41 (45.5%) were from Group A and 43 (43.0%) were from the Group B. Twenty-one (11%) HIV-positive patients had oral lesions in the oropharyngeal cavity and *Candida* species were isolated on culture.

Five different clinical presentations of OPC were noted on examination in HIV-positive patients (Table 1). Pseudomembranous candidiasis was the most common clinical presentation of OPC. *C. albicans* was the most frequent species isolated in both HIV-positive (76/84; 90.5%) and HIV-negative patients (20/20; 100%). It was also the most frequently isolated yeast (95.2%) in all clinical presentations of OPC. Non-*albicans Candida* was isolated in only one patient with OPC (1/21; 4.76% *C. tropicalis*) and seven patients with colonization (7/63; 11.1%). The correlation between oral CC and OPC with CD4⁺ cell counts in HIV-positive patients is shown in Table 1. The range of CD4⁺ cell counts in colonized and non-colonized HIV-positive patients was 2 to 776 cells/mm³ (median = 183) and 8 to 756 cells/mm³ (median = 247), respectively. CC was seen in 37 (58.7%), 21 (33.4%) and 5 (7.9%) HIV-positive

Table 1. Profile of HIV patients with *Candida* colonization and infection and its correlation with ART and CD4⁺ count

Demographic Data	HIV Positive patients			Group C HIV Negative patients N = 75
	Total N = 190	Group A N = 90	Group B N = 100	
Male	116 (61)	48 (53.3)	68 (68)	57 (68)
Age range (Years)	6-55	20-55	6-50	6-64
Total <i>Candida</i> isolates	84 (44.2)	41 (45.5)	43 (43)	20 (26.6) *
Oral <i>Candida</i> Colonization (CC)	63 (33.1)	25 (27.8)	38 (38)	20 (26.7)
CD4⁺ cell Count				CD4 ⁺ count not done
≤ 200				
201-500	37 (58.7)	14 (56)	23 (60.5)	
> 500	21 (33.4)** 05 (7.9)**	9 (36) 02 (8)	12(31.6) 03 (7.9)	
Oro-pharyngeal candidiasis (OPC)	21 (11)	16 (17.8)	5 (5)***	0
CD4⁺ cell count				
≤ 200	18 (85.7) **	13 (81.2)	5 (100)	
201-500	03 (14.3) **	03 (18.8)	0	
> 500	0	0	0	
Types of oral lesions				
Pseudomembranous (P)	16 (76.2)	12 (75)	4 (80)	
Erythematous (E)	0	0	0	
Angular chelitis (AC)	2 (9.5)	1 (6.2)	0	
AC +E	1 (4.8)	1 (6.2)	0	
AC + E + P	2 (9.5)	2 (2.2)	1 (20)	
<i>Candida</i> species isolated				
<i>C. albicans</i>	76 (90.5)	40 (97.6)	36 (83.8)	20 (100)
<i>C. tropicalis</i>	1 (1.2)	0	1 (2.3)	-
<i>C. glabrata</i>	4 (4.8)	0	4 (9.3)	-
<i>C. krusei</i>	1 (1.1)	0	1 (2.3)	-
<i>C. kefyr</i>	2 (2.4)	1 (2.4)	1 (2.3)	-

Note: All figures in parentheses represent percentages.

*p < 0.01 when compared with total HIV patients

**p < 0.001 when compared with CD4⁺ cell count ≤ 200

***p < 0.05 when compared to Group A

patients with CD4⁺ cell counts ≤ 200cells/mm³, between 201 and 500 cells/mm³ and more than 500cells/mm³, respectively (p < 0.001). The majority of HIV-positive patients with OPC (18/21; 85.7%) had a CD4⁺ cell count ≤ 200 cell/mm³ (p < 0.001). OPC was not seen in patients with CD4⁺ cell counts greater than 500 cells/mm³. In the present study, OPC was found to be significantly higher in patients in Group A (16/90; 17.8%) as compared to Group B (5/100; 5%),

(P < 0.05). Antiretroviral therapy did not influence the status of colonization.

Discussion

Asymptomatic carriage of *Candida* species in the oral cavity is found irrespective of the immune status of individuals. Many studies have been conducted on oral CC in healthy and immunocompromised individuals. Due to differences in the sample collection techniques used, time and frequency of

sampling, yeast assessment methods, and the study population, results from the studies are not comparable. In addition, CC rate can be affected by several factors such as hospitalization, abnormal nutrition, and smoking [2]. Oral CC in HIV-positive asymptomatic patients has been reported and is known to be higher than in patients in other risk groups such as diabetes mellitus or other systemic disease [13]. Similarly, in our study, the isolation of *Candida* was significantly higher in the HIV-positive group compared to that in the controls. The prevalence of OPC (11%) and CC (33%) in HIV-positive patients in the present study was lower compared to that in other studies (62.6% to 81%) [14-17]. The reason for the low isolation rate in the present study could be explained by single sampling performed in this study as compared to multiple sampling in other investigations.

OPC infections are the most common of opportunistic infections in HIV-positive patients, occurring in up to 90% of patients during the course of the disease [13]. In India, OPC is reported to be the second most common opportunistic infection in HIV-positive patients [18]. Three clinical presentations of OPC have been commonly reported in HIV-positive patients: pseudomembranous, erythematous and angular cheilitis [17,19]. In our study, acute pseudomembranous candidiasis (exudative) was the

most common form of OPC (Table 1), while angular cheilitis may be part of vitamin deficiency superimposed with *Candida* infection seen in 9.5% of the patients. These results are in concordance with those result of several other studies [16,20]. *C. albicans* was also the predominant species isolated from the oral cavities of the patients in the control group.

In HIV-positive patients with OPC, *C. albicans* is the most frequently identified species; however, non-*albicans Candida* has also been reported recently [20,21]. *C. albicans* was the most frequent species isolated from colonized and infected HIV-positive subjects (90.5%) in our study (Table 1). In the present study, a relatively small proportion (9.5%) of isolates were non-*albicans Candida*. In a study conducted in Italy, Barchiesi *et al.* described an increase in the frequency of isolation of non-*albicans Candida* species from 3% to 4% of isolates in 1988/1989 to 16% to 18% of isolates in 1990/1991 [15]. Similarly, in another Italian study, Morace *et al.* (1990) found that 25% of the yeast species isolated from persons with AIDS were non-*albicans Candida* [22]. In Spain, Masia *et al.* evaluated 153 HIV-positive patients and found that 21% of these patients had non-*albicans Candida*, the most common being *C. glabrata* [23]. Table 2 shows the distribution of *Candida* species isolated from HIV-positive patients with OPC from

Table 2. Studies on OPC in HIV infected patients from India in last decade

Study Group	Duration of study	Patient size and characteristics	Predominant species
Delhi (Lattif AA <i>et al.</i>) [24]	1999-2001	75 (OPC)	<i>C. albicans</i> (86%) <i>C. parapsilosis</i> (8%) <i>C. glabrata</i> and <i>C. krusei</i> (3%) <i>C. dubliniensis</i> (0%)
Chennai (Menon T <i>et al.</i>) [25]	2001	46 (Infected or colonized)	<i>C. albicans</i> (73.9%) <i>C. tropicalis</i> (21.7%) <i>C. dubliniensis</i> (0%)
Mumbai (Baradkar VP <i>et al.</i>) [21]	2005-2008	40 (OPC)	<i>C. albicans</i> (70%) <i>C. parapsilosis</i> (15%) <i>C. glabrata</i> (7.5%) <i>C. tropicalis</i> (5%) <i>C. dubliniensis</i> (2.5%)
Karnataka (Nadagir SD <i>et al.</i>) [26]	2008	132 Patients 135 Isolates(OPC)	<i>C. albicans</i> (66.6%) Non <i>albicans Candida</i> (33.3%) <i>C. dubliniensis</i> (48.9%)
Lucknow Present study	2007-2008	21 (OPC)	<i>C. albicans</i> (95.2%) <i>C. tropicalis</i> (4.76%) <i>C. dubliniensis</i> (0%)

OPC: Oropharyngeal candidiasis

India in the last decade [21,24-26]. The findings showed that in India *C. albicans* continue to be the predominant pathogen and non-*albicans Candida* has been reported in approximately 14% to 30% of patients.

The reports of an association between CD4⁺ cell counts and OPC/CC are also contradictory; Costa *et al.* did not find a significant correlation between CC and CD4⁺ cell counts, as 16.25%, 61.2% and 22.6% of the patients in their study had CD4⁺ cell counts \leq 200, 201 to 500 and more than 500 respectively [27]. Little correlation was also observed by Barchiesi and colleagues, with a median CD4⁺ cell count of 397 cell/mm³ and 442 cells/mm³ in colonized and non-colonized their patients, respectively [28]. Schoofs *et al.*, however, reported a significant relationship between CC and CD4⁺ cell counts less than 200 cell/mm³ [29]. Fong and colleagues also found a strong correlation between asymptomatic CC, the development of thrush, and CD4⁺ cell counts [30]. OPC in HIV-positive persons keeps recurring as the immunity decreases. In our study, CC was significantly higher in the group with CD4⁺ cell counts \leq 200 cell/mm³ ($p < 0.001$), suggesting that the group is prone to develop OPC. Prevention of opportunistic infections in patients with HIV is important because in all HIV-infected individuals, the risk of infection increases as the absolute CD4 T-lymphocyte count falls. Data from prospective controlled trials indicate that fluconazole prophylaxis can reduce the risk for mucosal (*e.g.*, oropharyngeal, esophageal, and vaginal) candidiasis among patients with advanced HIV disease. However, routine primary prophylaxis is not recommended because mucosal disease is associated with very low attributable mortality, acute therapy is highly effective, prophylaxis can lead to disease caused by drug-resistant species, prophylactic agents can produce drug interactions, and prophylaxis is expensive [31]. It is recommended that the HIV-infected asymptomatic cases with CD4⁺ cell counts \leq 200 cell/mm³ should be screened for oral candidiasis to improve the quality of life.

ART has been associated with dramatic decreases in the rate of HIV-related opportunistic infections. Our study suggests that ART does not influence oral CC, but it significantly prevented the development of OPC. In a follow-up study, Yang *et al.* reported that ART was only marginally effective in eliminating colonization [32]. Similarly, Sanchez-Vargas *et al.* reported that ART did not influence colonization. The authors observed that 36.1% of patients undergoing

ART suffered from OPC compared to 45.9% of patients not receiving ART [1].

Conclusion

Oral *Candida* colonization and invasive infection occur more frequently in HIV-positive patients and is significantly more common in patients with CD4⁺ cell counts \leq 200 cell/mm³. ART significantly reduces OPC. *C. albicans* continues to be the most frequently isolated species in both OPC and CC, suggesting endogenous infection.

References

1. Sanchez-Vargas LO, Ortiz-Lopez NG, Villar M, Moragues MD, Aguirre JM, Cashat-Cruz M, Lopez-Ribot JL, Gaitán-Cepeda LA, Quindos G (2005) Point prevalence, microbiology and antifungal susceptibility patterns of oral *Candida* isolates colonizing or infecting Mexican HIV/AIDS patients and healthy persons. *Rev Iberoam Micol* 22: 83-92.
2. Scully C, el-Kabir M, Samaranyake LP (1994) *Candida* and oral candidiasis: a review. *Crit Rev Oral Biol Med* 5: 125-157.
3. Joint United Nations Programme on HIV/AIDS (UNAIDS) (2010) AIDS epidemic update 2010.
4. National AIDS Control Organisation (NACO) (2010) Department of AIDS control, Ministry of Health and Family Welfare India, Annual Report: 2009-2010.
5. Samaranyake LP, Holmstrup P (1989) Oral candidiasis and human immunodeficiency virus infection. *J Oral Pathol Med* 18: 554-564.
6. Samaranyake LP (1992) Oral mycoses in HIV infection. *Oral Surg Oral Med Oral Pathol* 73: 171-180.
7. Gugrani HC, Becker K, Fegeler W, Basu S, Chattopadhyaya D, Baveja U, Satyanarayana S, Kalghatgi T, Murlidhar A (2003) Oropharyngeal carriage of *Candida* species in HIV-infected patients in India. *Mycoses* 46: 299-306.
8. Darouiche RO (1998) Oropharyngeal and esophageal candidiasis in immunocompromised patients: treatment issues. *Clin Infect Dis* 26: 259-272.
9. Badiie P, Alborzi A, Davarpanah M, Shakiba E (2010) Distributions and antifungal susceptibility of *Candida* species from mucosal sites in HIV positive patients. *Arch Iran Med* 13: 282-287.
10. National AIDS Control Organisation (NACO) (2007) NACO Guidelines on HIV Testing.
11. Koneman EW, Allen SD, Janda WM, Schreckenberger PC, Winn Jr WC (1992) Color atlas and textbook of diagnostic microbiology. 4th ed. Philadelphia: JB Lippincott Company. 813-850.
12. Rippon JW (1988) Candidiasis and pathogenic yeasts. In: Martin Wonsiewicz editor. *Medical Mycology*. Philadelphia: WB Saunders. 531-581.
13. Vargas KG, Joly S (2002) Carriage frequency, intensity of carriage, and strains of oral yeast species vary in the progression to oral candidiasis in human immunodeficiency virus-positive individual. *J Clin Microbiol* 40: 341-350.
14. Campisi G, Pizzo G, Milici ME, Mancuso S, Margiotta V (2002) *Candida* carriage in the oral cavity of human immunodeficiency virus-infected subjects. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 93: 281-286.

15. Barchiesi F, Del Poeta M, Morbiducci V, Ancarani F, Scalise G (1993) Turbidimetric and visual criteria for determining the *in vitro* activity of six antifungal agents against *Candida* spp. and *Cryptococcus neoformans*. *Mycopathologia* 124: 19-25.
16. Anil S, Challacombe SJ (1997) Oral lesions of HIV and AIDS in Asia: an overview. *Oral Dis* 1: 36-40.
17. Ranganathan K, Reddy BV, Kumarasamy N, Solomon S, Viswanathan R, Johnson NW (2000) Oral lesions and conditions associated with human immunodeficiency virus infection in 300 south Indian patients. *Oral Dis* 6: 152-157.
18. National AIDS Control Organisation (NACO) (2007) Guidelines for prevention and management of common opportunistic infection/malignancies among HIV infected adult and adolescent
19. Sullivan D, Coleman D (1997) *Candida dubliniensis*: an emerging opportunistic pathogen. *Curr Top Med Mycol* 8: 15-25.
20. Gillespie GM, Marino R (1993) Oral manifestations of HIV infection: a Panamerican perspective. *J Oral Pathol Med* 22: 2-7.
21. Baradkar VP, Kumar S (2009) Species identification of *Candida* isolates obtained from oral lesions of HIV infected patients. *Indian J Dermatol* 54: 385-386.
22. Morace G, Tamburrini E, Manzara S, Antinori A, Dettori GG (1990) Epidemiological and clinical aspects of mycoses in patients with AIDS related pathologies. *Eur J Epidemiol* 6: 398-403.
23. Masiá Canuto MM, Gutiérrez F, Rodero V, Ortiz de la Tabla Durcasse V, Martín González C, Escolano Hortelano CM, Mora Rufete A, Martín Hidalgo A (1999) Epidemiology of yeast colonization and oropharyngeal infection other than *Candida albicans* in patients with HIV infection. *Med Clin* 112: 211-214.
24. Lattif AA, Banerjee U, Prasad R, Biwas A, Wig N, Sharma N, Haque A, Gupta N, Baquer Z.N, Mukhopadhyay G (2004) Susceptibility pattern and molecular type of species-specific *Candida* in oropharyngeal lesions of Indian human immunodeficiency virus-positive patients. *J Clin Microbiol* 42: 1260-1262.
25. Menon T, Umamaheswari K, Kumaraswamy N, Solomon S, Thyagarajan SP (2001) Efficacy of fluconazole and itraconazole in the treatment of oral candidiasis in HIV patients. *Acta Trop* 80: 151-154.
26. Nadgir SD, Chunchanur SK, Halesh LH, Yasmeen K, Chandrasekhar MR, Patil BS (2008) Significance of isolation and drug susceptibility testing of non-*Candida albicans* species causing oropharyngeal candidiasis in HIV patients. *Southeast Asian J Trop Med Public Health* 39: 492-495.
27. Costa CR, Cohen AJ, Fernandes OF, Miranda KC, Passos XS, Souza LK, do Rosario Rodrigues Silva M (2006) Asymptomatic oral carriage of *Candida* species in HIV-infected patients in the highly active antiretroviral therapy era. *Rev Inst Med Trop Sao Paulo* 48: 257-261.
28. Barchiesi F, Maracci M, Radi B, Arzeni D, Baldassarri I, Giacometti A, Scalise G (2002) Point prevalence, microbiology and fluconazole susceptibility patterns of yeast isolates colonizing the oral cavities of HIV-infected patients in the era of highly active antiretroviral therapy. *J Antimicrob Chemother* 50: 999-1002.
29. Schoofs AG, Odds FC, Colebunders R, Ieven M, Goossens H (1998) Cross-sectional study of oral *Candida* carriage in a human immunodeficiency virus (HIV)-seroinfected population: predisposing factors, epidemiology and antifungal susceptibility. *Mycoses* 41: 203-211.
30. Fong I W, Laurel M, Burford-Mason A (1997) Asymptomatic oral carriage of *Candida albicans* in patients with HIV infection. *Clin Invest Med* 20: 85-93.
31. CDC (2009) Guidelines for prevention and treatment of opportunist infections in HIV-infected adults and adolescents: the National Institutes of Health, and the HIV Medicine Association of the Infectious Diseases Society of America. *MMWR* 58 (No.RR-4).
32. Yang YL, Lo HJ, Hung CC, Li Y (2006) Effect of prolonged HAART on oral colonization with *Candida* and candidiasis. *BMC Infect Dis* 6: 8.

Corresponding author

Vijeta Maurya
 Department of Microbiology
 VMMC and Safdarjung Hospital
 Chhatrapati Sahu Ji Maharaja Medical University
 Lucknow, UP
 India
 Telephone: 9971269254
 Fax: 011-91-27123677
 Email: mauryavije@rediffmail.com

Conflict of interests: No conflict of interests is declared