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Malassezia Species Associated Seborrheic Dermatitis and Its Comparison between HIV Positive and Negative Patients

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ABSTRACT

Introduction: *Malassezia* yeasts are lipophilic organisms causing certain skin diseases. Seborrheic dermatitis (SD) is the second most common skin infection caused by *Malassezia* as well as in HIV/AIDS.
Aim: To determine the frequency of association of *Malassezia* species in HIV infected and HIV non-infected patients with Seborrheic dermatitis.
Materials and Methods: The prevalence of Seborrheic dermatitis is 5% in the general population. Hence a sample size of 80 was derived, 40 each of HIV seropositive and HIV seronegative adult patients clinically suspected of having Seborrheic Dermatitis. Specimens were collected by scraping and cellophane tape for KOH and Chicago Sky Blue (CSB) stain, and were cultured on Sabouraud's dextrose agar. Data were analysed using SPSS version 16.0. $P \leq 0.05$ was considered as significant.
Results: Majority of the patients i.e. 46 (57.5%) out of 80 were in the age group of 18-30 years with male preponderance. All HIV positive patients with SD had scaly, greasy, itchy, hypo-pigmented and erythematous lesions, & neck (23) and groin (20) were commonest sites. In 39 HIV positive and 22 HIV negative patients, >2 sites were involved. Majority of the HIV negative patients with SD had scaly (40), itchy (24) and hypo-pigmented lesions (27) & dandruff, and scalp (24) & neck (18) were commonest sites. ($P<0.05$). Twenty HIV positive patients had CD4 count ranging from 200-350 cells/mm^3. *Malassezia* was detected in 38 and 34 HIV positive & negative patients respectively in laboratory diagnosis.
Conclusion: Seborrheic Dermatitis has severe presentation at multiple sites in HIV positive patients as compared to HIV negative patients.

Key Words: *Malassezia*, Seborrheic dermatitis, HIV positive, HIV negative

INTRODUCTION

Malassezia yeasts are lipophilic organisms and have been members of normal human cutaneous flora as well as agents of certain skin diseases. *Malassezia* genus is classified in the order Malasseziales among the Exobasidio-mycetes. It is associated with mild, frequently causing recurrent cutaneous infections and also has been associated with skin and deep invasive infections in immunocompromised patients. [1-2]

Seborrheic dermatitis (SD) is the second most common infection associated with *Malassezia* which is a common chronic inflammatory superficial eczematous

dermatitis, either sub-acute or chronic, characterized by erythematous plaques with dry or oily scales. [3-4]

Seborrheic dermatitis affects 3 to 5% of the global population affecting males more than females. [2] It is seen over areas rich in sebaceous glands, namely the scalp, face, chest, back and flexural areas.[5] A milder variant is dandruff, which is manifested by dry, flaking scales on the scalp.[4]

The exact pathogenesis of adult Seborrheic dermatitis is not known. A combination of hormone levels, weakened immune system, lack of certain nutrients, or nervous system problems may predispose to this infection.[6]

Seborrheic dermatitis is most common skin infection in HIV/AIDS and its reported prevalence ranges between 20 and 40% in HIV-1-seropositive patients and between 40 and 80% in those with AIDS.[7] Its incidence and severity are closely related to the stage of HIV infection and inversely correlate with the absolute CD4 helper T cell counts. [5] Seborrheic dermatitis is more severe and more resistant to therapy in HIV/AIDS patients.[7]

The diagnosis of Seborrheic dermatitis is often done clinically. Early diagnosis can be done by microscopy & culture. Samples are usually collected by direct scraping of skin. The mycological study is most commonly done by potassium hydroxide (KOH) mount and culture.[8]

This study aimed to determine the frequency of association of *Malassezia* species in HIV positive and HIV negative patients with Seborrheic dermatitis.

MATERIALS AND METHOD

A cross-sectional study was carried out at a tertiary care center over a period of 1 year. Institutional and ethical approvals were obtained. Given a prevalence of seborrheic dermatitis of 5% in the general population [1] and 40% in HIV positive patients [7] , and taking a confidence level of 95% and power of 80%, a sample size of 80 was derived for this study, forty each of HIV seropositive and HIV seronegative patients.

Eighty consecutive adult patients (age >18 years) clinically suspected of having Seborrheic dermatitis and presenting to the dermatology clinic during the study period were considered for enrolment. Clinical diagnosis was based on the presence of scaly, greasy lesions. Only those patients whose HIV antibody testing was done in the previous three months were enrolled if willing.

Patients who had been treated with oral or topical antifungal agents in the past one month were excluded. Specimens were collected by scraping and cellophane tape, one each for KOH mount, KOH and CSB mount, and were cultured on Sabouraud's dextrose agar (SDA) with chloramphenicol and gentamicin overlaid with sterile olive oil on tubes and plates of SDA respectively. (Figure 1 & 2)

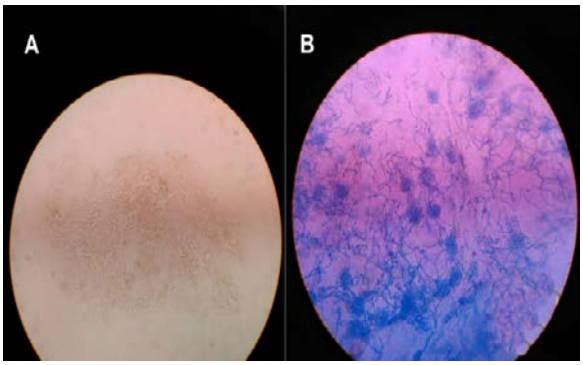

Figure 1. Hyphae and spores exhibiting characteristic appearance of 'Spaghetti and Meatball' appearance in A. KOH mount & B. CSB stain:

Figure 2: Rough folded colonies of *Malassezia* on SDA (Plate and Tube):

Figure 3. Erythematous lesion on face, hypo pigmented scaly lesion affecting the scalp line and dandruff in Seborrheic Dermatitis patient:

STATISTICAL ANALYSIS

Data were analyzed using SPSS version 16.0. Descriptive statistics mean, median, minimum, maximum, and percentages were calculated to describe the demographic data. The categorical variables were compared using Pearson's Chi-square test & McNemar test when appropriate. P value ≤ 0.05 was considered as significant.

RESULT

Table 1: Age distribution:

Age	HIV status		Total (%)
	Positive (%)	Negative (%)	
18-30 years	18 (39.1%)	28 (60.9%)	46 (57.5%)
30-40 years	13 (61.9%)	8 (38.1%)	21 (26.25%)
> 40 years	9 (69.2%)	4 (30.8%)	13 (16.25%)
Total	40	40	80

Majority of the patients i.e. 46 (57.5%) out of 80 were in the age group of 18-30 years. Eighteen (39.1%) HIV positive SD patients and 28 (60.9%) HIV negative SD patients were less than 30 years of age and there was no statistically significant difference between these two groups. (P=0.068)

Table 2: Gender distribution:

Sex	HIV status		Total (%)
	Positive (%)	Negative (%)	
Female	16 (47.1%)	18 (52.9%)	34 (42.5%)
Male	24 (52.2%)	22 (47.8%)	46 (57.5%)
Total	40	40	80

Male to female ratio was 1.35:1 with a slight male preponderance. However, there was no statistically significant difference between both sexes in HIV positive and negative patients. (P=0.821)

Table 3: Clinical presentation:

	HIV status		Total	P value	Significance
	Positive	Negative			
Scaly lesions	40	40	80	No stats	No stats as scale is constant
Greasy lesions	40	10	50	0.000	Significant
Itchy lesions	40	24	64	0.000	Significant
Hypopigmented patch	40	27	67	0.000	Significant
Hyperpigmented patch	0	5	5	0.055	Not Significant
Erythematous rash	40	8	48	0.000	Significant
Dandruff	0	8	8	0.005	Significant

All HIV positive patients with SD had scaly, greasy, itchy, hypo-pigmented and erythematous lesions. Dandruff was not seen in HIV positive patients. All HIV positive patients had multiple type of lesions.

Majority of the HIV negative patients with SD had scaly (40), itchy (24) and hypo-pigmented lesions (27) & dandruff in 8 patients.

The difference between the proportion of itchy, greasy, hypo-pigmented, erythematous lesions, and dandruff between HIV Positive and Negative groups, was statistically significant. (P< 0.05)

The most common sites affected in HIV positive SD patients were neck (23) and groin (20), followed by back (16), face (14) and chest (14) and in HIV negative SD patients was scalp (24), followed by neck (18), back (13), forehead (11) and nose (10).

The difference between the proportion of Scalp, axilla, and groin

involvement between HIV positive and negative patients, was statistically significant. (P<0.05)

Table 4: Sites involved:

Site	HIV positive	HIV negative	P value	Significance
Scalp	14	24	0.043	Significant
Nose	4	10	0.139	Not Significant
Face	14	3	0.005	Not Significant
Forehead	4	11	0.083	Not Significant
Neck	23	18	0.371	Not Significant
Chest	14	6	0.069	Not Significant
Back	16	13	0.642	Not Significant
Extremeties	15	4	0.154	Not Significant
Axilla	7	0	0.012	Significant
Groin	20	0	0.000	Significant

Table 5: Number of sites involved:

Number of sites	HIV status		Total
	Positive	Negative	
1	0	3	3
2	1	15	16
> 2	39	22	61
Total	40	40	80

In 39 HIV positive and in 22 HIV negative patients, >2 sites were involved and the difference was statistically significant. (P=0.000)

CD4 count in HIV positive SD patients:

In HIV positive patients, 20 patients had CD4 count ranging from 200-350 cells/mm^3 & mean CD4 cell count was 348.72. (±101.944) Two patients had count less than 200 cells/mm^3, 15 patients had count ranging from 350 to 500 cells/mm^3 & 3 patients had count more than 500 cells/mm^3. However, no significant association was found between clinical presentation & sites involved and CD4 count.

Laboratory Diagnosis:

Using scraping/cellotape with KOH mount/Chicago Sky Blue 6B stain, *Malassezia* was detected in 38 and 34 HIV positive & negative patients respectively by any of the collection/staining methods used and the difference was not statistically significant.

DISCUSSION

Seborrhoeic dermatitis (SD) is a common, relapsing dermatitis that is characterized by erythematous patches and superficial scaling affecting sebaceous-rich areas – namely the scalp, face, central chest and anogenital areas.[9]

Seborrheic dermatitis has two peak prevalence, an infantile self-limited form during the first 3 months of life and an adult form that is chronic.[10] The course of adult SD in affected individuals is variable throughout adulthood, some noting only occasional periods of exacerbation and others experiencing greater chronicity with more frequent recurrences.[9]

SD has been reported to be more common in the age group of 30 to 60 years as reported by majority of authors.[11-12] Manapajon Araya et al has reported 41.1 years as a mean age of SD in a study conducted at Bangkok, Thailand. [11] Similarly, in the study conducted by J.Peyri et al, at Barcelona, Spain 43.6 years was the mean age of diagnosis of SD.[12]

In present study, only adults were included. The age of the patients ranged from 18 years to 70 years. Majority of the patients i.e. 46 out of 80 were less than 30 years and the mean age was 31.5 years.(Table 1) There was no significant difference in age between HIV positive and HIV negative patients of SD. Similar findings have been reported by Yulien Amado et al.[13]

Age-related changes in the extent of colonization and the composition of the *Malassezia* microbiota are closely associated with age-related changes in sebaceous gland activity and the fatty acid composition of sebum. [14]

SD is a multifactorial skin disease that needs endogenous and exogenous predisposing factors for its development. Various authors have reported a male preponderance.[15-17] R.U Peter et al in Munich, Germany, reported that out of 575 patients, 404 were males and 171 were females. [17] Similarly, Schaub NA et al and Valentina et al have also reported a male preponderance.[15-16] This suggests a significant hormonal influence, mainly of androgens. [9, 18] However, few authors have

also reported a female preponderance. [11, 19-20]

In present study, out of 80 patients, 46 patients were males and 34 were females with a Male to female ratio of 1.35:1. (Table 2) There was no significant difference in both the sexes (P= 0.411). Our observation is consistent with studies of Yulien et al and Valentina et al. [13, 16]

Gender might not be the significant factor in the development of SD. But this can be more associated with the possible opportunistic activity of *Malassezia* spp., which can use their lipid machinery from human sebum, for growth, releasing fatty acids, and thus generating epidermis damage, which may be related to the development of SD.[13]

Adult SD is reported to involve one or more sites which includes scalp, eyebrows, forehead, nasal alar creases, chest and genital region.[12] SD tends to occur early in the course of HIV positive cases, and is usually more severe and difficult to diagnose and treat than in the general population. [21-22] Most of the authors like, Manapajon Araya et al, J Peyri et al, Barbara M Mathews et al have reported face and scalp as the most common sites of SD followed by thorax, nasolabial folds and eyebrows. [11-12, 13]

Manapajon Araya et al reported pruritus as the most common complaint while study J. Peyri et al, reported scales and erythema as the most common complaint. [11-12] Seborrheic dermatitis in HIV positive patients is explosive, inflammatory, and severe and is recalcitrant to treatment.

Clinically, as showed in Table 3 and Table 4, there was extensive involvement of sites in HIV positive SD patients beyond typical seborrheic areas, including the extremities, groin, axillae and scalp as compared to SD in HIV negative patients. It was also noted that there was multiple sites involvement in HIV positive SD patients as shown in Table 5 compared to HIV negative SD patients. In HIV negative SD patients, scalp was the most common site with dandruff as the most common complaint. (Figure 3) HIV positive patients with SD had scaly, greasy, itchy, hypo-pigmented and erythematous lesions. (Table 3 & 4) This is consistent with the findings of other reports. [21-23]

Several studies revealed a significant association between disease progression, immunologic status and manifestation and severity of SD.[24-26] A defect in immune response (specific or not) may facilitate fungal survival in the skin. Bergbrant et al. has reported that in AIDS patients who often show abnormal T-cell function, the function of a different *Malassezia*-compliant method that does not require T-cell function is suppressed and may be the explanation for the inflammatory response.[22] Parry and Sharpe suggested that SD is not caused by altered immune responses to *Malassezia* yeast and proposed toxic production or lipase activity as possible mechanisms..[27] Other researchers have shown that the lipophilic yeast is able to activate the alternative pathway of complement. All of the above processes can cause indirect skin inflammation. In short, impairment in the immune response (direct or not) may facilitate the survival of the fungus on the skin.[2,22,28] HIV/AIDS affects the response of keratinocytes to stress signals. It also has detrimental effects on Langerhans cells and their cross-talk with CD-4 memory lymphocytes resulting in virus replication and destruction of both subsets of immune cells, thus destabilizing the skin immune system.[29] The increase in prevalence of SD in people with AIDS likely correlates with T cell lymphopenia, affecting counts of CD4+ cells involved in immune surveillance.[9]

In the study conducted by Yitayih et al in Gondar, out of 292 HIV positive SD cases, 245 patients had CD4 count above 200 cells/mm3 and 47 patients had less than 200cells/mm3. [30] Nnoruka et al, in a study from Nigeria in the year 2007 found that SD was the second most common mucocutaneous disorder with a mean CD4 cell count of 453.8 ± 153 cells/mm3. [31] CD4 count was between 200 to 500

cells/mm3 in 69 patients out of 99. The high mean CD4+ cell count and high mean total lymphocyte count observed in these disorders can be explained by the fact that both mucocutaneous disorders occur early in HIV infection, as similarly reported elsewhere.[31] In the studies carried out by, Manapajon Araya et al,(11) Yitayih et al, Nnoruka EN et al, R Lifson et al, Amy K Forrestel et al, mean CD4 count ranged from 200 to 400 cells/mm3. [11, 30-33] Goh BK et al found that CD4 less than 200 cells/mm3 are prone to skin diseases. [34] In the study done at Basel, Switzerland in 1998, by Schaub N et al found that neither the initial CD4 T cell count nor antiretroviral treatment is of any significance in the development of SD. [9]Similar findings were seen in the current study which could be due to the higher values of CD4 count and history of ART in almost all patients. (38 patients) However, no significant association was found between clinical presentation & sites involved and CD4 count.

The diagnosis of SD is based on typical clinical picture, positive direct microscopy and culture of the lesion.[9] The disease can be easily diagnosed by an experienced dermatologist, but this should always be confirmed with direct microscopy or culture to demonstrate pseudohyphae and blastoconidia in typical "spaghetti and meatballs" pattern. In this study, samples were collected using scalpel for scraping as well as cellotape from the affected site and were subjected to KOH mount and Chicago Sky Blue 6B stain. *Malassezia* was detected in 38 and 34 HIV positive & negative patients respectively by any of the collection/staining methods used and the difference was not statistically significant.

CONCLUSION

Seborrheic Dermatitis has an unusual and severe presentation at multiple sites in HIV positive patients as compared to HIV negative Seborrheic Dermatitis.

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Ethical Approval: Approved

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