ORIGINAL ARTICLE

Psoriasis Patients with Human Immunodeficiency Virus Infection: Data from the Malaysian Psoriasis Registry

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Abstract

Background

Psoriasis can be a presenting feature of human immunodeficiency virus (HIV) infection. Our objective was to determine the frequency of HIV infection among patients with psoriasis and to describe the clinical features, treatment and quality of life in this population.

Methods

This is a multi-centre retrospective cross-sectional study of psoriasis patients who were registered to the Malaysian Psoriasis Registry (MPR) from January 2007 to December 2018.

Results

Of a total of 21,735 patients registered, 105 (0.5%) had HIV infection. Among these patients, 90 (85.0%) were male, mean age was 40.90 ± 10.85 years, and plaque psoriasis was the most frequently encountered presentation (85.7%). Significantly more patients with HIV had severe psoriasis (61.3% vs 49.9%, p=0.043), face and neck (62.7% vs 51.4%, p=0.022) involvement, and nail disease (69.9% vs 56.2%, p=0.005) compared to those without HIV. Only n patients (8.7%) had psoriatic arthropathy, and only 9 (8.8%) received systemic therapy, namely acitretin and methotrexate. None received a biologic, and only one patient was treated with narrowband ultraviolet-B therapy. The mean Dermatology Life Quality Index (DLQI) score at enrolment was 10.98 ± 7.07 for the HIV cohort compared to 8.68 ± 6.60 for the non-HIV cohort (t=2.190, p=0.029). More patients with HIV reported a DLQI score >10 compared to those without HIV (51.5% vs 40.2%, p=0.021).

Conclusion

The frequency of HIV infection among patients with psoriasis in the MPR was 0.5%. Patients with HIV had more severe disease, more nail, face and neck involvement, and greater impairment of quality of life. Treatment of HIV patients with psoriasis remains conservative in Malaysia.

Key words: *Psoriasis, Human immunodeficiency virus, Acquired immunodeficiency syndrome*

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Introduction

Psoriasis is a chronic, immune-mediated, inflammatory skin disease, in which the aetiopathogenesis is unclear.[1,2] It can be a presenting feature of human immunodeficiency virus (HIV) infection and disease severity may reflect the state of immune dysfunction.[3] Psoriasis in HIV patients is often more severe and refractory to treatment.[4]

Prevalence of psoriasis among HIV patients ranges from 2-3% in the western cohort,[2,5] similar to the prevalence in the general population.[4,6] Although there are many epidemiological studies on psoriasis, data on HIV-infected psoriasis patients are lacking. The aim of this study was to determine the prevalence of HIV infection among patients with psoriasis in Malaysia, and to describe the clinical features, treatment and quality of life in this population.

Materials and Methods

This was a multicentre retrospective cross-sectional study of psoriasis patients who were entered into to the Malaysian Psoriasis Registry (MPR) from January 2007 to December 2018. Psoriasis patients had to have two positive HIV antibody tests to be diagnosed as having HIV infection. The MPR is a prospective systematic collection of data of psoriasis patients in Malaysia from 32 public hospitals and 2 private hospitals. All psoriasis patients were enrolled at their first visit and those on systemic treatment or phototherapy were seen every 6 months thereafter f. This study was approved by the Malaysian Medical Research and Ethics Committee (MREC) number NMRR-20-802-53706.

Body surface area (BSA) involvement was used to assess disease severity, whereas the Dermatology Life Quality Index (DLQI) was used to assess the impact of psoriasis on quality of life.[7] Severe impact of psoriasis on health-related quality of life (HRQoL) is defined as a DLQI score of more than 10.[8] Body surface area (BSA) involvement of more than 10% with or without a DLQI score of more than 10 is also considered severe psoriasis.[8]

Descriptive analyses were performed for characterizing sociodemographic characteristics, clinical pattern, and treatment modalities of the patients. Data were tabulated and analysed using IBM® Statistical Package for the Social Sciences (SPSS) Statistics for Windows, Version 24.0. Armonk, NY: IBM Corp. Descriptive data were presented as numbers and percentages for categorical variables. Mean with standard deviation (SD) was used for continuous data. Pearson Chi-square test and Fisher's exact test were used to analyse categorical data where applicable. Comparison of means was performed using independent sample T-test. A p value of <0.05 was considered as statistically significant.

Results

There were 105 patients (0.5%) reported to be infected with HIV among a total 21,735 psoriasis patients registered in the MPR between January 2007 and December 2018. Sociodemographic characteristics are presented in Table 1. There was a significantly higher proportion of males in the HIV group compared to those not infected with HIV (86.7% vs 55.3%, $p<0.001$). The mean age for HIV-infected patients was 40.95 ± 10.93 years. Overall, 11.4% of HIV-infected patients had a positive family history of psoriasis, compared to 22.4% in the non-HIV infected group ($p=0.004$). About twenty percent of the HIV-infected psoriasis patients were obese, with a body mass index (BMI) ≥ 25mg/kg^2 (Table 2). Other comorbidities associated with the HIV-infected cohort were dyslipidaemia (13.1%), hypertension (7.8%), diabetes mellitus (4.9%) and ischemic heart disease (2.0%). Six patients had co-infection with either hepatitis B or hepatitis C.

Table 1. Demographic characteristics of study population

Demographic Characteristic		HIV n=105 (%)	Non-HIV n=21630	*p*-value
Age (years)	Mean (SD)	40.95 (10.93)	41.80 (17.48)	*p*=0.621
	Min, Max	19, 82	1, 92	
Gender	Male	91 (86.7)	11967 (55.3)	***p*<0.001**
	Female	14 (13.3)	9663 (44.7)	
*Ethnicity	Malay	39 (37.5)	11762 (54.4)	***p*<0.001**
	Chinese	42 (40.4)	4112 (19.0)	***p*<0.001**
	Indian	13 (12.5)	3537 (16.4)	*p*=0.288
	Others	10 (9.6)	2210 (10.2)	*p*=0.840
Family history of psoriasis		12 (11.4)	4841 (22.4)	***p*=0.004**
Comorbidities	Dyslipidaemia	13 (13.1)	3401 (15.7)	*p*=0.584
	Hypertension	8 (7.8)	4979 (23.0)	***p*<0.05**
	Diabetes mellitus	5 (4.9)	3323 (16.4)	***p*<0.05**
	Ischemic heart disease	2 (2.0)	1015 (4.7)	*p*=0.345
	Cerebrovascular disease	0 (0)	311(1.4)	*p*=0.401

*HIV, human immunodeficiency virus; SD, standard deviation; *HIV n=104, Non-HIV n=21621*

Table 2. Clinical characteristics of study population

Clinical Characteristic		HIV n=98 (%)	Non-HIV n=20355 (%)	*p*-value
Type of psoriasis	Plaque	84 (85.7)	18905 (92.9)	***p*=0.002**
	Guttate	9 (9.2)	729 (3.5)	***p*=0.003**
	Erythrodermic	4 (4.1)	384 (1.9)	*p*=0.116
	Palmoplantar non-pustular	1 (1.0)	85 (0.4)	*p*=0.339
	Inverse	0 (0)	104 (0.5)	*p*=1.000
	Generalised pustular	0 (0)	92 (0.5)	*p*=1.000
	Localised pustular	0 (0)	56 (0.3)	*p*=1.000
[#]Body mass index (BMI) (kg/m2)	<18.5	21 (22.1)	1358 (7.2)	***p*<0.001**
	18.5-22.9	38 (40.0)	4129 (21.9)	***p*<0.001**
	23-24.9	17 (17.9)	2730 (14.5)	*p*=0.348
	>25	19 (20.0)	10616 (56.4)	***p*<0.001**
[b]Body surface area (BSA) (%)	<5	30 (36.6)	7392 (44.8)	*p*=0.134
	5-10	19 (23.2)	5173 (31.4)	*p*=0.110
	11-90	28 (34.1)	3498 (21.2)	***p*=0.004**
	>90	5 (6.1)	422 (2.6)	***p*=0.044**
[c]Face and neck involvement		64 (62.7)	10439 (51.4)	***p*=0.022**
[d]Nail disease		72 (69.9)	11797 (56.2)	***p*=0.005**
[e]Scalp		87 (85.3)	16674 (81.2)	*p*=0.293
[f]Psoriatic arthropathy		9 (8.7)	2747 (13.1)	*p*=0.180
[g]DLQI >10		53 (51.5)	7909 (40.2)	***p*=0.021**
[h]Severe psoriasis (BSA >10 and/ or DLQI >10)		49 (61.3)	7553 (49.9)	***p*=0.043**

HIV, human immunodeficiency virus; BMI, body mass index; BSA, body surface area; DLQI, dermatology life quality index; [#]HIV n=95, non-HIV n=18833; [b]HIV n=82, non-HIV n=16485; [c]HIV n=102, non-HIV n=20301; [d]HIV n=103, non-HIV n=20988; [e]HIV n=102, non-HIV n=20529; [f]HIV n=104, non-HIV n=20973; [g]HIV n=103, non-HIV n=19651; [h]HIV n=80, non-HIV n=15128

The most common type of psoriasis in our HIV cohort was plaque psoriasis (85.7%), followed by guttate psoriasis (9.2%), erythrodermic psoriasis (4.1%) and non-pustular palmoplantar psoriasis (1.0%), as shown in Table 2. Non-plaque forms of psoriasis were significantly more frequently encountered in HIV-infected psoriasis patients (p=0.002), with guttate and erythrodermic psoriasis reported at a higher rate among those with HIV infection. Scalp involvement in the HIV cohort (85.3%) was slightly higher compared to the non-HIV group (81.2%). HIV-infected psoriasis patients reported a higher rate of face and neck involvement (62.7% vs 51.4%) (*p*=0.022) and nail disease (69.9% vs 56.2%) (*p*=0.005) compared to non-HIV psoriasis patients. Psoriatic arthropathy was observed in 8.7% of the HIV cohort, less frequently than in the non-HIV-infected group (13.1%). Oligo-mono-arthropathy was the most common type of psoriatic arthropathy in both groups, followed by distal hand joint involvement and symmetrical rheumatoid-like polyarthropathy.

In terms of disease severity, 40.2% of HIV-infected psoriasis patients had severe disease with a BSA of >10%, compared to 23.8% in the non-HIV infected group (*p*=0.004). The rate of erythroderma was observed to be significantly higher in the HIV cohort (6.1% vs 2.6%, *p*=0.044).

Fifty-three (51.5%) of the HIV-infected psoriasis patients had a DLQI score of more than 10 at enrolment versus 40.2% in the non-HIV group. The mean DLQI score for the HIV cohort was 11.58 ± 7.02, which was significantly higher compared to the non-HIV psoriasis group (9.61 ± 6.75 *p*=0.003). The domains that displayed a significant difference between the two groups were personal relationship (70.0% vs 56.4%, *p*=0.022) and work and school (65.8% vs 46.1%, *p*<0.001) (Table 3). In aggregate, 61.3% of HIV-infected psoriasis patients had severe psoriasis defined by a BSA of more than 10% and/ or a DLQI of more than 10, compared to 49.9% in the non-HIV infected cohort (*p*=0.043).

Similar to the non-HIV cohort, the majority of the HIV patients were prescribed topical treatment with topical corticosteroids and emollients as the mainstays of treatment (Table 4).

Only 9 patients (8.8%) in the HIV-infected category received systemic therapy (7 (6.9%) were prescribed acitretin and 2 (2%) were prescribed methotrexate), compared to 14.3% of the non-HIV-infected psoriasis patients. Only one HIV-infected psoriasis patient underwent narrow band ultra-violet B (NBUVB) phototherapy treatment. None of the HIV-infected patients received a biologic treatment, whereas 0.4% of non-HIV patients were

treated with a biologic agent.

Table 3. Comparison of Dermatology Life Quality Index (DLQI) scores between patients with human immunodeficiency virus infection and those without

Domain	HIV (%)	Non-HIV (%)	*p*-value
[t]Physical symptoms and feeling	96.1	94.8	*p*=0.324
[v]Daily activities	83.3	76.0	*p*=0.402
[w]Leisure	83.3	75.2	*p*=0.318
[x]Work and school	65.8	46.1	***p*<0.001**
[y]Personal relationship	70.0	56.4	***p*=0.022**
[z]Treatment	70.7	60.8	*p*=0.149

HIV, human immunodeficiency virus; DLQI, dermatology life quality index; [t]HIV n=102, non-HIV n=19583; [v]HIV n=96, non-HIV n=18725; [w]HIV n=90, non-HIV n=17122; [x]HIV n=73, non-HIV n=12879; [y]HIV n=70, non-HIV n=14580, [z]HIV n=92, non-HIV n=18590

Table 4. Types of treatment for psoriasis in study population

Treatment Option		HIV n=104 (%)	Non-HIV n=20792 (%)	*p*-value
Treatment	Topical	96 (92.3)	19733 (94.9)	*p*=0.23
	[α]Phototherapy	1 (1.0)	550 (2.7)	*p*=0.282
	[β]Systemic therapy	9 (8.8)	2949 (14.3)	*p*=0.116
	Acitretin	7 (6.9)	574 (2.8)	***p*=0.013**
	Methotrexate	2 (2.0)	2289 (11.1)	***p*=0.003**
	Systemic corticosteroids	0	179 (0.9)	*p*=0.345
	Cyclosporin	0	144 (0.7)	*p*=0.398
	Biologics	0	78 (0.4)	*p*=0.534
	Hydroxyurea	0	23(0.1)	*p*=0.736

HIV, human immunodeficiency virus; [α]HIV n=103, non-HIV n=20461; [β]HIV n=102, non-HIV n=20461

Discussion

The reported prevalence of psoriasis varies in different regions of the world based on different epidemiological studies.[4,6,9,10] In Western countries, it is estimated to affect 2-4% of the general population[1,11] and the prevalence of psoriasis among HIV patients ranges from 2-3%.[2,5,12] A literature search revealed limited studies on the prevalence of HIV in psoriasis patients. It had reported that 1.8% of psoriasis patients in Thailand were infected with HIV,[13] which is slightly higher than the 1.1% prevalence of HIV in the general population there.[14] It was estimated that there were 87,041 people living with HIV in Malaysia with a prevalence of 0.4%.[14] The frequency of HIV infection among individuals with psoriasis in our cohort (0.5%) was comparable to the prevalence of HIV in the general population of Malaysia.[14] The lower rate of HIV infection among psoriasis patients in the MPR (0.5%) compared to Thailand was possibly be due to voluntary notification, non-mandatory HIV testing among psoriasis patients or management of HIV patients with mild psoriasis by primary care doctors who are not registered with the registry. A French survey study revealed that currently health practitioners only screen psoriasis patients for HIV based on risk factors such as high-risk behaviour, clinical severity and assessment prior to systemic and biologic therapy.[15] An updated guideline on the timing to screen for HIV in psoriasis patients is needed to ensure HIV infection is detected early and managed accordingly.

The clinical manifestations of psoriasis observed in HIV-infected patients appear to be similar to those in non-HIV-infected persons in our cohort. Plaque psoriasis is the most common presentation in the HIV psoriasis population in Western countries.[16] However, psoriasis patients with HIV can present with multiple subtypes concurrently.[3,4,17] Several studies from the United States of America, the United Kingdom and South Africa reported that guttate, inverse and erythrodermic psoriasis occur with higher frequency in HIV-infected psoriasis patients.[3,4,16] Similar findings were also evident in our cohort, with the exception of inverse psoriasis as none was reported.

Interestingly, we observed a greater number in the HIV cohort having face and neck involvement. The literature revealed that sebo-psoriasis is a psoriasis variant associated with increased incidence in HIV infection.[17,18] Sebo-psoriasis represents a clinical overlap between seborrheic dermatitis and plaque psoriasis, affecting the flexural areas, scalp and retro- auricular region.[17,18] Lesions tend to be more greasy and less scaly compared to scalp psoriasis.[17] The MPR, however, does not capture this phenotypic variant of psoriasis. Hence, sebo-psoriasis may be labelled as flexural psoriasis or as psoriasis involving the face and neck. It is important to recognise sebo-psoriasis as a separate entity as it can occur in HIV patients with a normal CD4 count and/or those with a suppressed viral load. A high index of suspicion among patients presenting with prominent sebo-psoriasis is therefore paramount as it may act as an indicator of early HIV infection.[18]

As sebo-psoriasis is reported to respond well to topical or systemic antifungal treatment[18], could be overlooked easily.

Up to 50% of psoriasis patient have nail involvement[13,19] and lifetime incidence is up to 90%.[20] Common manifestations include onycholysis (69.6%) and pitting of the nails (62.6%).[13] It has been reported that 32% of HIV-infected psoriasis patients, have nail involvement[21], which may present in a similar manner to nail psoriasis in non-HIV infected patients (i.e. superficial pitting, distal and lateral onycholysis, subungual hyperkeratosis and onychodystrophy).[3,22] Interestingly, in our study, nail disease in the HIV cohort was found to be significantly more prevalent than in the non-HIV infected cohort. Nail changes in psoriasis are strongly associated with arthropathy, which affects 23-50% of HIV-positive psoriasis patients worldwide.[22,23] The prevalence of arthropathy is higher among HIV-infected patients, compared to the general psoriasis population(5.8% to 19%),[2,24,25] and it is associated with more severe types of joint disease.[2,23,25,26] Symmetrical polyarthropathy, enthesopathy and dactylitis represent the most frequently observed types of psoriatic arthropathy in HIV patients.[2,27,28] In contrast, our study revealed a significantly lower rate of arthropathy among patients in the HIV-infected group. The most common type of arthropathy described in our HIV cohort was oligo-arthritis.

Interestingly we did not observe any cases of either localized or generalized pustular psoriasis in our cohort. Pustular psoriasis may be associated with specific variants of the *IL36RN*, *AP1S3* and *CARD14* genes, resulting in different clinical manifestations and treatment response.[29] IL36, which is implicated in the pathogenesis of pustular psoriasis, has not been described in HIV infection.[30,33] Morar *et al.* observed a reactive arthritis-like syndrome in HIV-infected patients; these patients presented with keratoderma blenorrhagica with palmoplantar psoriasiform plaque.[3] Histology of skin lesions from these patients is identical to that seen in pustular psoriasis.[3] Mikhail et al reported successful use of etanercept to treat a case of von Zumbusch pustular psoriasis in a HIV patient on antiretroviral therapy (ART) with a CD4 count of 435/μL.[31]

Psoriasis has a detrimental effect on a person's quality of life comparable to other chronic illnesses.[32] To our knowledge, there are no other published data assessing DLQI among psoriasis patients with HIV. In our study, more than one third of patients at enrolment had a DLQI score of more than 10, which indicates severe impairment in quality of life. The higher mean DLQI among HIV-infected patients could be attributed to their manifesting more severe disease compared to non-HIV infected patients.[4] The DLQI domain most affected was physical symptoms and feelings among both HIV- and non-HIV-infected psoriasis patients. In our study, work and school, and personal relationship were significantly more impaired in the HIV group compared to the non-HIV group. These items could be influenced by other co-morbidities related to HIV infection that could cause disturbances in daily activities as well as social interaction with others.

Management of psoriasis in HIV patients is challenging due to the abnormal immune status of patients at various phases of their disease course.[30,33] Imbalance in the $CD8^+$ to $CD4^+$ ratio in HIV has been associated with the pathogenesis of psoriasis, which is $CD8^+$ T-cell mediated. A low $CD4^+$ T-cell count relative to CD8+ T-cells in the setting of HIV, may lead to a propensity to develop more severe psoriasis.[34,35]

Some reports showed that psoriasis may improve after treatment with ART.[36-39] Case reports from Italy and Chile have reported improvement of erythrodermic psoriasis in HIV patients after ART was initiated.[36,37] ART as a first line therapy has been shown to control the progression of HIV and also effectively treat HIV-associated psoriasis, which commonly occurs when $CD4^+$ T-cell counts fall below 350cells/mm^3.[4,40] Zidovudine, a thymidine analogue and a nucleoside reverse transcriptase inhibitor (NRTI) has also been reported to be beneficial in improving psoriasis in HIV patients.[16,41] Likewise, antiretroviral combinations of protease inhibitors and a non-nucleoside reverse transcriptase inhibitor (NRTI), or two NRTIs plus an entry inhibitor, have demonstrated efficacy in treating HIV-associated psoriasis.[39] However, data on neither $CD4^+$ T-cell counts nor the use of ART was available from our registry.

On the contrary, there have been reports of exacerbation of psoriasis as a manifestation of

immune reconstitution inflammatory syndrome (IRIS) upon commencement of antiretroviral therapy in HIV-infected psoriasis patients.[35] Flare of plaque psoriasis has been reported after commencement of ART.[35,42] Tripathi *et al.* reported a HIV-infected patient who had IRIS with worsening of the pre-existing psoriasis plaques and acral involvement after switching to new ART (elvitegravir, tenofovir, cobicistat and emtricitabine). The patient's condition improved after ART was reintroduced with concurrent phototherapy using psoralen and ultraviolet A therapy together with topical clobetasol.[42] Another case report from Lebanon described the occurrence of psoriasis for the first time in 2 patients after ART was introduced, manifesting as IRIS.[35] However, both patients were not compliant with their ART leading to altered $CD4^+/CD8^+$ T-cell ratios, which further contributed to their psoriasis flare.[35] These two scenarios reveal that worsening of psoriasis in the context of IRIS may occur at commencement or interruption of ART.[35]

HIV-infected patients who have mild psoriasis were generally prescribed topical medications, such as topical corticosteroids, calcipotriene, tar preparations and emollients.[33] Phototherapy with ultraviolet (UV) radiation in combination with antiretroviral therapy is recommended as a first-line treatment for moderate-to-severe psoriasis in patients with HIV.[4] Phototherapy does not induce immunosuppression and low dose ultraviolet light has been shown to be beneficial in HIV-infected patients with skin disease.[43,44] However, it was not prescribed as a preferred modality in our cohort. Reasons for low usage of phototherapy in the HIV-infected cohort could be adherence issues for these patients and the lack of access to phototherapy. Of note, the use of phototherapy in HIV patients with Kaposi's sarcoma is prohibited as it may lead to worsening of Kaposi's sarcoma.[41,45,46]

A majority of dermatologists were most comfortable with prescribing acitretin, followed by phototherapy, for the treatment of psoriasis in HIV-infected patients based on a survey in France.[15] Our data showed that only 10% of HIV-infected patients received systemic therapy despite having more severe disease than patients in the non-HIV cohort. This showed that our dermatologist was cautious in prescribing these agents for fear of the compounded immunosuppressive effect in the HIV cohort or hesitancy of HIV patients on ART to be on numerous medications. Further studies to assess factors contributing to the lack of use of systemic therapy or phototherapy for treating HIV-infected psoriasis patients are needed. Acitretin, an oral retinoid, can improve psoriasis signs and symptoms, and is a preferred systemic treatment option over methotrexate and cyclosporin by virtue of its non-immunosuppressive effect.[4,21] Monitoring of fasting serum lipid levels is important as risk of hypertriglyceridemia and pancreatitis may be increased when oral retinoids are used together with antiretroviral medications.[4]

Treatment of HIV-infected patients with recalcitrant psoriasis who have failed or were unable to tolerate first line therapy is challenging. Use of methotrexate should be avoided in HIV-infected psoriasis patients.[51] A report from Duvic *et al* discouraged the use of methotrexate due to the risk of opportunistic infections.[41] Another case series reported *Pneumocystis carinii* infection in 2 of 4 patients treated with methotrexate.[47] On the other hand, treatment with cyclosporin, a calcineurin inhibitor, had limited reports, though it has been used safely and with good responses in HIV-infected psoriasis patients.[48,49] Of note, monitoring cyclosporin levels may be required as the bioavailability may be increased by the concomitant use of protease inhibitors.[50] Due to the risk of nephrotoxicity and hypertension, short-term use is recommended for up to 12 weeks.[3] Systemic treatment for HIV-infected psoriasis patients should be individualised with close monitoring of the CD4 count and viral load.[51] Although unavailable in Malaysia, apremilast, a phosphodiesterase-4 inhibitor is approved for the treatment of moderate to severe psoriasis.[49] Data on the usage of apremilast in HIV-infected psoriasis patients is scarce. There were a few reports of successful treatment of plaque and palmoplantar psoriasis in HIV-infected psoriasis patients.[53-55] Sacchelli *et al.* had also noted improvement in psoriatic nail disease in their cohort of patients.[56]

There are no guidelines on the usage of biologics in psoriasis patients with HIV. Not with standing, biologic agents are predicted to be safe in HIV-infected psoriasis patients especially for those on ART.[57] Biologic agents such as etanercept, adalimumab, infliximab and ustekinumab have

been used to treat HIV-infected psoriasis patients with good clinical response, safety outcomes and tolerability.[40,57,58] Clinical responses among HIV-infected psoriasis patients treated with a biologic are reported to be comparable to those with non-HIV-infected psoriasis.[31,59,60] Bardazzi *et al.* suggested that biologic agents may be considered in psoriasis patients with stable HIV infection, and with close monitoring of CD4 counts and HIV viral load levels.[57] The use of biologic agents for the treatment of psoriasis in our cohort study was low among non-HIV-infected patients, and no patients in the HIV-infected group were treated with a biologic. Many factors such as budget constraints and policies may have contributed to this.

Limitations

The registry database does not discern whether more than one type of psoriasis occurred in the same patient, as only the predominant type is documented. Data on antiretroviral therapy, CD4 counts and whether a diagnosis of psoriasis preceded HIV infection, or vice versa, are also not available. As with all retrospective studies, missing data may not be accounted for and causal relationships could not be determined.

Recommendations

A prospective study focusing on the evolution of psoriasis throughout the different stages of HIV/AIDS would be valuable. Research assessing systemic and biological treatments for psoriasis patients with HIV infection is needed. Registries dedicated to studying HIV-infected psoriasis patients should be established to allow for more comprehensive data collection.

Conclusion

The prevalence of HIV infection among psoriasis patients in the MPR was 0.5%. Most of the HIV-infected psoriasis patients in this cohort had plaque psoriasis. Higher proportion of HIV-infected patients had more nail, face and neck involvement, and more severe disease, which significantly affected quality of life. Treatment of psoriasis in patients with HIV infection was comparatively less aggressive compared to treatment of non-HIV-infected psoriasis patients in the MPR cohort.

Conflict of Interest Declaration

The authors have no conflict of interest to declare.

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