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Clinical profile, treatment and quality of life of patients with psoriatic arthritis in Malaysia: A population-based cross-sectional study

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Abstract

Psoriatic arthritis (PsA) is a major comorbidity of psoriasis and may lead to irreversible joint damage and disability. This study aims to describe the clinical profile, treatment and quality of life (QoL) of patients with PsA in Malaysia. This is a multicentre retrospective cross-sectional study of psoriasis patients who were notified to the Malaysian Psoriasis Registry (MPR) from January 2007 to December 2018. Of 21 735 psoriasis patients, 2756 (12.7%) had PsA. The male to female ratio was 1:1. The mean age of psoriasis onset for PsA patients was 34.73 ± 14.44 years. They had a higher rate of family history of psoriasis (26% vs. 22.4%, $p < 0.001$), scalp (82.7% vs. 81.0%, $p = 0.04$) and nail involvement (73.3% vs. 53.3%, $p < 0.001$), obesity (62.6% vs. 54.4%, $p < 0.001$), dyslipidaemia (23.8% vs. 15.4%, $p < 0.001$), hypertension (31.1% vs. 22.7%, $p < 0.001$) and diabetes mellitus (20.9% vs. 15.2%, $p < 0.001$) compared to non-PsA patients. More than half (54.3%) had severe psoriasis [(body surface area >10% and/or Dermatology Life Quality Index (DLQI) >10)]. Most had oligo-/monoarthropathy (40.3%), followed by distal interphalangeal arthropathy (31.3%), symmetrical polyarthropathy (28.3%), spondylitis/sacroiliitis (8.2%) and arthritis mutilans (3.2%). Nearly 40% of PsA patients received systemic treatment, but only 1.6% received biologic agents. QoL was more significantly affected in PsA than in non-PsA patients (mean DLQI 10.12 ± 7.16 vs. 9.52 ± 6.67, $p < 0.001$). One in eight patients with psoriasis in Malaysia had PsA. They had a higher incidence of comorbidities, severe disease, impaired QoL and were more likely to receive systemic and biological treatment compared to non PsA patients.

KEYWORDS
inflammatory arthritis, psoriasis, psoriatic arthritis, quality of life

1 | INTRODUCTION

Psoriatic arthritis (PsA) is a chronic, immune-mediated, progressive inflammatory disease of the joints and entheses associated with psoriasis.[1,2] Although the aetiology of psoriasis is unknown, it is believed that it involves a complex interplay between genetics, environmental factors and immune-mediated inflammation.[2] PsA has heterogeneous clinical manifestations, characterised by diverse clinical phenotypes involving the skin and nails, peripheral arthritis, dactylitis, enthesitis, spondylitis and uveitis. Delay in diagnosis

and treatment may cause permanent joint deformity and disability leading to severe impairment of patients' quality of life (QoL).[3,4] In addition, PsA is associated with comorbidities such as obesity, metabolic syndrome, cardiovascular disease, cerebrovascular disease and peripheral vascular disease,[5] that can influence treatment decisions and management strategies.

The prevalence of PsA varies widely in different countries ranging from 0.05% to 0.25% in the general population around the world,[6,7] and up to 30% of psoriasis patients will develop PsA.[8–10] To date, little is known on the clinical profile of patients with PsA in Malaysia. Thus, this study aims to describe the clinical profile, treatment and QoL of patients with PsA in Malaysia.

2 | MATERIALS AND METHODS

This is a multicentre retrospective cross-sectional study of psoriasis patients who were registered to the Malaysian Psoriasis Registry (MPR) from January 2007 to December 2018. The MPR is a prospective, collection of data on patients with psoriasis in Malaysia. Psoriasis patients were confirmed to have PsA once diagnosed by a rheumatologist. All patients diagnosed with PsA were included for analysis. Data on sociodemographic characteristics, symptoms, clinical patterns, treatment modalities and QoL of psoriasis and psoriatic arthropathy patients were collected and analysed. Concurrent comorbidities were also obtained. Incomplete or missing data on joint disease in the MPR were excluded for analysis.

Patients were categorised according to the Asia-Pacific body mass index (BMI) classification as underweight (BMI $<18.5\,kg/m^2$), normal weight (BMI18.5–22.9 kg/m^2), overweight (BMI 23.0–24.9 kg/m^2) and obese (BMI $\geq 25\,kg/m^2$).[11]

Body surface area (BSA) involvement was used to assess the cutaneous disease severity, in which it was divided into four scales which were <5%, 5%–10%, 11%–90% and >90%. The Dermatology Life Quality Index (DLQI) was used to assess patients' QoL in our cohort. Our data on DLQI was recorded at the first point of contact with the physician at the notifying centre, either at diagnosis, before or after treatment if the patient had been seen by other physicians from referring centres. The DLQI is a validated QoL assessment tool, specifically designed to measure the impact of a dermatological disease on the QoL of an affected individual,[12] which comprises of 10 multiple choice questions assessing patients' symptoms and feelings, daily activities, leisure, work and school, personal relationships and treatment in the past 1 week. The higher the score, the more the QoL is impaired. A BSA involvement of more than 10% with or without a DLQI score of more than 10 indicated severe psoriasis.

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

Ethical approval for this study was obtained from the Medical Research and Ethics Committee (MREC), Ministry of Health Malaysia (NMRR-21-89-58092).

3 | RESULTS

3.1 | Demographics characteristics

A total of 21735 psoriasis patients were registered at 34 centres contributing to the MPR from January 2007 to December 2018. Of these, 2756 (12.7%) had PsA. A total of 647 subjects were excluded due to missing data on joint disease. The demographics and clinical characteristics of the study population are summarised in Table 1. In the PsA cohort, the male to female ratio was 1:1. The ethnic distribution of PsA patients were Malay (48.6%), Chinese (18.7%), Indian (22.1%) and others (10.7%). PsA was significantly more common among Indians in our cohort (17.5% vs. 10.9%, $p<0.001$). A significantly higher family history of psoriasis was reported in the PsA cohort compared to the non-PsA cohort (26% vs. 22.4%, $p<0.001$).

Patients with PsA presented with psoriasis at an older age (34.73 ± 14.44 years) compared to the non-PsA cohort (33.11 ± 17.29 years) ($p<0.001$). Furthermore, the PsA cohort also revealed a longer disease duration (11.49 ± 10.14 years) compared to the non-PsA cohort (8.02 ± 9.40 years) ($p<0.001$).

3.2 | Clinical characteristics

The PsA cohort had a statistically significant higher rate of comorbidities in comparison to the non-PsA cohort. A higher incidence of obesity (BMI of $\geq 25\,kg/m^2$) was reported in the PsA cohort (62.6% vs. 54.4%, $p<0.001$) compared to psoriasis patients without PsA. In addition, PsA patients also had a higher frequency of dyslipidaemia (23.8% vs. 15.4%, $p<0.001$), hypertension (31.1% vs. 22.7%, $p<0.001$) and diabetes mellitus (20.9% vs. 15.2%, $p<0.001$) in comparison to the non-PsA cohort.

The most common type of PsA in our study cohort was oligo-/monoarthropathy (40.3%), followed by distal interphalangeal arthropathy (31.3%), symmetrical polyarthropathy (28.3%), spondylitis/sacroiliitis (8.2%) and the least common was arthritis mutilans (3.2%) (Table 2). The majority of PsA patients were observed within the group of 18–60 years old (Table 2). Arthritis mutilans was not seen and axial disease was less than 5% in those less than 18 years. Morning stiffness of more than 30 min was reported in 32.6% of PsA patients, whereas enthesopathy was reported in 13.9% of the cohort. Most PsA patients experienced pain (79.5%) at the time of presentation, 35.5% had periarticular swelling, while 23.2% had joint deformity.

Plaque psoriasis was the most common subtype of psoriasis in our PsA cohort, accounting for 93.3%, followed by erythrodermic psoriasis (3.7%), and guttate psoriasis (2.0%). Other less common

TABLE 1 Demographics, clinical characteristics and treatment of psoriasis patients with and without PsA.

Demographic characteristics	Psoriasis with PsA	Psoriasis without PsA	*p*-Value
Gender, *n* (%)	*n* = 2756	*n* = 18 332	<0.001
Male	1357 (49.2%)	10 335 (56.3%)	
Female	1399 (50.8%)	7997 (43.6%)	
Male: Female	1:1	1:0.7	
Ethnicity, *n* (%)	*n* = 2756	*n* = 18 324	
Malay	1339 (48.6%)	10 082 (55.0%)	
Chinese	515 (18.7%)	3504 (19.1%)	
Indian	608 (22.1%)	2857 (15.5%)	
Others	294 (10.7%)	1881 (10.3%)	
Age for onset of psoriasis (years) (mean ± SD)	34.73 ± 14.44	33.11 ± 17.29	<0.001
Mean Psoriasis disease duration (years) (mean ± SD)	11.49 ± 10.14	8.02 ± 9.40	<0.001
Family history of psoriasis, *n* (%)	*n* = 2710	*n* = 18 049	<0.001
	704 (26%)	4050 (22.4%)	
Smoking history, *n* (%)	*n* = 1490	*n* = 11 132	<0.001
	399 (26.8%)	3503 (31.5%)	
Body mass index[a] (kg/m^2), *n* (%)	*n* = 2432	*n* = 16 413	
<18.5	84 (3.5%)	1276 (7.8%)	
18.5–22.9	467 (19.2%)	3724 (22.7%)	
23–24.9	358 (14.7%)	2477 (15.1%)	
≥25	1523 (62.6%)	8936 (54.4%)	
Comorbidities, *n* (%)			
Dyslipidaemia	633 (*n* = 2660), 23.8%	2728 (*n* = 17 719), 15.4%	<0.001
Hypertension	841 (*n* = 2701), 31.1%	4069 (*n* = 17 907), 22.7%	<0.001
Diabetes mellitus	563 (*n* = 2695), 20.9%	2714 (*n* = 17 885), 15.2%	<0.001
Ischemic heart disease	136 (*n* = 2692), 5.1%	862 (*n* = 17 875), 4.8%	0.61
Stroke	38 (*n* = 2691), 1.4%	266 (*n* = 17 866), 1.5%	0.76
Face and neck involvement, *n* (%)	*n* = 2648	*n* = 17 611	0.06
	1408 (52.3%)	9014 (51.2%)	
Scalp involvement, *n* (%)	*n* = 2665	*n* = 17 820	0.04
	2204 (82.7%)	14 441 (81.0%)	
Nail disease, *n* (%)	*n* = 2756	*n* = 18 332	
	2019 (73.3%)	9763 (53.3%)	<0.001
Pitting	1412 (51.2%)	7160 (39.1%)	<0.001
Discoloration	708 (25.7%)	2800 (15.3%)	<0.001
Total nail dystrophy	117 (4.2%)	463 (2.5%)	<0.001
Onycholysis	1087 (39.4%)	4375 (23.9%)	<0.001
Subungual hyperkeratosis	351 (12.7%)	1250 (6.8%)	<0.001
Types of psoriasis	*n* = 2638	*n* = 17 589	
Plaque	2462 (93.3%)	16 310 (92.7%)	
Erythrodermic	97 (3.7%)	284 (1.6%)	
Generalised pustular	9 (0.3%)	84 (0.5%)	
Localised pustular	6 (0.2%)	49 (0.3%)	
Guttate	54 (2.0%)	674 (3.8%)	
Flexural	5 (0.2%)	99 (0.6%)	
Palmoplantar non-pustular	4 (0.2%)	82 (0.5%)	
Purely nails	1 (<0.1%)	7 (<0.1%)	

(Continues)

TABLE 1 (Continued)

Demographic characteristics	Psoriasis with PsA	Psoriasis without PsA	*p*-Value
Body surface area of psoriasis	*n* = 2245	*n* = 14 098	
<5%	910 (40.5%)	6421 (45.5%)	
5%–10%	655 (29.2%)	4466 (31.7%)	
11%–90%	567 (25.3%)	2904 (20.6%)	
BSA > 90%	113 (5.0%)	307 (2.2%)	
Severe psoriasis (BSA > 10% and/or DLQI > 10)	*n* = 2198	*n* = 12 808	<0.001
	1194 (54.3%)	6306 (49.2%)	
Treatment	*n* = 2700	*n* = 17 915	
Systemic therapy	*n* = 1063	*n* = 1868	
Acitretin, *n* (%)	120 (11.3%)	**454 (24.3%)**	<0.001
Methotrexate, *n* (%)	**933 (87.8%)**	1339 (71.6%)	<0.001
Systemic corticosteroid, *n* (%)	48 (4.5%)	**127 (6.8%)**	<0.001
Cyclosporine, *n* (%)	41 (3.9%)	**100 (5.4%)**	<0.001
Hydroxyurea, *n* (%)	7 (0.7%)	**16 (0.9%)**	0.01
Biologic therapy, *n* (%)	**43 (1.6%)**	35 (0.2%)	<0.001
Phototherapy, *n* (%)	95, 3.6% (*n* = 2657)	449, 2.5% (n = 17 765)	0.002
DLQI scores, mean ± SD	*n* = 2668	*n* = 16 486	
Symptoms and feelings	2.84 ± 1.57	2.86 ± 1.54	0.61
Daily activities	2.20 ± 1.79	2.11 ± 1.73	0.02
Leisure	2.41 ± 1.93	2.23 ± 1.85	<0.001
Work and school	0.91 ± 1.05	0.74 ± 0.96	<0.001
Personal relationship	1.41 ± 1.63	1.30 ± 1.53	0.002
Treatment	1.04 ± 0.97	0.93 ± 0.93	<0.001
Mean DLQI	**10.12 ± 7.16**	**9.52 ± 6.67**	<0.001

Note: Bold indicates the higher numbers of treatment use in particular group of patients, and the higher mean DLQI in PsA group of patients.

[a]BMI category based on Asia-Pacific Classification.

Abbreviation: BSA, body surface area, DLQI, dermatology life quality index; PsA, psoriatic arthritis; SD, standard deviation.

TABLE 2 Subanalysis of type of PsA according to age.

	Overall	<18 years old	18–60 years old	>60 years old
Age	***n* = 21 735**	***n* = 1783**	***n* = 16 422**	***n* = 3530**
Patients with PsA, *n* (%)	2756 (12.7%)	44 (2.5%)	2297 (14.0%)	415 (11.8%)
Oligo/monoarthropathy, *n* (%)	1111 (40.3%)	26 (59.1%)	946 (41.2%)	139 (33.5%)
Distal interphalangeal Predominant, *n* (%)	864 (31.3%)	10 (22.7%)	731 (31.8%)	123 (29.6%)
Symmetrical polyarthropathy (Rheumatoid like), *n* (%)	781 (28.3%)	6 (13.6%)	649 (28.3%)	126 (30.4%)
Spondylitis/Sacroiliitis, *n* (%)	227 (8.2%)	2 (4.5%)	200 (8.7%)	25 (6.0%)
Arthritis mutilans, *n* (%)	87 (3.2%)	0	66 (2.9%)	21 (5.1%)

Abbreviation: PsA, psoriatic arthritis.

subtypes included generalised pustular psoriasis (0.3%), localised pustular psoriasis (0.2%), flexural psoriasis (0.2%), palmoplantar non-pustular psoriasis (0.2%) and nail psoriasis (<0.1%). The rate of PsA in patients with erythrodermic psoriasis was significantly higher than non-erythrodermic psoriasis patients (25.5% vs. 12.8%, $p < 0.001$).

Data collected showed that the PsA cohort had more scalp (82.7% vs. 81.0%, $p = 0.04$) and nail involvement (73.3% vs. 53.3%, $p < 0.001$) compared to non-PsA patients. The PsA cohort recorded more severe cutaneous disease (BSA > 10%) compared to the non-PsA group (30.3% vs. 22.8%, $p < 0.001$) (Table 1).

3.3 | Treatment in PsA

Systemic and biological therapy were the mainstay treatment for PsA patients. A total of 1063 (39.4%) PsA patients received systemic therapy compared to 10.4% in the non-PsA cohort. The most frequently used systemic agents were methotrexate (87.8%), followed by acitretin (11.3%), systemic corticosteroids (4.5%), cyclosporine (3.9%) and hydroxyurea (0.7%) (Table 1). Only 1.6% of the PsA patients received biological therapy, notably this was higher than those without PsA (0.2%), $p < 0.001$. The biologics used included interleukin (IL)-17, IL-12/23 and tumour necrosis factor alpha (TNF-α) inhibitors.

Systemic therapy was used more frequently in patients with only axial PsA (54.5%), followed by those with both peripheral and axial PsA (46.9%) and least in peripheral PsA only (38.5%) (Table 3). In addition, patients with both axial and peripheral PsA received biological therapy more often (2.8%) compared to patients with peripheral only (1.5%) and axial only (1.3%) PsA patients.

3.4 | PsA and quality of life

QoL was significantly more affected in PsA versus non-PsA patients with a mean DLQI score of 10.12 ± 7.16 versus 9.52 ± 6.67 ($p < 0.001$), respectively. PsA patients reported significantly higher scores than non-PsA patients in all the domains of the DLQI except for the symptoms and feelings domain (Table 1), which was similar in both groups. A tenth of PsA patients had reported an extremely large effect on their DLQI compared to 7.4% in non-PsA patients ($p < 0.001$) (Figure 1).

4 | DISCUSSION

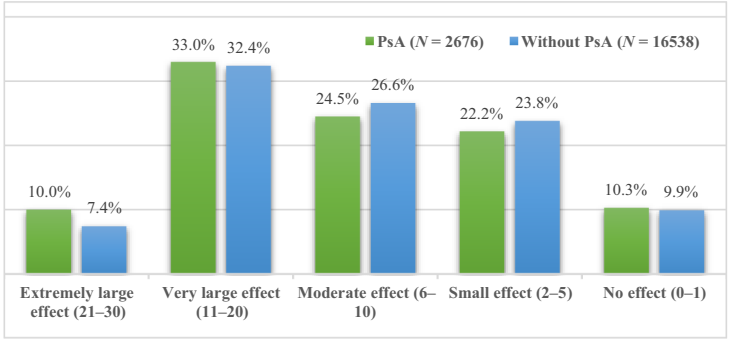
4.1 | Epidemiology and demography of psoriatic arthritis

The PsA prevalence among patients with psoriasis in Malaysia was similar to that in other Asian countries.[13] These findings were consistent with previous multi-ethnic studies done in Singapore.[14,15] The variation in the prevalence reported may indicate ethnic or genetic differences between different populations. Divergent distribution of HLA in different ethnic groups and other genetic determinants may account for these prevalence differences.[16] The overall prevalence of PsA was noted to be 1% lower than a previous local report[17] which only analysed data of psoriasis in adults. Our study, however, studied all patients in the MPR including paediatric patients, who had a lower incidence of PsA. A few studies revealed that the PsA incidence was

TABLE 3 Treatment of PsA.

Treatment	Peripheral PsA only $n = 2480$	Axial PsA only $n = 77$	Axial and peripheral PsA $n = 143$
Systemic therapy, *n* (%)	954 (38.5%)	42 (54.5%)	67 (46.9%)
Acitretin, *n* (%)	99 (4%)	12 (15.6%)	9 (6.3%)
Methotrexate, *n* (%)	848 (34.2%)	30 (39%)	55 (38.5%)
Systemic corticosteroid, *n* (%)	40 (1.6%)	3 (4%)	5 (3.5%)
Cyclosporine, *n* (%)	34 (1.4%)	2 (2.6%)	5 (3.5%)
Hydroxyurea, *n* (%)	5 (0.2%)	1 (1.3%)	1 (0.7%)
Biologic therapy, *n* (%)	38 (1.5%)	1 (1.3%)	4 (2.8%)

Abbreviation: PsA, psoriatic arthritis.



PsA, *n*	267	883	656	594	276
Non-PsA, *n*	1223	5352	4397	3930	1636
***p*-Value**	<0.001	0.51	0.02	0.08	0.50

FIGURE 1 DLQI scores in psoriasis patients with and without PsA.

highest in the age range 40–59 years, and it declined thereafter.[18,19] Our study demonstrated comparable findings (Table 2).

PsA is thought to have a stronger genetic heritability than psoriasis as evidenced by higher sibling heritability even though both are inter-related disorders.[20] The recurrence risk ratio (defined as the risk of disease manifestation in siblings versus the risk in the general population) for PsA was approximately 40 times higher among first-degree relatives of PsA patients compared with the general population.[16,21,22] Unfortunately, the data on family history of PsA was not recorded in the MPR for further analysis.

4.2 | Clinical risk factors and predictors of PsA

Several studies have shown that certain psoriasis-related factors such as psoriasis severity, nail involvement, scalp psoriasis and inverse-type psoriasis were associated with a higher likelihood of developing PsA.[23,24] In our study, patients with longer disease duration, severe psoriasis, scalp and nail involvement were more likely to have PsA, which are compatible with previous studies.[25–28] From the pathophysiologic perspective, the association between severe psoriasis and PsA may be explained by the systemic inflammation which leads to synovio-entheseal inflammation.[29,30] The prevalence of nail involvement has been reported to be higher in patients with PsA than that of patients with psoriasis only, particularly onycholysis and nail pitting.[6,25,31–33] This was also reflected in our cohort.

In 1973, Moll and Wright described five clinical subtypes of PsA,[34] which highlight the heterogeneity of the disease. The five clinical subtypes of PsA include distal interphalangeal arthropathy, asymmetrical oligo/monoarthritis, symmetrical polyarthritis, spondylitis/sacroiliitis and arthritis mutilans. Oligo/monoarthritis was the most reported subtype.[34] The oligoarticular subtype which affects four or fewer joints was also found to be the most common type of PsA in our study. The prevalence of oligo-/monoarthropathy was 37.9%, from our previous registry data reported by Mohd Affandi A et al.[17] This local report included only adult patients 18 years old and above and had a total of 15 974 subjects. Our study included patients of all ages and again oligo-/monoarthropathy was the most common subtype albeit at a much higher rate of 40.3%. Extra-musculoskeletal manifestations including uveitis and inflammatory bowel disease (IBD) were significantly increased in psoriasis patients with PsA.[35] Regrettably, data on uveitis or IBD was not collected in our study cohort.

4.3 | Smoking

Smoking was inversely associated with PsA development in our study cohort. Similar findings on the association of smoking and PsA development were also reported in other studies.[36] Research has shown that nicotine can activate the alpha-7 nicotinic acetylcholine receptor (α7nAChR) that inhibits intracellular pro-inflammatory pathways that are associated with the development of arthritis.[37] In a recent study, Chen et al. showed that a selective agonist of α7nAChR can inhibit the STAT3 and nuclear factor κB (NF-κB) signalling pathways, thereby impeding T helper 17-related inflammatory immune responses in psoriasis, and exerting a protective role in psoriasis inflammation.[38]

4.4 | Comorbidities in PsA

PsA is increasingly recognised as a systemic inflammatory disease associated with a higher incidence and prevalence of psychological and cardiometabolic comorbidities.[5,6,39,40] The abnormal inflammatory response in PsA resulting in release of pro-inflammatory cytokines, include TNF-α, IL-1β, IL-6, IL-12 and IL-17, which take part in the development of cardiovascular disease, hypertension, dyslipidaemia, insulin resistance and obesity.[41,42] The presence of comorbidities in PsA imposes a significant psychosocial impact on the QoL and also accelerated mortality.[43,44] In addition, cardiometabolic comorbidities in PsA patients are associated with more severe disease and a lower likelihood of response to therapy.[5] In our study, PsA patients were shown to have a statistically significant higher rate of dyslipidaemia, hypertension and diabetes mellitus, compared to the non-PsA cohort.

Marti et al. defined obesity as a pathological condition characterised by excessive fat deposition as compared to expected values for a given stature, sex and age.[45] In Malaysia, the National Health and Morbidity 2019 survey showed that 50.5% of the Malaysian population had a BMI of >25 kg/m^2. The association between obesity and PsA has been described in numerous studies,[46,47] and concurs with our study findings. The relationship between obesity and PsA is complex, bidirectional and may involve multiple mechanisms. Obesity is a state of chronic inflammation with higher levels of pro-inflammatory cytokines resulting in an increased risk of PsA development in predisposed subjects.[46,48] On account of the high predisposition to cardiometabolic disease in PsA patients, advice for healthy lifestyle and appropriate screening should be implemented in the long-term management of PsA patients for prevention of complications.[49]

4.5 | Treatment of psoriatic arthritis

The primary goal of PsA treatment is to maximise health related QoL, through the alleviation of symptoms, prevention of structural damage and complications, and normalisation of function and social participation.[50] Pharmacological treatment options for PsA have significantly increased over the past years. The treatment options now include not only conventional synthetic disease modifying antirheumatic drugs (csDMARDs) such as methotrexate, sulfasalazine, cyclosporine and leflunomide, but also other targeted biological disease-modifying antirheumatic drugs (bDMARDs) which target tumour necrosis factor (TNF), IL-17A or IL-12/23,

as well as novel targeted synthetic DMARDs (tsDMARDs) that inhibit phosphodiesterase-4 (PDE4) and Janus kinases (JAKs).[51] Rapid initiation of conventional synthetic DMARDs have been recommended as the first step in the treatment of peripheral joint involvement in PsA to halt the disease progression and prevent irreversible joint damage.[50–52] TNF-α and IL-17A inhibition should be started as first line DMARDs in axial PsA.[50] These bDMARDs have been shown to be effective in clinical trials and real-life experiences, in allaying arthritis, skin, enthesitis and dactylitis symptoms.[53]

Systemic treatment usage was considerably low in our cohort of psoriasis patients with PsA, and most patients receiving systemic therapy were on methotrexate. Only a small proportion of these patients received biologic therapies. Biologic therapies are considerably expensive when compared to conventional systemic immunomodulating therapies such as methotrexate and cyclosporine.[54] Even though biologic therapies could significantly improve the lives of PsA patients, the access to biologics remains a challenge for many patients with poor socioeconomic statuses. Under the local government healthcare system, funding for biologic therapies is insufficient, and is heavily reliant on national healthcare subsidies. Additional funding for biologics therapies may be obtained through other public sector organisations such as the Department of Public Service for current or retired civil servants; state government funding for state employed civil servants; the Medical Aids Foundation under the Ministry of Health; and Zakat for Muslims. Zakat or tithe is an obligatory almsgiving in which a part of the Muslims' wealth is given to the poor or other beneficiaries in Malaysia. There is a lack of published cost-effective analyses comparing various treatment modalities in PsA. Biologic therapies have been shown to exhibit the highest efficacy with favourable safety profiles in treating moderate-to-severe psoriasis arthritis.[54] Hence, high quality cost-effective studies should be carried out to translate current evidence into applicable evidence to guide government funding decisions for biologic therapy in PsA.

4.6 | Impact of PsA on health-related quality of life (HRQoL)

The presence of PsA exerts a deleterious effect on the QoL of psoriasis patients, affecting their emotional, psychological and physical well-being.[55,56] Several studies have revealed that the considerable psychological and functional burden of PsA was similar in magnitude to those of spondyloarthritis and rheumatoid arthritis.[57,58] The DLQI focuses primarily on cutaneous involvement and does not encompass the full impact of articular disease in PsA. In our study, patients with PsA had significantly poorer HRQoL than those with psoriasis alone, but no difference was found in the domain of symptoms and feelings scores. To gain a more complete profile of the burden of PsA, the DLQI should be used in parallel with other instruments that more specifically assess the impact of joint disease. There are generic or arthritis-specific patient-reported questionnaires which focus on physical impairment of PsA such as the Medical Outcomes Study Short Form-36 Health Survey, the EuroQol-5D, Health Assessment Questionnaire-Disability Index (HAQ-DI), Arthritis Impact Measurement Scale (AIMS) and PsA-specific quality of life (PsAQoL) instrument. Of note, the psychosocial burden of PsA was found to be far more important than musculoskeletal and skin involvement in improving HRQoL.[54] PsA patients often suffer from sleep disorders, fatigue, stress, mood disorders, poor body image and reduced work productivity.[45] Thus, the evaluation of patients with PsA should include the assessment of psychological parameters such as depression and anxiety, in addition to physical function.

4.7 | Limitations

Data on PsA disease activity, and radiographical features of the damaged joints, was not available for analysis. Additional data collection on family history of psoriatic arthritis, associated comorbidities including uveitis and inflammatory bowel disease, and PEST scores for all psoriasis patients would help in early detection and determine suitable choices of treatment.

5 | CONCLUSION

This study revealed that 12.7% of psoriasis in the MPR had PsA. A higher incidence of comorbidities, severe disease and impaired QoL. PsA patients were more likely to receive systemic and biological therapy compared to the non-PsA cohort. The risk factors for PsA development were family history of psoriasis, longer duration of disease, nail psoriasis and scalp involvement. They were more likely to receive systemic and biological therapy.

AUTHOR CONTRIBUTIONS

MM Tang was involved in study conception and design. Suganthy Robinson was involved in acquisition of data. Suganthy Robinson and MM Tang were involved in analysis and interpretation of data and critical revision. SF Goh and SB Wong were involved in drafting of manuscript. SF Goh, MM Tang and Suganthy Robinson were involved in literature review. All authors contributed to, read and approved the final manuscript.

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CONFLICT OF INTEREST STATEMENT

The authors hereby certify that, to the best of our knowledge, the work has not received financial support from any pharmaceutical

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DATA AVAILABILITY STATEMENT

Data sharing is not applicable to this article as no new data were created or analyzed in this study.

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REFERENCES

1. Armstrong AW, Read C. Pathophysiology, clinical presentation, and treatment of psoriasis: a review. *JAMA*. 2020;323(19):1945-1960.
2. Veale DJ, Fearon U. The pathogenesis of psoriatic arthritis. *Lancet*. 2018;391(10136):2273-2284.
3. Edson-Heredia E, Zhu B, Guo J, Maeda-Chubachi T, Lebwohl M. Disease burden and quality of life in psoriasis patients with and without comorbid psoriatic arthritis: results from National Psoriasis Foundation panel surveys. *Cutis*. 2015;95(3):173-178.
4. Gladman DD, Stafford-Brady F, Chang CH, Lewandowski K, Russell ML. Longitudinal study of clinical and radiological progression in psoriatic arthritis. *J Rheumatol*. 1990;17:809-812.
5. Karmacharya P, Ogdie A, Eder L. Psoriatic arthritis and the association with cardiometabolic disease: a narrative review. *Ther Adv Musculoskelet Dis*. 2021;13:1759720X2199827.
6. Ogdie A, Weiss P. The epidemiology of psoriatic arthritis. *Rheum Dis Clin N Am*. 2015;41(4):545-568.
7. Gelfand JM, Gladman DD, Mease PJ, et al. Epidemiology of psoriatic arthritis in the population of the United States. *J Am Acad Dermatol*. 2005;53(4):573.
8. Alinaghi F, Calov M, Kristensen LE, et al. Prevalence of psoriatic arthritis in patients with psoriasis: a systematic review and meta-analysis of observational and clinical studies. *J Am Acad Dermatol*. 2019;80(1):251-265.
9. Prey S, Paul C, Bronsard V, et al. Assessment of risk of psoriatic arthritis in patients with plaque psoriasis: a systematic review of the literature. *J Eur Acad Dermatol Venereol*. 2010;24:31-35.
10. Henes JC, Ziupa E, Eisfelder M, et al. High prevalence of psoriatic arthritis in dermatological patients with psoriasis: a cross-sectional study. *Rheumatol Int*. 2014;34(2):227-234.
11. WHO Expert Consultation. Appropriate body-mass index for Asian populations and its implications for policy and intervention strategies. *Lancet*. 2004;363(9403):157-163.
12. Finlay AY, Khan GK. Dermatology life quality index (DLQI)-a simple practical measure for routine clinical use. *Clin Exp Dermatol*. 1994;19(3):210-216.
13. Tam LS, Leung YY, Li EK. Psoriatic arthritis in Asia. *Rheumatology (Oxford)*. 2009;48(12):1473-1477.
14. Thumboo J, Tham SN, Tay YK, et al. Patterns of psoriatic arthritis in oriental. *J Rheumatol*. 1997;24(10):1949-1953.
15. Tey HL, Ee HL, Tan AS, Theng TS, Wong SN, Khoo SW. Risk factors associated with having psoriatic arthritis in patients with cutaneous psoriasis. *J Dermatol*. 2010;37(5):426-430.
16. Gladman DD, Anhorn KA, Schachter RK, Mervart H. HLA antigens in psoriatic arthritis. *J Rheumatol*. 1986;13(3):586-592.
17. Mohd Affandi A, Khan I, Ngah SN. Epidemiology and clinical features of adult patients with psoriasis in Malaysia: 10-year review from the Malaysian psoriasis registry (2007-2016). *Dermatol Res Pract*. 2018;2018:4371471.
18. Deike M, Brinks R, Meller S, Schneider M, Sewerin P. Risk of psoriatic arthritis depending on age: analysis of data from 65 million people on statutory insurance in Germany. *RMD Open*. 2021;7(3):e001975.
19. Karmacharya P, Crowson CS, Bekele D, et al. The epidemiology of psoriatic arthritis over five decades: a population-based study. *Arthritis Rheumatol*. 2021;73(10):1878-1885.
20. Chandran V, Schentag CT, Brockbank JE, et al. Familial aggregation of psoriatic arthritis. *Ann Rheum Dis*. 2009;68(5):664-667.
21. Karason A, Love TJ, Gudbjornsson B. A strong heritability of psoriatic arthritis over four generations—the Reykjavik psoriatic arthritis study. *Rheumatology (Oxford)*. 2009;48(11):1424-1428.
22. FitzGerald O, Haroon M, Giles JT, Winchester R. Concepts of pathogenesis in psoriatic arthritis: genotype determines clinical phenotype. *Arthritis Res Ther*. 2015;17(1):115.
23. Solmaz D, Eder L, Aydin SZ. Update on the epidemiology, risk factors, and disease outcomes of psoriatic arthritis. *Best Pract Res Clin Rheumatol*. 2018;32(2):295-311.
24. Zabotti A, De Lucia O, Sakellariou G, et al. Predictors, risk factors, and incidence rates of psoriatic arthritis development in psoriasis patients: a systematic literature review and meta-analysis. *Rheumatol Ther*. 2021;8(4):1519-1534.
25. Eder L, Law T, Chandran V, et al. Association between environmental factors and onset of psoriatic arthritis in patients with psoriasis. *Arthritis Care Res*. 2021;63(8):1091-1097.
26. Thumboo J, Uramoto K, Shbeeb MI, et al. Risk factors for the development of psoriatic arthritis: a population based nested case control study. *J Rheumatol*. 2002;29(4):757-762.
27. Rouzaud M, Sevrain M, Villani AP, et al. Is there a psoriasis skin phenotype associated with psoriatic arthritis? Systematic literature review. *J Eur Acad Dermatol Venereol*. 2014;28:17-26.
28. Ogdie A, Langan S, Love T, et al. Prevalence and treatment patterns of psoriatic arthritis in the UK. *Rheumatology*. 2013;52(3):568-575.
29. Scher JU, Ogdie A, Merola JF, Ritchlin C. Preventing psoriatic arthritis:focusing on patients with psoriasis at increased risk of transition. *Nat Rev Rheumatol*. 2019;15:153-166.
30. Eder L, Haddad A, Rosen CF, et al. The incidence and risk factors for psoriatic arthritis in patients with psoriasis: a prospective cohort study. *Arthritis Rheumatol*. 2016;68(4):915-923.
31. Aydin SZ, Castillo-Gallego C, Ash ZR, et al. Ultrasonographic assessment of nail in psoriatic disease shows a link between onychopathy and distal interphalangeal joint extensor tendon enthesopathy. *Dermatology*. 2012;225(3):231-235.
32. Castellanos-González M, Joven BE, Sánchez J, et al. Nail involvement can predict enthesopathy in patients with psoriasis. *J Dtsch Dermatol Ges*. 2016;14(11):1102-1107.
33. Love TJ, Gudjonsson JE, Valdimarsson H, Gudbjornsson B. Psoriatic arthritis and onycholysis—results from the cross-sectional Reykjavik psoriatic arthritis study. *J Rheumatol*. 2012;39(7):1441-1444.
34. Moll JM, Wright V. Psoriatic arthritis. *Semin Arthritis Rheum*. 1973;3(1):55-78.
35. Charlton R, Green A, Shaddick G, et al. Risk of uveitis and inflammatory bowel disease in people with psoriatic arthritis: a population-based cohort study. *Ann Rheum Dis*. 2018;77(2):277-280.
36. Eder L, Shanmugarajah S, Thavaneswaran A, et al. The association between smoking and the development of psoriatic arthritis among psoriasis patients. *Ann Rheum Dis*. 2012;71(2):219-224.
37. Bencherif M, Lippiello PM, Lucas R, Marrero MB. Alpha7 nicotinic receptors as novel therapeutic targets for inflammation-based diseases. *Cell Mol Life Sci*. 2011;68(6):931-949.
38. Chen Y, Lian P, Peng Z, et al. Alpha-7 nicotinic acetylcholine receptor agonist alleviates psoriasis-like inflammation through inhibition of the STAT3 and NF-κB signaling pathway. *Cell Death Dis*. 2022;8(1):141.

39. Ferguson LD, Siebert S, McInnes IB, Sattar N. Cardiometabolic comorbidities in RA and PsA: lessons learned and future directions. *Nat Rev Rheumatol*. 2019;15(8):461-474.
40. Gudu T, Etcheto A, de Wit M, et al. Fatigue in psoriatic arthritis - a cross-sectional study of 246 patients from 13 countries. *Joint Bone Spine*. 2016;83(4):439-443.
41. Ritchlin CT, Colbert RA, Gladman DD. Psoriatic arthritis. *N Engl J Med*. 2017;376(10):957-970.
42. Moghbeli M, Khedmatgozar H, Yadegari M, Avan A, Ferns GA, Ghayour MM. Cytokines and the immune response in obesity-related disorders. *Adv Clin Chem*. 2021;101:135-168.
43. Gudu T, Gossec L. Quality of life in psoriatic arthritis. *Expert Rev Clin Immunol*. 2018;14(5):405-417.
44. Husni ME, Merola JF, Davin S. The psychosocial burden of psoriatic arthritis. *Semin Arthritis Rheum*. 2017;47(3):351-360.
45. Martí A, Marcos A, Martínez JA. Obesity and immune function relationships. *Obes Rev*. 2001;2(2):131-140.
46. Thomsen RS, Nilsen TI, Haugeberg G, Gulati AM, Kavanaugh A, Hoff M. Adiposity and physical activity as risk factors for developing psoriatic arthritis: longitudinal data from a population-based study in Norway. *Arthritis Care Res*. 2021;73(3):432-441.
47. Love TJ, Zhu Y, Zhang Y, et al. Obesity and the risk of psoriatic arthritis: a population-based study. *Ann Rheum Dis*. 2012;71(8):1273-1277.
48. Soltani-Arabshahi R, Wong B, Feng BJ, Goldgar DE, Duffin KC, Krueger GG. Obesity in early adulthood as a risk factor for psoriatic arthritis. *Arch Dermatol*. 2010;146(7):721-726.
49. Elmets CA, Leonardi CL, Davis DMR, et al. Joint AAD-NPF guidelines of care for the management and treatment of psoriasis with awareness and attention to comorbidities. *J Am Acad Dermatol*. 2019;80(4):1073-1113.
50. Gossec L, Baraliakos X, Kerschbaumer A, et al. EULAR recommendations for the management of psoriatic arthritis with pharmacological therapies: 2019 update. *Ann Rheum Dis*. 2020;79(6):700-712.
51. Ogdie A, Coates LC, Gladman DD. Treatment guidelines in psoriatic arthritis. *Rheumatology*. 2020;59(Supplement_1):i37-i46.
52. Coates LC, van der Windt DA, O'Sullivan D, Soriano ER, Kavanaugh A. GRAPPA treatment recommendations: 2021 update. *J Rheumatol*. 2022;49(6 Suppl 1):52-54.
53. Raychaudhuri SP, Wilken R, Sukhov AC, Raychaudhuri SK, Maverakis E. Management of psoriatic arthritis: early diagnosis, monitoring of disease severity and cutting edge therapies. *J Autoimmun*. 2017;76:21-37.
54. Beyer V, Wolverton SE. Recent trends in systemic psoriasis treatment costs. *Arch Dermatol*. 2010;146(1):46-54.
55. Freites Nuñez D, Madrid-García A, Leon L, et al. Factors associated with health-related quality of life in psoriatic arthritis patients: a longitudinal analysis. *Rheumatol Ther*. 2021;8(3):1341-1354.
56. Haugeberg G, Michelsen B, Kavanaugh A. Impact of skin, musculoskeletal and psychosocial aspects on quality of life in psoriatic arthritis patients: a cross-sectional study of outpatient clinic patients in the biologic treatment era. *RMD Open*. 2020;6(1):e001.
57. Husted JA, Tom BD, Farewell VT, Schentag CT, Gladman DD. Description and prediction of physical functional disability in psoriatic arthritis: a longitudinal analysis using a Markov model approach. *Arthritis Rheum*. 2005;53(3):404-409.
58. Zink A, Thiele K, Huscher D, et al. Healthcare and burden of disease in psoriatic arthritis. A comparison with rheumatoid arthritis and ankylosing spondylitis. *J Rheumatol*. 2006;33(1):86-90.

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