South African Rheumatism and Arthritis Association 2024 guidelines for the management of peripheral spondyloarthritis

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Peripheral spondyloarthritis (SpA) includes psoriatic arthritis (PsA), inflammatory bowel disease (IBD)-associated arthritis, reactive arthritis and undifferentiated peripheral SpA. These South African guidelines offer information on diagnosis, assessment and therapy of peripheral SpA. Emphasis is placed on a multidisciplinary team, and a treat-to-target strategy with escalation of therapy if the target of minimal or very low disease activity is achieved. Screening for and treatment of comorbidities are paramount.

Keywords: peripheral spondyloarthritis, South Africa

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Spondyloarthritis (SpA) is an umbrella term that includes various inflammatory joint diseases affecting the axial spine as well as peripheral joints (Fig. 1).[1] These guidelines pertain to psoriatic arthritis (PsA), inflammatory bowel disease (IBD)-associated arthritis, reactive arthritis and undifferentiated peripheral SpA, and are to be used in conjunction with guidelines for the management of axial SpA and guidelines for the use of biological and targeted synthetic disease-modifying antirheumatic drugs.

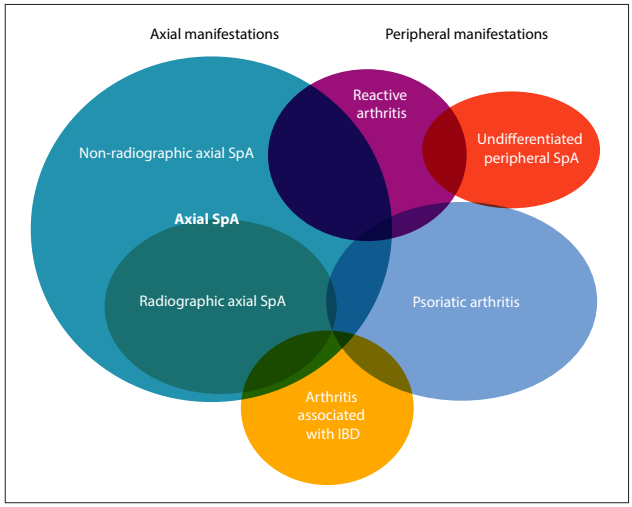

Fig. 1. Disorders that comprise the family of spondyloarthritides (reproduced with permission).[1] (SpA = spondyloarthritis; IBD = inflammatory bowel disease.)

Psoriatic arthritis

PsA is a chronic inflammatory arthritis that is associated with the skin disease psoriasis.[2] Psoriasis affects 1 - 3% of the general population, and approximately one-third of patients with psoriasis develop PsA.[3-5] Nail disease, severe skin disease, obesity and a positive family history seem to be the key risk factors for the development of PsA among psoriasis patients.[6] Both psoriasis and PsA significantly negatively impact the patient's health-related quality of life (HRQoL). Disease-modifying antirheumatic drugs (DMARDs) to treat these diseases have improved outcomes dramatically in recent years.

Diagnosis of psoriatic arthritis

The diagnosis of PsA is made by a combination of clinical findings supplemented by special investigations. The ClASsification for Psoriatic ARthritis (CASPAR) is a useful reference (Table 1).[7]

Assessment of psoriatic arthritis

PsA is a heterogeneous disease, and the group for research and assessment of psoriasis and PsA (GRAPPA) recommends that PsA be assessed in the following five domains: peripheral arthritis; skin and nail disease; axial arthritis; enthesitis; and dactylitis.[8]

Assessment of peripheral arthritis

Compared with rheumatoid arthritis (RA), peripheral joint involvement in PsA is much more extensive. The 28 tender joint count (TJC) and swollen joint count (SJC) used in RA do not represent all the joints affected in PsA. It is recommended that the 68 TJC and 66 SJC are used in PsA.[9,10] The South African Rheumatism and Arthritis Association recommends the Disease Activity in PsA (DAPSA) as a disease activity index for peripheral PsA (Table 2). Based on specific cut-off points, disease activity can be classified into states of remission, low, moderate, or high disease activity.

Skin assessment

In ~70% of patients, psoriasis will antedate the development of the musculoskeletal manifestations. In 10 - 15% of patients, there will be a simultaneous appearance in both skin and musculoskeletal manifestations, and in 10 - 15%, arthritis occurs before psoriasis.[11] In the last group of patients, there may be markers of PsA that may be pointers to the disease, e.g. enthesitis or dactylitis. The two commonly used outcome measures of skin involvement in psoriasis and PsA are the Psoriasis Area and Severity Index (PASI) and body surface area.[12]

Nail assessment

Nail involvement occurs in ~50% of patients with psoriasis, and as many as 80% of patients with PsA.[13-15] A modified Nail Psoriasis Severity Index (mNAPSI) can be used to score nail severity.[16,17]

Axial spine assessment

Axial involvement occurs in ~40% of patients with PsA.[18-20] Although there are similarities between axial SpA (axSpA) (also known as ankylosing spondylitis) and axial involvement in PsA, significant differences are present.[18] The assessment tools and outcome measures for axial involvement in PsA are borrowed from axSpA, including the ankylosing spondylitis disease activity score (ASDAS) or Bath ankylosing spondylitis disease activity index (BASDAI).[21]

Dactylitis assessment

Dactylitis is a term used to describe swelling of the entire digit, and is defined as a >10% difference in the circumference base of the digit compared with the normal contralateral digit.[21] Dactylitis occurs in ~40% of patients with PsA. A simple numerical count of the fingers involved is used in the Leeds Dactylitis Index.

Enthesitis assessment

Enthesitis is prevalent in 25 - 78% of patients with PsA, and may sometimes be the presenting feature.[22] It is thought that the "deep Koebner phenomenon" is a trigger for PsA. The most common entheseal indices are the Spondyloarthritis Research Consortium of Canada (SPARCC) index; Leeds Enthesitis Index (LEI) and Maastricht Ankylosing Spondylitis Enthesitis Score (MASES).

Global assessment

Minimal disease activity (MDA) is a 'state' of disease activity in PsA, and is a simple index that is widely used clinically observationally.[23] A patient achieves MDA when 5 of 7 criteria are met, and very low disease activity (VLDA) is achieved when 7 of 7 criteria are met (Table 2).

Laboratory measures

Inflammatory markers such as erythrocyte sedimentation rate and C-reactive protein are elevated in only ~50% of patients with PsA, despite having active disease. These measures may, however, have prognostic value.[24,25]

Management principles

Patient information and decision-making

The aim of treatment is to maintain a good quality of life and function. Treatment of each patient should be tailored to meet the needs of the patient according to the disease burden, taking into consideration the predominant manifestation (axial or peripheral arthritis, enthesitis, dactylitis, skin and nail) together with extra-articular manifestations and comorbidities. A management plan should be developed based on shared decision-making between patients and clinicians, predicated on patients' values, goals, preferences and comorbidities.

Patient education should include information about PsA disease and complications, assessment of disease, treatment goals, medications and adherence. Self-management interventions should be emphasised.

Referral to a rheumatologist and multidisciplinary team

Care of the PsA patient requires a multidisciplinary holistic approach, which might include a dermatologist, occupational therapist, podiatrist, physiotherapist, clinical psychologist or social worker, as appropriate. A rheumatology nurse can offer patient education and support, with positive effects on adherence to therapy and on HRQoL.[26]

Lifestyle interventions

Regular exercise and lifestyle modification, including smoking cessation and increased physical activity, and participation in patient support groups, should be encouraged. Disease-related problem-solving, attention to emotional wellbeing and communication skills are vital.

Comorbidities and extra-articular disease

The vast majority of PsA patients have one or more comorbidity, leading to premature mortality, functional impairment and reduced HRQoL. Obesity, accelerated atherosclerosis and cardiovascular events, non-alcoholic fatty liver disease, infections and osteoporosis are the major comorbidities in PsA, and need regular screening and evidence-based management.[27]

Screening for hepatitis B, hepatitis C, HIV and tuberculosis should be done at presentation. The vaccination status, pregnancy plans, contraception and lactational status of the patient should be regularly reviewed and discussed.[28]

Goal of therapy

The goal of therapy is to achieve MDA or VLDA in the global assessment of PsA, or low disease activity using DAPSA if peripheral arthritis.

Treat to target

Clinicians must aim to achieve MDA in all patients as soon as possible – aiming for 50% improvement in disease activity score within 3 months, and target reached by 6 months. A treat-to-target paradigm should be followed to achieve this. This strategy entails:

- use of composite disease activity score (global assessment of PsA or DAPSA if peripheral arthritis) at each visit
- frequent follow-up every 1 - 3 months in the first 6 - 18 months of treatment
- escalation or switching of therapy until the goal is achieved (Table 3).

Details of therapies

Non-steroidal anti-inflammatory drugs

Non-steroidal anti-inflammatory drugs (NSAIDs) are useful and frequently used for symptomatic treatment in patients with PsA,

Table 1. CASPAR criteria: Confirmed inflammatory articular disease (joint, spine, or entheseal) with ≥3 points is classified as having definite PsA

Criterion	Details	Points
Skin psoriasis (maximum 2 points)	Present	2
	or previously present	1
	or family history of psoriasis (first- or second-degree relative)	1
Nail dystrophy	Oncolysis, pitting, hyperkeratosis	1
Dactylitis	Current or documented by rheumatologist in past	1
X-ray hands or feet	Juxta-articular new bone formation	1
Rheumatoid factor	Negative	1

CASPAR = ClASsification for Psoriatic Arthritis; PsA = psoriatic arthritis.

Table 2. Disease activity assessment tools for psoriatic arthritis

Tool	Measurement	Assessment
Global assessment of PsA	TJC ≤1 SJC ≤1 PASI ≤1 or body surface area ≤3% Pain VAS* ≤1.5 cm PGA VAS† ≤2 cm HAQ-DI ≤0.5 Tender entheseal points ≤1	MDA 5 of 7 criteria are met VLDA 7 of 7 criteria are met
DAPSA (for peripheral arthritis)	66SJC + 68TJC + PGA (cm) + pain (cm) + CRP mg/dL	Remission ≤4 LDA >4 ≤14 MDA >14 ≤28 HDA >28

PsA = psoriatic arthritis; TJC = tender joint count; SJC = swollen joint count; PASI = psoriasis area and severity index; VAS = visual analogue scale; PGA = patient global assessment; HAQ-DI = health assessment questionnaire disability index; DAPSA = disease activity in PsA; CRP = C-reactive protein; MDA = minimal disease activity; VLDA = very low disease activity; LDA = low disease activity; HDA = high disease activity.
*Pain VAS 'How would you describe the overall level of joint pain during the last week?' (0 = none; 10 = very severe).
†PGA VAS 'How active was your rheumatic disease on average in the last week?' (0 = not active; 10 = very active).

Table 3. Stepwise algorithm for therapy in psoriatic arthritis

Therapy	Peripheral arthritis/dactylitis	Enthesitis	Axial disease	Inadequate response within 3 months
First-line therapy	Polyarthritis: MTX (other csDMARDs if MTX not tolerated/contraindicated) Mono-/oligoarthritis: intra-articular GC and NSAIDs	NSAIDs GC injection MTX	NSAID	Proceed to second-line therapy
Second-line therapy	Combination or switch csDMARD	b/tsDMARD (TNFi/IL-17i/JAKi/IL-12-23i/IL-23i/PDE4i/abatacept)	b/tsDMARD (TNFi/IL-17i/JAKi)	Proceed to third-line therapy
Third-line therapy	b/tsDMARD (TNFi/IL-17i/JAKi/IL-12-23i/IL-23i/PDE4i/abatacept)	Alternative b/tsDMARD (TNFi/IL-17i/JAKi/IL-12-23i/IL-23i/PDE4i/abatacept)	Alternative b/tsDMARD (TNFi/IL-17i/JAKi)	Proceed to third-line therapy
Fourth-line therapy	Alternative b/tsDMARD			-
De-escalation	After sustained remission for 6 months, consider slow taper of b/tsDMARD, maintain csDMARD			Restart b/tsDMARD if flares

MTX = methotrexate; DMARD = disease-modifying antirheumatic drug; csDMARD = conventional synthetic DMARD; GC = glucocorticoid; NSAID = non-steroidal anti-inflammatory drug; b/tsDMARD = biologic/targeted synthetic DMARD; TNFi = tumour necrosis factor inhibitor; IL-17i = interleukin-17 inhibitor; IL-12-23i = interleukin 12/23 inhibitor; IL-23i = interleukin-23 inhibitor; JAKi = Janus kinase inhibitor; PDE4i = phosphodiesterase-4 inhibitors.

particularly those with oligoarthritis, axial disease and enthesitis. They have no disease-modifying properties in peripheral PsA.[29]

Glucocorticoids

Systemic glucocorticoids (GC) are not advocated for use in PsA. The risk of pustular psoriasis when stopping or tapering systemic corticosteroids is small but well known.[30,31] Intra-articular injections of GC are effective and are generally used for mono- or oligoarticular flares of PsA.[32]

Disease-modifying antirheumatic drugs

Disease-modifying antirheumatic drugs (DMARDs) are divided into three broad groups: conventional synthetic DMARDs (csDMARDs); biologic DMARDs (bDMARDs); and targeted synthetic DMARDs (tsDMARDs).

The csDMARDs to treat PsA include methotrexate (MTX), leflunomide (LEF), and sulfasalazine (SSZ). Antimalarial therapy (chloroquine or hydroxychloroquine) may worsen skin lesions and

should be avoided in PsA. The bDMARDs are either biologic original (boDMARDs) or biosimilar (bsDMARDs), and include:

- tumour necrosis factor inhibitors (TNFis): either receptor blockers (etanercept (original and biosimilar)), monoclonal antibodies (infliximab (original and biosimilar)), or adalimumab (original and biosimilar) and golimumab)
- abatacept
- IL-17 inhibitor (IL-17i) (secukinumab and ixekizumab)
- IL-12/23 inhibitors (IL-12/23i) (ustekinumab), and IL-23 inhibitor (IL-23i) (guselkumab.

The tsDMARDs include the Janus kinase inhibitors (JAKis) tofacitinib, and upadacitinib. The phosphodiesterase 5 inhibitor PDE4i (apremilast) also has a place in the therapy of mild PsA.

Important notes on b/tsDMARDs

The use of combination bDMARDs is not recommended.

In SpA (axial or peripheral), there is little evidence that co-prescription of MTX with b/tsDMARDs is necessary.

All b/tsDMARDs should be initiated by a rheumatologist or dermatologist. All patients with a rheumatic disease who are on b/tsDMARDs must be included, with patient consent, in the South African Rheumatism and Arthritis Association (SAARA) biologics registry (https://www.saraa.co.za).

Before commencing b/tsDMARD, appropriate screening and treatment for latent tuberculosis, hepatitis B, hepatitis C and HIV, in addition to vaccination, should be done.

Choice of b/tsDMARD

The b/tsDMARDs may be used in any order. Consideration of patient preference, tuberculosis risk, comorbidities, route of administration and costs is appropriate. There are possible different efficacies with respect to skin and arthritis domains that may influence the rheumatologist's choice of b/tsDMARD (Table 4[34-36]). In uveitis, monocloncal TNFi therapy is preferred, as the soluble receptor TNFi (etanercept), IL-17i (including sekukinumab and ixekizumab) and JAKi have not been shown to be efficacious. Similarly, the soluble receptor TNFi (etanercept) and IL-17i should not be used in patients with active IBD. For patients with significant skin involvement, preference should be given to MTX/IL-17i/IL-12/23i or IL-23i therapies.

Switching therapy

Should the patient have an inadequate response to one b/tsDMARD after 3 months, or toxicity, switching to another is indicated.

Details of therapies for specific domains

Peripheral arthritis

First-line therapy: MTX (7.5 mg - 25 mg weekly). In the case of mono- or oligoarthritis, a good response may be obtained with NSAIDs and/or intra-articular GC.

Second-line therapy: addition of/switching to SSZ or LEF is recommended. These may be monotherapy or combination therapy.

Third-line therapy: b/tsDMARDS.

Axial disease

First-line therapy: NSAIDs. Two courses of at least two different NSAIDs at the maximum recommended doses/maximum tolerated doses over 4 weeks. Importantly, csDMARDs have no proven benefits in axial disease. Intra-articular GC into the sacro-iliac joint(s) can be considered.

Second-line therapy: patients who have an inadequate response to NSAIDs (ASDAS ≥2.1 or BASDAI ≥4) should be considered for b/tsDMARD therapy. The efficacy of IL-12/23i or IL-23i in psoriatic axial disease is unclear.[37-39]

Enthesitis

First-line therapy: NSAIDs. Intralesional corticosteroid injections can be tried, but there is no evidence of benefit. A recent study showed MTX resolved enthesitis in 43.1% of PsA patients.[40]

Second-line therapy: b/tsDMARDs.

Dactylitis

First-line therapy: NSAIDs.
Second-line therapy: MTX.
Second-line therapy: b/tsDMARDs.

SARAA eligibility criteria for b/tsDMARD therapies for PsA

- Polyarthritis treated with at least two conventional DMARDs (maximal dosages, sequentially or in combination) with lack of improvement (50% of DAPSA) within 3 months or failure to achieve MDA within 6 months.
- Severe persistent oligoarthritis with a major demonstrable influence on wellbeing of the patient where treatment has failed, with at least two csDMARDs and appropriate intra-articular therapy with lack of improvement within 3 months or failure to achieve MDA within 6 months.
- Axial disease with poor response to NSAIDs (as per SARAA recommendation for axSpA).
- Refractory dactylitis, enthesitis or extra-articular disease.

Table 4. Impact of DMARDs on specific SpA domains

Condition	csDMARDs	Monoclonal TNFi	TNF receptor blocker	IL-17i	IL-12/23i	IL-23i	JAKi	PDE4i
Arthritis	++	++	++	++	++	++	++	+
Axial disease	–	++	++	++	?	?	++	–
Enthesitis	+	+	+	+	+	++	+	+
Dactylitis	+	+	+	+	+	++	++	+
Skin	+	++	++	+++	+++	+++	+/++	++
Nail dystrophy	–	++	++	+++	+++	+++	++	+
Uveitis	+	++	×	×	?	?	?	–
IBD	+	++	×	×	++	++	CD + UC ++	+

DMARD = disease-modifying antirheumatic drug; SpA = spondyloarthritis; csDMARDs = conventional synthetic DMARDs; TNFi = tumour necrosis factor inhibitor; IL-17i = interleukin 17 inhibitor; IL-23i = interleukin-23 inhibitor; IL-12/23i = interleukin 12/23 inhibitor; JAKi = Janus kinase inhibitor; PDE4i = phosphodiesterase-4 inhibitors; + = effective; – = ineffective; × = aggravates; ? = unknown; IBD = inflammatory bowel disease; CD = Crohn's disease; UC = ulcerative colitis.

Inflammatory bowel disease-associated arthritis

IBD-associated arthritis occurs in patients with Crohn's disease or ulcerative colitis.[41] Enteropathic arthritis can also occur in other gastrointestinal problems, namely, coeliac disease, Whipple's disease and intestinal bypass surgery. Patients with IBD-associated arthritis share many of the clinical features of the other SpAs. Of note, degenerative joint and spine disease, fibromyalgia syndrome, osteoporosis and gout are common causes of musculoskeletal symptoms in patients with IBD. This group of disorders is best managed by a multidisciplinary team, which includes a gastroenterologist, a rheumatologist and an ophthalmologist if necessary.

Type I disease

Mono- or oligoarticular, and generally self-limiting. Arthritis may occur early in the disease course and may be associated with erythema nodosum and other extra-articular manifestations. Flares in arthritis coincide with a worsening of the IBD, and treatment of the IBD often results in a resolution of arthritis.

Symptomatic treatment with bed rest and intra-articular corticosteroid injections. NSAIDs may be used with caution after discussion with the gastroenterologist.[42] If persistent, SSZ or MTX may be considered.[43,44] Poor responders may require monoclonal TNFi: infliximab, golimumab, or adalimumab.[45] Alternatively, IL-12/23 inhibitor (ustekinumab) can be used, noting the different drug dosing regimens. The soluble receptor TNFi (etanercept) and IL-17i are not recommended in IBD.

Type II disease

Polyarticular and erosive, affecting large and small joints and has an independent course to the IBD. This arthritis is strongly associated with uveitis.

Treatment with SSZ is recommended as first-line therapy. With an inadequate response, consider cyclosporine, or LEF.[46] If there is a poor response, monoclonal TNFi including infliximab, golimumab, or adalimumab may be used.[45] The soluble receptor TNFi (etanercept) and IL-17i are not recommended in IBD.

Type III disease

Peripheral and axial joints. The spondylitis generally precedes the onset of IBD and runs an independent course.

Treatment includes NSAIDs, SSZ, MTX, LEF and TNFi. Isolated sacroiliitis is usually non-progressive and responds to NSAIDs. Failure to respond to NSAIDs is an indication for b/tsDMARD therapy.[47]

Reactive arthritis

Reactive arthritis (ReA) is an inflammatory arthritis that manifests several days to weeks after a gastrointestinal or genitourinary infection. Because the pathogens cannot be cultured from the affected joints, this is not a septic arthritis but rather an aberrant immune response to the preceding infection. Organisms causing ReA include salmonella, shigella, yersinia, campylobacter and chlamydia, with HIV, *Escherichia coli*, *Clostridioides difficile* and *Chlamydia pneumoniae* recently added to the list of causative agents. The triad of arthritis, urethritis and conjunctivitis was previously called 'Reiter syndrome'.

More common in men, and in patients who are HLA-B27 positive, ReA typically presents as an asymmetrical oligoarthritis of the lower limbs. While large joints are most often involved, the small joints of the hands may also be affected. Enthesitis, including plantar fasciitis or Achilles tendon enthesitis, may also be noted, as may dactylitis and sacroiliitis. Other features include urethritis, iritis, conjunctivitis and mucocutaneous lesions (balanitis, keratoderma blenorrhagicum).

ReA is usually self-limiting, with most patients having a complete resolution of the articular symptoms within 6 months. Five to 10% of patients develop persistent arthritis/axial SpA. Prognostic markers for chronicity include HLA-B27 positivity, positive family history for SpA and the presence of chronic gut inflammation. Apart from increasing the probability of chronicity, HLA-B27 positivity is associated with severe disease, frequent spine involvement and extra-articular features.

Management

Symptomatic treatment of the joints using NSAIDs and intra-articular steroid injections is recommended. Treatment of antecedent infection with antibiotics is not recommended, although there is controversy regarding benefits of long-term antibiotic treatment of chlamydia-related ReA.[48] Patients with persistent (>3 - 6 months) peripheral joint involvement may require csDMARDs, including SSZ or MTX. Failure to respond to csDMARDs is an indication for TNFi. Patients with axial involvement should follow the axSpA guidelines.

SARAA eligibility criteria for b/tsDMARD therapies for other SpA (including ReA, IBD-associated arthritis and undifferentiated SpA)

Failure of conventional therapy for peripheral arthritis, enthesitis, dactylitis, axSpA or severe extra-articular disease.

Summary points for the management of peripheral SpA

- Disease activity is measured with the global assessment of PsA, MDA or VLDA, and the goal is MDA or VLDA, or DAPSA if peripheral arthritis, with a goal of low disease activity.
- A treat-to-target approach should be used, with frequent assessments and escalation or switching of therapy until the goal is achieved.
- Screening and management of comorbidities are essential, and a holistic approach with a multidisciplinary team is recommended.

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