Chapter

Practical Management of Psoriasis in Special Clinical Circumstances

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Abstract

This chapter thoroughly discusses the management strategies for psoriasis within complex clinical scenarios and special patient groups. It examines therapeutic options and safety considerations for systemic and biologic therapies in patients who have coexisting hepatic or renal disease, cardiovascular issues, malignancies, or inflammatory bowel disease, and addresses specific cases such as pregnancy and lactation. Furthermore, the management of psoriasis complicated by psoriatic arthritis, latent tuberculosis, or neurological disorders such as multiple sclerosis is detailed. The chapter emphasizes evidence-based, personalized treatment approaches informed by contemporary clinical trials and real-life patient data.

Keywords: psoriasis, treatment algorithm, pregnancy, malignancy, psoriatic arthritis, biologic therapy, guideline

1. Introduction

Psoriasis is a chronic inflammatory skin condition linked with significant systemic complications and numerous comorbidities. Although standard treatments often effectively control disease activity in many patients, certain clinical circumstances, including pregnancy, impaired organ function, cancer, and coexisting autoimmune or infectious disorders, require customized therapeutic approaches. Managing psoriasis in these specific groups involves carefully balancing treatment efficacy with safety, taking into account altered drug metabolism, immune-related risks, and particular vulnerabilities. This chapter provides a practical, evidence-based framework to support clinical decision-making in complex psoriasis cases, focusing on achieving optimal dermatological outcomes and integrated multidisciplinary care.

2. Pregnancy and breastfeeding

The effect of pregnancy on psoriasis is variable; symptoms may improve in some patients yet worsen in others, highlighting the need for individualized therapeutic strategies based on clinical presentation and established safety profiles. Topical therapies remain the cornerstone of treatment for mild psoriasis during pregnancy. Emollients and oatmeal-based baths are universally safe and should be used liberally to maintain skin barrier function and reduce inflammation. Low- to

moderate-potency topical corticosteroids are preferred, particularly when applied in limited quantities to localized plaques. High-potency corticosteroids may be used cautiously for short periods in refractory cases but are associated with potential risks, including fetal growth restriction if extensively applied or used long-term. Topical calcineurin inhibitors, such as tacrolimus and pimecrolimus, although off-label in pregnancy, are considered relatively safe for sensitive areas such as the face and intertriginous zones due to their minimal systemic absorption [1].

Phototherapy, specifically NB-UVB, is considered safe for treating moderate-to-severe psoriasis throughout all trimesters and poses no known risk to the fetus; however, as prolonged NB-UVB therapy may lower maternal folate levels, supplementing with folic acid during early pregnancy is recommended. While a standard course of NB-UVB treatment did not affect serum or red blood cell folate levels in psoriasis patients, El-Saie et al. found that high cumulative doses of NB-UVB led to folate photodegradation and a reduction in serum folate levels, with this effect being directly linked to the total cumulative dose, highlighting the potential risk associated with prolonged NB-UVB therapy [2, 3]. In contrast, psoralen plus ultraviolet A (PUVA) therapy is contraindicated during pregnancy due to its mutagenic potential and should be avoided entirely [4].

In patients requiring systemic treatment, cyclosporine remains the most studied and accepted systemic agent during pregnancy. Short-term use at the lowest effective dose is recommended, and careful monitoring for hypertension and nephrotoxicity is essential. Among biologic therapies, TNF-α inhibitors are the preferred class for use during pregnancy [5]. Certolizumab pegol, in particular, is favored due to its lack of an Fc fragment, which minimizes placental transfer. The CRIB study confirmed negligible fetal exposure to certolizumab pegol, establishing it as the biologic of choice for pregnant women requiring systemic disease control [6]. Etanercept, a fusion protein, exhibits substantially lower placental transfer than IgG1 monoclonal antibodies such as adalimumab and infliximab, with reported cord blood:maternal serum ratios as low as 1:14 [7]. Importantly, the use of TNF inhibitor exposure after the 20th gestational week should be minimized as they have been associated with persistence of the drug in neonatal serum for several months, prompting recommendations to avoid live vaccines, such as BCG, for at least 6 months postpartum. On the other hand, current data have not demonstrated any increased risk of congenital malformations or low birth weight associated with in utero exposure to TNF-α inhibitors [5].

Data on IL-17 and IL-23 inhibitors during pregnancy are limited, and these agents should generally be avoided unless no safer alternative exists and disease severity mandates their use [5]. Agents that are unequivocally contraindicated during pregnancy include methotrexate, acitretin, and topical retinoids such as tazarotene. Methotrexate is highly teratogenic and associated with pregnancy loss and congenital anomalies including microcephaly, craniosynostosis, tetralogy of Fallot and limb defects [8]. Acitretin possesses a notably prolonged elimination half-life, significantly increasing the risk of severe teratogenic effects; consequently, reliable contraception must be maintained for an extended duration even following cessation of therapy. Similarly, topical tazarotene, classified as a retinoid derivative, is contraindicated during pregnancy due to its recognized teratogenic potential [9].

Most topical therapies, including emollients and low-potency corticosteroids, are considered safe during lactation, provided that medications are not directly applied to the nipple-areola complex prior to nursing. NB-UVB phototherapy remains a safe option. Certolizumab pegol is again the preferred biologic during breastfeeding due to its minimal secretion into breast milk. Other TNF-α inhibitors may also be

acceptable but require case-by-case evaluation, ideally in consultation with pediatrics. Methotrexate, acitretin, and tazarotene are contraindicated during breastfeeding because of potential drug transfer and toxic effects on the infant [10].

3. Hepatic impairment

The management of psoriasis in patients with hepatic impairment requires particular caution due to the hepatic metabolism of many systemic therapies and the frequent co-occurrence of liver disease, especially non-alcoholic fatty liver disease (NAFLD). Psoriasis patients have up to a twofold increased risk of NAFLD, driven by shared risk factors such as metabolic syndrome, obesity, and chronic inflammation. Careful selection of therapy is essential to avoid further hepatic injury while achieving disease control [11].

Baseline evaluation should include liver function tests (ALT, AST, GGT, ALP, bilirubin) and mandatory screening for hepatitis B and C (HBsAg, anti-HBc, anti-HBs). Imaging studies, including liver ultrasound or transient elastography, may be necessary to assess fibrosis. In patients with cirrhosis or decompensated liver disease, systemic immunosuppressives should be used with extreme caution or avoided altogether [12].

For mild cases, treatment primarily involves topical therapies such as emollients, corticosteroids of low to moderate potency, vitamin D analogs, and calcineurin inhibitors. Additionally, NB-UVB phototherapy serves as a highly effective, non-hepatotoxic alternative for patients with extensive disease who are unsuitable for systemic treatment [13].

Methotrexate, a key systemic therapy for moderate-to-severe psoriasis, carries a notable risk of liver toxicity. It can lead to cumulative liver fibrosis and, in rare instances, cirrhosis. Patients with any pre-existing liver issues should generally avoid methotrexate. Additionally, non-invasive tests like FibroScan or a liver biopsy might be necessary when considering long-term methotrexate treatment. Similarly, acitretin, although primarily metabolized hepatically, is associated with alterations in lipid metabolism and potential hepatotoxicity, making it an unfavorable option in patients with active liver disease or dyslipidemia [14].

Cyclosporine, another systemic agent with hepatic metabolism, may cause cholestasis or hepatocellular injury, though less commonly than nephrotoxicity. Its use should be reserved for short-term indications in carefully monitored patients with only mild hepatic impairment, and it should be avoided in cases of significant liver dysfunction. On the other hand, the oral phosphodiesterase-4 inhibitor apremilast is generally well-tolerated in patients with hepatic impairment and does not require dose adjustment in mild to moderate liver dysfunction [14].

Biologic therapies offer a safer alternative for psoriatic patients with hepatic comorbidities. IL-17 (secukinumab, ixekizumab) and IL-23 inhibitors (guselkumab, risankizumab, tildrakizumab) are metabolized primarily by proteolysis, avoiding hepatic cytochrome P450 pathways. Long-term data have not shown increased hepatotoxicity with these agents [15].

Ustekinumab, an IL-12/23 inhibitor, has been used successfully in hepatitis B carriers; however, cohort data report a 17.4% reactivation rate in HBsAg-positive patients not receiving antiviral prophylaxis, while no reactivations occurred in those on prophylaxis [16]. Similarly, TNF-α inhibitors carry a substantial risk of hepatitis B virus (HBV) reactivation, particularly with infliximab and adalimumab. Reactivation

rates up to 39% have been reported in HBsAg-positive patients without prophylaxis, compared to less than 10% in those receiving antiviral therapy [17].

IL-17 inhibitors appear safer in this context. A prospective study reported 0% reactivation in patients receiving secukinumab alongside antiviral prophylaxis, versus 15.2% without it. While IL-23 inhibitors have limited data, no HBV reactivation has been reported in phase III trials, though rare isolated cases exist. Their safety profile remains promising for HBsAg-negative/anti-HBc-positive patients and may be cautiously considered under antiviral cover [15, 18].

For patients with chronic hepatitis B infection, antiviral prophylaxis with nucleoside analogs should be initiated before starting immunosuppressive therapy to reduce the risk of reactivation. Prophylaxis should continue for 6 to 12 months after treatment cessation, particularly for TNF-α and IL-12/23 blockers. For patients with hepatitis C infection, recent advances in antiviral therapy have improved the management of coexistent HCV and psoriasis, allowing more flexibility in systemic treatment selection once viral eradication is achieved [15].

Ultimately, management of psoriasis in the setting of hepatic impairment requires an individualized approach. Topical and phototherapeutic options should be optimized, hepatotoxic systemic agents minimized, and biologic therapies should be selected preferentially where indicated. Close collaboration with hepatologists is advisable for complex cases involving advanced fibrosis, cirrhosis, or concomitant viral hepatitis.

4. Renal impairment

Renal impairment presents a significant clinical challenge in the management of psoriasis, given the altered pharmacokinetics of many systemic therapies and the increased susceptibility to drug-induced nephrotoxicity. Chronic kidney disease (CKD) is more prevalent among patients with moderate-to-severe psoriasis compared to the general population, with systemic inflammation, hypertension, diabetes, and medication side effects contributing to renal decline. Furthermore, psoriasis itself has been recognized as a systemic inflammatory condition capable of promoting endothelial dysfunction and renal injury independently of traditional risk factors [13].

Comprehensive baseline renal evaluation is mandatory before initiating systemic treatment in psoriatic patients, particularly those with known risk factors or comorbidities. Laboratory assessments should include serum creatinine, estimated glomerular filtration rate (eGFR), blood urea nitrogen (BUN), electrolytes, and urinalysis to detect proteinuria or hematuria. In patients with moderate (eGFR 30–59 mL/min/1.73 m^2) or severe (eGFR <30) renal impairment, systemic therapy selection must be adjusted accordingly to minimize further renal insult [19].

For patients with limited cutaneous involvement, topical therapies remain the primary treatment option and pose no risk to renal function. Emollients, corticosteroids, vitamin D analogs, and topical calcineurin inhibitors are all safe. Narrowband UVB phototherapy also provides an effective and kidney-friendly treatment modality, particularly valuable in patients with moderate-to-severe disease who are not candidates for systemic immunomodulation. In cases requiring systemic intervention, biologics and certain oral agents offer safer profiles compared to traditional immunosuppressants. **Table 1** summarizes the renal safety, dialysis compatibility, and known adverse events of systemic psoriasis treatments.

Systemic agent	Use in CKD	Use in dialysis	Renal adverse events
Methotrexate	Contraindicated in eGFR <60	Not recommended	Risk of toxic accumulation, myelosuppression, mucositis
Cyclosporine	Avoid due to dose-dependent nephrotoxicity	Not recommended	Acute and chronic nephrotoxicity, hypertension
Acitretin	Use with caution	Caution advised	No direct renal toxicity; worsen hyperlipidemia, xerosis
Apremilast	Safe with dose reduction if eGFR <30	Can be used at 30 mg/day	Rare case reports of renal tubular acidosis
TNF-α inhibitors	Safe in stable renal function	Can be used; off-dialysis day	Rare acute kidney injury; infection risk in CKD
IL-17 inhibitors	Safe; no renal clearance required	Can be used; off-dialysis day	No reported renal adverse events
IL-23 inhibitors	Safe; primarily cleared proteolytically	Can be used; off-dialysis day	No reported renal adverse events
Ustekinumab	Safe; no dose adjustment needed	Can be used; off-dialysis day	No renal toxicity observed in trials or registries

Table 1.
Renal safety and dialysis considerations of systemic treatments for psoriasis [9, 19–21].

5. Diabetes mellitus

Patients with moderate-to-severe psoriasis are at significantly increased risk for developing type 2 diabetes, independent of obesity or metabolic syndrome. This is largely driven by chronic systemic inflammation involving TNF-α, IL-6, and IL-17, which contribute to insulin resistance and endothelial dysfunction. A baseline workup should include HbA1c, lipid profile, blood pressure, BMI, and screening for diabetic complications, such as nephropathy and retinopathy, which may impact treatment selection [22].

Topical therapies and NB-UVB phototherapy remain safe options for limited or moderate disease, with no adverse effects on glycemic control. Prolonged use of potent topical corticosteroids should be limited due to potential systemic absorption and glucose dysregulation [23].

Among systemic agents, methotrexate requires close hepatic monitoring because diabetics are at higher risk for NAFLD. Acitretin can exacerbate dyslipidemia and should be avoided in patients with poorly controlled lipid profiles or cardiovascular disease. Cyclosporine is usually not favored due to potential side effects like hypertension, nephrotoxicity, and increased insulin resistance; its link to new-onset diabetes has been well-established in transplant patients [12].

Apremilast has shown a metabolically neutral profile and favorable safety in real-world use among diabetic patients. Observational studies indicate that it does not lead to insulin resistance and may promote modest weight loss, offering additional benefit in overweight individuals with psoriasis [24].

Biologic therapies are extensively utilized among diabetic patients and demonstrate excellent safety profiles. TNF-α inhibitors, such as adalimumab and etanercept, have shown modest improvements in insulin sensitivity in some cohorts [25]. IL-17 and IL-23 inhibitors are metabolically neutral and have not been associated with worsening glucose levels in real-world studies [26]. Moreover, by reducing systemic

inflammation, these agents may indirectly contribute to improved metabolic homeostasis, particularly in patients with underlying insulin resistance or metabolic syndrome. Ustekinumab (anti-IL12/23) is similarly safe and convenient for patients managing multiple comorbidities due to its long dosing interval [12].

Close collaboration with endocrinology, careful monitoring of metabolic parameters, and patient education regarding lifestyle modification are essential components of successful long-term management.

6. Congestive heart failure

Congestive heart failure (CHF) is an important comorbidity that influences treatment decisions in patients with moderate-to-severe psoriasis. The presence of CHF complicates psoriasis management due to the cardiovascular risks associated with several systemic treatments, particularly biologic agents targeting the TNF-α pathway. Therefore, careful assessment of cardiac function and tailored therapeutic strategies are essential for safe and effective management [27].

Baseline evaluation of psoriatic patients with known or suspected CHF should include determination of the New York Heart Association (NYHA) functional class and measurement of left ventricular ejection fraction (LVEF). Patients with NYHA class III or IV symptoms or an LVEF less than 40% are considered high-risk and require especially cautious therapeutic selection [13].

In terms of conventional systemic therapies, methotrexate may be cautiously considered in patients with stable, compensated CHF. Although observational studies in psoriasis suggest methotrexate may reduce cardiovascular risk by lowering inflammation, a recent cohort study found no difference in ischemic heart disease rates between methotrexate and other nonbiologic treatments. Regardless, careful monitoring of fluid balance, renal function, and hematologic parameters is necessary during methotrexate therapy in CHF patients [28, 29]. Acitretin, while not directly cardiotoxic, may exacerbate hyperlipidemia, a known contributor to atherosclerotic progression and heart failure. Its use should be limited or avoided in patients with poorly controlled lipid profiles or established coronary artery disease [30]. Cyclosporine, owing to its effects on sodium and water retention, hypertension, and nephrotoxicity, is contraindicated in moderate-to-severe CHF. Use of cyclosporine in this patient population can precipitate or worsen CHF and is generally avoided [28].

Biologic therapies offer substantial efficacy in psoriasis, but their use must be carefully considered in the context of CHF. TNF-α inhibitors, such as infliximab and etanercept, have been associated with worsening heart failure, particularly at higher doses [31]. Randomized clinical trials in heart failure populations demonstrated increased mortality and hospitalization rates with TNF-α blockade in NYHA class III–IV patients [32]. Consequently, TNF inhibitors are contraindicated in patients with advanced CHF. Even in NYHA class I–II CHF, TNF-α inhibitors should only be considered after consultation with a cardiologist and a baseline echocardiography. Treatment should be discontinued if symptoms worsen or ejection fraction drops below 50% [33].

In contrast, IL-17 inhibitors and IL-23 inhibitors have demonstrated no increased risk of new-onset or worsening CHF in clinical trials or post-marketing registries. A pooled analysis of long-term ixekizumab trials involving 3736 patients reported no CHF exacerbations [34]. A meta-analysis also confirmed no significant risk of new-onset CHF with IL-17 or IL-23 inhibitors [35]. Their favorable cardiac safety profile,

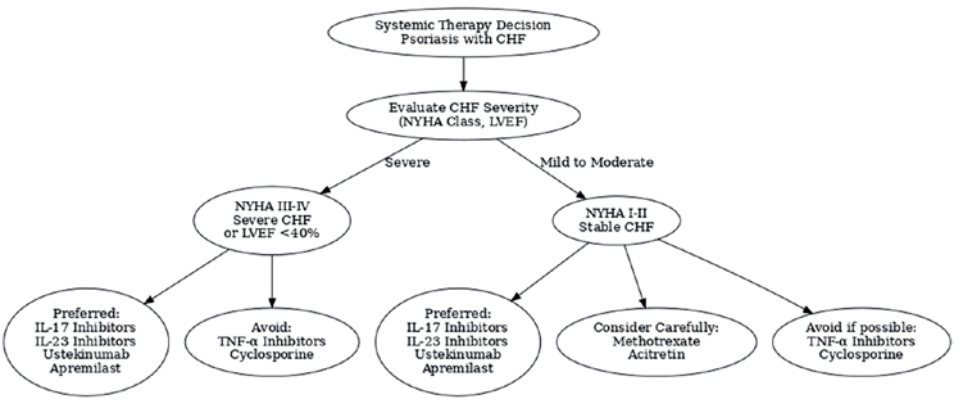

Figure 1.
Clinical treatment algorithm for psoriasis management in patients with congestive heart failure.

rapid onset of action, and strong efficacy in both skin and joint involvement make them well-suited for psoriatic patients with coexisting heart failure requiring systemic treatment. While long-term cardiovascular outcome data are more extensive for IL-17 inhibitors, available evidence for IL-23 inhibitors, including guselkumab and risankizumab, remains reassuring, with no signal of adverse cardiac events reported to date. Ustekinumab, likewise demonstrates a reassuring cardiac safety profile and may be an alternative choice, particularly for patients with concomitant arthritis or inflammatory bowel disease [36]. A summarized treatment algorithm outlining the clinical approach to psoriasis management in patients with CHF is presented in **Figure 1**.

7. Neurologic disorders

The association between psoriasis and neurologic disorders, particularly multiple sclerosis (MS), has garnered increasing attention due to overlapping immunopathogenic mechanisms and the potential impact of systemic therapies on neural tissues. Both psoriasis and MS are characterized by dysregulation of T-cell-mediated immune responses, notably involving the IL-17/Th17 axis [37]. Importantly, several treatments for psoriasis, especially TNF-α inhibitors, have been implicated in either exacerbating or inducing demyelinating disorders, necessitating cautious therapeutic planning in patients with neurologic comorbidities [19].

Prior to initiating systemic therapy in patients with psoriasis and a history of demyelinating disease, a comprehensive neurologic evaluation is essential. Patients with unexplained neurologic symptoms such as visual disturbances, muscle weakness, sensory changes, or gait abnormalities should be thoroughly investigated to rule out latent or subclinical MS. Similarly, a family history of MS or other autoimmune neurologic diseases should prompt heightened vigilance in systemic therapy selection [38].

When systemic therapy is required in psoriatic patients with comorbid neurological disorders, conventional agents such as methotrexate and acitretin are generally considered safe. Neither has been associated with central demyelination, although rare cases of reversible leukoencephalopathy and cognitive effects have been reported with low-dose methotrexate [39]. Cyclosporine is associated with neurotoxic side effects in up to 28% of patients, including tremor, paresthesia, seizures, and cognitive changes [40]. These effects are dose-dependent and more likely in settings of

blood-brain barrier disruption. Given its narrow therapeutic window, nephrotoxicity, and potential for neurotoxicity, cyclosporine should be used cautiously and only when other agents are contraindicated. Apremilast is considered safe in demyelinating diseases, with no known CNS toxicity. It does not cross the blood-brain barrier and is particularly appealing in MS patients who suffer from limited mobility or metabolic comorbidities due to its weight-reducing effect and favorable safety profile [41].

TNF-α inhibitors are contraindicated in MS due to strong mechanistic and clinical evidence linking them to central and peripheral demyelination [42]. All five TNF inhibitors, including the newer agents certolizumab and golimumab, have been implicated in new-onset or worsening MS, with real-world reports showing most events occur within the first year of therapy [43]. Among 84 reported cases of psoriasis, only 33% achieved full recovery after stopping treatment, while several progressed or showed new silent lesions. Consequently, TNF-α inhibitors should be strictly avoided in this population [44].

IL-17 inhibitors, particularly secukinumab, are currently regarded as the most appropriate biologic class for patients with psoriasis and coexisting MS, given that IL-17A and IL-17F levels have been implicated in the pathogenesis of both conditions. Clinical trial data involving patients with relapsing-remitting MS revealed a 49% reduction in new MRI lesions and a 67% reduction in active lesions with secukinumab treatment [45]. Real-world evidence further supports its safety: in a small series, 80% of patients with both MS and psoriasis who were treated with secukinumab remained clinically stable, with no progression of neurologic disease and no reported cases of de novo central demyelination [44]. For other IL-17 inhibitors, such as ixekizumab and bimekizumab, no robust data are currently available. To date, only a single case report suggests the safe use of ixekizumab in a patient with pre-existing multiple sclerosis, and there are no published neurologic safety data for bimekizumab [46].

Ustekinumab, targeting both IL-12 and IL-23, is another biologic agent with a reassuring neurologic safety profile [47]. IL-23 inhibitors, including guselkumab, risankizumab, and tildrakizumab, are also regarded as safe choices in this setting. Although long-term neurologic outcome data are still emerging, there is currently no evidence suggesting that IL-23 blockade promotes demyelination [48, 49].

8. Latent tuberculosis and HIV

Management of psoriasis in patients with latent tuberculosis (TB) requires careful screening and strategic treatment selection, especially prior to the initiation of systemic immunosuppressive agents. Interferon-gamma release assays (IGRAs) such as QuantiFERON-TB Gold are preferred over tuberculin skin tests (TST) in BCG-vaccinated individuals. A chest X-ray is essential to rule out active TB. If latent TB is confirmed, chemoprophylaxis, typically with isoniazid for 6–9 months, should be started. Most guidelines allow systemic therapy to begin after 1 month of prophylaxis, though this may vary by region. Close clinical and radiological monitoring throughout treatment is strongly advised [50].

Among systemic agents, TNF-α inhibitors pose the highest risk of TB reactivation. TNF is essential for granuloma integrity and host control of *Mycobacterium tuberculosis*. Conventional systemic therapies, including methotrexate and cyclosporine, have lower TB reactivation potential, but screening and vigilance are still required. TB reactivation with these agents is rare but reported, particularly in long-term use or when combined with other immunosuppressants [51].

Apremilast and acitretin, given their non-immunosuppressive mechanism of action, have not been associated with TB reactivation. They do not require prophylaxis and are safe options for psoriasis patients with LTBI, particularly those ineligible for biologic therapies [17, 21].

Ustekinumab has shown a low risk of TB reactivation in clinical trials (0/3177 cases with prophylaxis); however, recent pharmacovigilance data from the FAERS database reported 121 cases of latent TB, indicating a potential real-world signal and reinforcing the need for strict pre-treatment screening and ongoing monitoring [52, 53].

IL-17 and IL-23 inhibitors have shown an excellent safety profile regarding tuberculosis reactivation, with no reported cases in large clinical trials involving 3430 patients treated with secukinumab, 5898 with ixekizumab, and no reactivations observed in pivotal trials of guselkumab, risankizumab, and tildrakizumab [14]. This low risk has been corroborated by the 2024 multicenter study by Torres et al., which evaluated 405 psoriasis patients with newly diagnosed LTBI who were treated with IL-17 or IL-23 inhibitors. Remarkably, only one TB reactivation (0.25%) occurred over a mean follow-up of 33 months, and 37.8% of patients did not receive full chemoprophylaxis [54].

Psoriasis in the setting of HIV infection often exhibits an atypical clinical course, characterized by increased severity, resistance to conventional treatments, and the potential for uncommon morphologic variants, including erythrodermic, rupioid, and palmoplantar forms. In some instances, the onset of severe psoriasis may herald previously undiagnosed HIV infection. Thus, HIV testing should be considered in patients with abrupt or unusually severe psoriasis presentations [55].

Management of psoriasis in HIV-positive patients demands a delicate balance between achieving disease control and minimizing further immunosuppression. Topical therapies and NB-UVB phototherapy remain first-line options, offering effective management without systemic immunologic compromise [56].

Systemic therapies must be selected cautiously. Methotrexate and cyclosporine, while effective for psoriasis, pose significant risks in HIV-positive patients due to their profound immunosuppressive effects. Their use should be reserved for cases where safer alternatives are unavailable, and only in patients with well-controlled HIV infection (CD4 counts >200 cells/μL and suppressed viral load under antiretroviral therapy). Even then, close monitoring for opportunistic infections is imperative [56].

Acitretin is considered a favorable systemic option for HIV-positive psoriasis patients. It is non-immunosuppressive, does not increase infection risk, and is particularly beneficial in hyperkeratotic variants of psoriasis, which are common in the context of HIV. However, its mucocutaneous side effects and impact on lipid metabolism must be weighed carefully [57].

Apremilast represents an excellent systemic treatment choice in HIV-positive individuals with psoriasis. It is not immunosuppressive, does not interact significantly with antiretroviral therapy, and has a favorable safety profile. Gastrointestinal side effects and mild weight loss are common but generally manageable [58].

Biologic agent therapy in HIV-positive patients is an evolving area. Emerging evidence suggests that TNF-α inhibitors, IL-17 inhibitors, and IL-23 inhibitors can be used cautiously in selected HIV-positive patients with well-controlled disease, particularly when other therapies fail. However, these cases should be managed collaboratively with infectious disease specialists. IL-17 and IL-23 inhibitors may be preferred over TNF-α inhibitors due to their more targeted immunomodulatory profiles and, theoretically, a lower impact on global immune surveillance [44].

In conclusion, psoriasis management in patients with latent tuberculosis or HIV infection necessitates rigorous screening, interdisciplinary coordination, and careful therapeutic selection. Topical therapies and NB-UVB phototherapy should be maximized for mild-to-moderate disease. For systemic therapy, IL-17 and IL-23 inhibitors, ustekinumab, and apremilast offer safe and effective options, while TNF-α inhibitors and conventional immunosuppressants must be used with caution or avoided. The primary goal is to achieve optimal disease control while minimizing infection risk and preserving immune function.

9. Inflammatory bowel disease

Psoriasis and inflammatory bowel disease (IBD) frequently coexist due to shared immunopathogenic mechanisms, especially involving the IL-23/Th17 axis. While some therapies are effective for both conditions, others—particularly IL-17 inhibitors—may increase the risk of IBD exacerbation or development [59]. Therefore, therapeutic selection in patients with concurrent psoriasis and inflammatory bowel disease requires careful consideration to balance dermatological effectiveness with gastrointestinal safety concerns optimally. Agents that are effective in both diseases, such as TNF-α inhibitors and IL-23 pathway inhibitors, are generally preferred, whereas therapies with the potential to worsen IBD must be avoided. A practical summary of treatment preferences based on IBD activity is presented in **Table 2**.

10. Malignancy

The presence of a current or previous malignancy, particularly lymphomas and skin cancers, significantly influences therapeutic decision-making in psoriasis management. In patients with a history of malignancy, topical therapies represent the safest initial treatment strategy. Emollients, corticosteroids, vitamin D analogs, and topical calcineurin inhibitors can be utilized without systemic immunosuppression. NB-UVB phototherapy is also considered safe in patients with a history of solid tumors or treated lymphoma, provided that prior skin cancer history is carefully considered [60]. In contrast, PUVA therapy should be avoided in individuals with a history of melanoma, multiple non-melanoma skin cancers (NMSCs), or significant actinic damage due to its mutagenic potential [61].

Among conventional systemic therapies, methotrexate warrants caution in the setting of malignancy. Long-term methotrexate use has been associated with an increased risk of lymphoproliferative disorders, particularly in patients positive for Epstein-Barr virus (EBV). Methotrexate-induced lymphomas may sometimes regress upon drug discontinuation, but reinitiation carries potential risk. Therefore, methotrexate is generally avoided in patients with active or recent lymphoma [62].

As a potent immunosuppressant, cyclosporine carries a well-established risk of NMSC and lymphomas, especially with prolonged use and in patients with prior ultraviolet exposure. Consequently, its use should be avoided in psoriasis patients with a history of cancer, except in short-term emergency situations under close oncologic monitoring [63]. Acitretin, in contrast, represents a favorable systemic option for psoriasis patients with prior malignancy as it exhibits chemopreventive effects against NMSC. Acitretin is particularly beneficial for psoriatic patients with a history of actinic keratoses, squamous cell carcinoma, or field cancerization, although its

IBD status	Preferred treatments	Treatments to consider carefully	Avoid	Supportive therapies
Active IBD [14]	• **TNF-α inhibitors** (adalimumab, infliximab) *(FDA-approved for Crohn and UC)*	• **Methotrexate** *(limited efficacy for IBD; mainly useful for joint symptoms)*	• **IL-17 inhibitors**	• Topical therapy
	• **Ustekinumab** *(FDA-approved for Crohn's disease)*			• NB-UVB
	• **Risankizumab** *(FDA-approved for Crohn's disease)*			
Controlled IBD [14]	• **Ustekinumab** *(FDA-approved for Crohn's disease)*	• **Tildrakizumab** *(psoriasis approved; limited data in IBD)*	• **IL-17 inhibitors**	• Topical therapy
	• **Risankizumab** *(FDA-approved for Crohn's disease)*	• **Tildrakizumab** *(psoriasis approved; limited data in IBD)*		• NB-UVB
	• **TNF-α inhibitors** (adalimumab, infliximab) *(FDA-approved for Crohn and UC)*	• **Apremilast** *(safe but no effect on IBD activity)*		
		• **Methotrexate** *(limited benefit)*		

Table 2.
Systemic treatment options for psoriasis with coexisting inflammatory bowel disease.

mucocutaneous side effects and teratogenicity must be managed appropriately [64]. Apremilast provides an additional safe systemic alternative. Its immunomodulatory effects are mild, and it has not been linked to an increased risk of malignancy in either clinical trials or real-world studies. It is especially advantageous for patients with a recent history of solid tumors or for those in whom conventional immunosuppressants are contraindicated [21].

TNF-α inhibitors have historically raised concern regarding malignancy risk, particularly for lymphoproliferative disorders [65]. While older meta-analyses yielded mixed results, recent large-scale data continue to support a cautious approach. A retrospective cohort study published in 2025 involving 7645 Korean patients with psoriasis and rheumatoid arthritis found no significant increase in the risk of common solid tumors—including colorectal, liver, lung, kidney, breast, and thyroid cancers—among TNF-α inhibitor users. However, the risk of lymphoma was significantly elevated in infliximab users, and leukemia risk increased in users of etanercept and adalimumab [66]. These findings reinforce current guideline recommendations to defer TNF-α inhibitor use for at least 5 years after remission in patients with a history of lymphoma or leukemia and to prefer other biologic classes when biological agent treatment is indicated [67].

IL-17 inhibitors, particularly secukinumab and ixekizumab, have demonstrated favorable safety profiles in patients with prior malignancy [68, 69]. Real-world case series and retrospective analyses of patients with various cancers treated with IL-17A inhibitors have shown no recurrence or progression during long-term follow-up periods [70]. This supports the cautious use of IL-17 blockers in psoriatic patients with a stable cancer history. Similarly, IL-23 inhibitors such as guselkumab and risankizumab have not been associated with malignancy recurrence in clinical trials or post-marketing data. These agents are increasingly considered safe and effective options in this clinical context [71]. Ustekinumab similarly exhibits a reassuring malignancy profile, with long-term follow-up data demonstrating no elevated cancer risk [14].

In conclusion, managing psoriasis in patients with a history of malignancy necessitates an individualized approach emphasizing oncologic safety. Topical therapies and NB-UVB phototherapy should be prioritized for mild disease. Among systemic options, acitretin, apremilast, and biologic agents targeting IL-17 or IL-23 pathways are preferred choices, offering effective disease control without compromising cancer surveillance. Multidisciplinary collaboration with oncology is essential to ensure that therapeutic strategies align with the patient's oncologic status and long-term health goals.

11. Psoriatic arthritis

Approximately 20–30% of patients with psoriasis will develop PsA during their lifetime, typically manifesting between the ages of 30 and 50 years. The pathogenesis of PsA shares common immunological pathways with cutaneous psoriasis, particularly involving the IL-23/Th17 axis, yet the clinical expression in musculoskeletal tissues necessitates specialized therapeutic strategies [11].

Early identification of PsA is critical to prevent irreversible joint damage and long-term disability. Screening questionnaires such as the Psoriasis Epidemiology Screening Tool (PEST) or the Toronto Psoriatic Arthritis Screen (ToPAS) can aid dermatologists in early detection. Diagnosis should be confirmed by rheumatologists

based on the Classification Criteria for Psoriatic Arthritis (CASPAR) criteria, and treatment should be initiated promptly to prevent structural joint damage [72].

In cases of mild psoriasis and limited joint involvement, topical therapies and NB-UVB phototherapy may provide relief for cutaneous lesions, but they have no effect on joint inflammation. Therefore, systemic therapy is mandatory in PsA, regardless of the severity of cutaneous involvement, when arthritis is clinically evident [73]. A summarized treatment algorithm outlining the clinical approach to PsA management is presented in **Figure 2**.

In patients experiencing mild peripheral arthritis, NSAIDs and intra-articular corticosteroids can help alleviate symptoms. Methotrexate is often the first-line conventional synthetic DMARD (csDMARD), especially for peripheral arthritis. Nonetheless, its effectiveness in axial PsA, enthesitis, and dactylitis remains limited. Cyclosporine may provide quick symptom relief during severe PsA flare-ups but carries risks such as nephrotoxicity, hypertension, and metabolic issues. Due to these potential risks and the availability of biologic therapies, cyclosporine is now typically used only in rare, short-term indications. Apremilast presents a safe alternative in patients with mild disease or in those ineligible for biologic therapy, though its efficacy is modest compared to targeted biologics [19].

When csDMARDs are insufficient or when patients present with moderate-to-severe PsA, biologic DMARDs (bDMARDs) are the mainstay of treatment. TNF-α inhibitors, including adalimumab, etanercept, infliximab, certolizumab pegol, and golimumab, are the longest-established class of biologic agents used in the treatment of PsA. TNF-α inhibitors have demonstrated robust efficacy across all major clinical domains of PsA—including peripheral arthritis, axial involvement, enthesitis, and dactylitis. They are particularly effective in polyarticular peripheral arthritis, with pivotal trials such as RAPID-PsA, ADEPT, and GO-REVEAL showing significant improvements in both clinical remission and prevention of structural joint damage. In axial PsA, TNF-α inhibitors remain among the only biologics with proven efficacy,

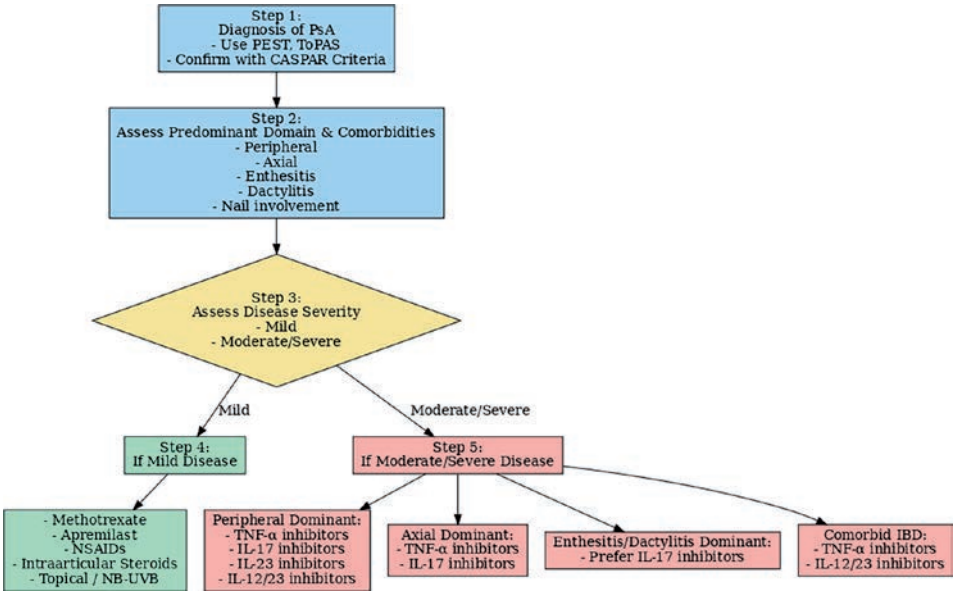

Figure 2.
Clinical treatment algorithm for the management of psoriatic arthritis.

reducing spinal inflammation and improving mobility, as validated by MRI and patient-reported outcomes [74].

IL-17 inhibitors, including secukinumab and ixekizumab, selectively block the IL-17A pathway and have demonstrated strong efficacy across multiple domains of PsA. These agents produce rapid and sustained clinical improvements in peripheral arthritis, enthesitis, dactylitis, and notably, axial disease [75]. In patients with axial PsA, secukinumab and ixekizumab significantly reduced spinal pain, morning stiffness, and MRI-confirmed inflammation. The phase 3 MAXIMISE trial specifically confirmed the efficacy of secukinumab in active sacroiliitis, reinforcing its value for axial involvement. Both agents have also been shown to achieve significant ACR20 response rates and inhibit radiographic progression over time [76].

Bimekizumab, a newer agent that targets both IL-17A and IL-17F, has further expanded the therapeutic landscape. In the BE OPTIMAL and BE COMPLETE phase 3 trials, bimekizumab demonstrated significantly greater efficacy than placebo in improving joint and cutaneous symptoms, achieving ACR50 rates of 44–46% and PASI100 responses in over 60% of patients by week 16. Importantly, bimekizumab showed superior efficacy in traditionally challenging domains such as enthesitis and dactylitis, with early onset of action. These data position IL-17 inhibitors as a potent option for comprehensive PsA management [77].

IL-23 inhibitors, such as guselkumab and risankizumab, have shown substantial efficacy in peripheral arthritis and skin disease, with emerging evidence supporting their potential benefit in enthesitis and dactylitis [78]. They demonstrated significant efficacy in joint involvement, with ACR20 response rates ranging from 57% to 64% in pivotal trials. Guselkumab, particularly at a dosing interval of every 4 weeks, was associated with superior inhibition of structural damage progression as measured by the van der Heijde-Sharp score compared to every 8-week dosing, placebo, and risankizumab. While both IL-23 inhibitors improved enthesitis and dactylitis outcomes, guselkumab demonstrated more consistent efficacy across multiple domains and trials, suggesting a potentially broader therapeutic impact in PsA disease phenotypes. However, their effectiveness in axial PsA is still under investigation, and current data suggest a less prominent role compared to TNF-α and IL-17 inhibitors in pure axial disease [79, 80].

Ustekinumab is effective for peripheral PsA, though its efficacy in axial disease is relatively lower. It remains a valuable option, particularly in patients with concomitant inflammatory bowel disease or contraindications to other biologic classes [73].

In conclusion, biologic therapies targeting TNF-α, IL-17, and IL-23 pathways have expanded the therapeutic arsenal and significantly improved patient outcomes. Optimal management demands careful consideration of the predominant disease domains, comorbidities, and long-term safety profiles of available agents, guided by current evidence-based recommendations.

12. Conclusion

Effective psoriasis management in special clinical contexts requires personalized, evidence-based strategies. Pregnancy and breastfeeding call for safer choices such as topical therapies, NB-UVB phototherapy, cyclosporine, and certolizumab pegol, while methotrexate and acitretin are contraindicated. Patients with hepatic or renal impairment benefit from IL-17 and IL-23 inhibitors due to favorable safety profiles. For those with congestive heart failure or demyelinating disease, IL-17 and IL-23 inhibitors are

preferred over TNF-α inhibitors. Managing psoriasis in latent tuberculosis or HIV-positive patients necessitates rigorous screening and cautious therapy selection, favoring IL-17/IL-23 inhibitors, acitretin, and apremilast. In concurrent IBD, TNF-α and IL-23 inhibitors are optimal, whereas IL-17 inhibitors should be avoided. In patients with a history of malignancy, topical therapies, phototherapy, acitretin, apremilast, and IL-17 or IL-23 biologics are preferable due to their favorable safety profiles. Psoriatic arthritis treatment is effectively managed with biologics that target the TNF-α, IL-17, and IL-23 pathways. In conclusion, individualized and interdisciplinary approaches ensure optimal outcomes across these complex clinical scenarios.

Conflict of interest

The authors declare no conflict of interest.

Appendixes and nomenclature

PsA	psoriatic arthritis
NB-UVB	narrowband ultraviolet B phototherapy
PUVA	psoralen plus ultraviolet A phototherapy
TNF-α	tumor necrosis factor alpha
IL-17	interleukin-17
IL-23	interleukin-23
IL-12	interleukin-12
NMSC	non-melanoma skin cancer
CASPAR	classification criteria for psoriatic arthritis
PEST	psoriasis epidemiology screening tool
ToPAS	Toronto Psoriatic Arthritis Screen
IGRA	interferon-gamma release assay
TST	tuberculin skin test

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