Research Review
EDUCATIONAL SERIES

Diagnosis and treatment of psoriasis in New Zealand

About the Reviewer

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Dr Louise Reiche spent her latter postgraduate advanced training years in the UK, working in High Wycombe, Amersham and Oxford Radcliffe Infirmary, and participated in senior registrar training at St John's London as well as Oxford. After several years of research with L'Oreal she wrote her MD thesis (Self-perceived sensitive skin). Louise has worked as a general physician and Dermatologist at Palmerston North Hospital and now as whole time physician general dermatologist. Louise has served on the executive of the NZ Dermatological Society and is their current Pharmac liaison dermatologist. She has also served on the Vitamin D Working Group for the Cancer Society.

About this Review

This review is intended as an educational resource for health professionals. It discusses how to confidently diagnose psoriasis, the incidence of the condition and the options available for treatment, with a focus on topical therapy in primary care. Peer-reviewed clinical trial evidence on current treatment options is presented with accompanying expert commentary that is intended to inform readers about current research in this area.

Psoriasis

Psoriasis is a common, chronic, relapsing, noninfectious, multisystem, immune-mediated disease, which manifests as several different clinical variants including plaque psoriasis (the most common type), flexural psoriasis, guttate psoriasis, pustular psoriasis, nail psoriasis, erythrodermic psoriasis and psoriatic arthritis (affecting 10–30% of patients with plaque psoriasis).[1-4] It is estimated that approximately 1 in 50 people have psoriasis, which may be mild and barely noticeable, or so severe that it requires intensive therapy and sometimes hospitalisation.[5] Approximately 80% of those affected by psoriasis have mild-to-moderate disease.[1] Psoriatic lesions, which vary in size and degree of inflammation, may be itchy, but seldom painful, and the disease tends to run a chronic course with exacerbations and remissions.[5,6]

Psoriasis affects both genders equally and may occur at any age, with two peak age ranges (16–22 years and 57–60 years).[5,6] In more than 33% of patients, the initial presentation of psoriasis occurs before the age of 20 years.[7,8] Females and those with a family history of psoriasis often develop the disease at an earlier age, and early onset tends towards more severe disease.[2,6] Those with severe disease have a higher risk of mortality than the general population.[9] Moreover, about 60% of patients report that their disease is a significant problem in their everyday life (women and children tend to experience a greater negative impact).[10,11]

Over time, the understanding of the pathogenesis of psoriasis has developed from one of a hyperkeratotic disorder of keratinocytes to a dysregulation of the immune system mediated by cytokines.[9] It is now understood that T-helper (Th)-1, Th-17 and Th-22 cell populations are expanded and stimulated to release inflammatory cytokines, including interleukin (*IL)-17*, *IL*-22 and tumour necrosis factor-alpha (TNF-α).[9]

The aetiology of psoriasis

While the aetiology of psoriasis is not fully understood, studies support the hypothesis that psoriasis develops as a result of the interaction of an individual's genetic susceptibility, specific environmental factors and the immune system.[9,12] Approximately 70% of individuals with paediatric psoriasis have a positive family history for the condition.[5,13] Psoriasis develops in 8% of siblings of persons with the disease when neither parent is affected, in 16% when one parent is affected and in 50% of siblings when both parents have the disease.[14]

A number of psoriasis-susceptibility gene loci (e.g. *PSOR1*) and genes involved in *IL*-23 signalling (*IL23A, IL23R, IL12B*), modulation of Th2 immune responses (*IL4, IL13*) and activated B cell (NF-κB) signalling (*TNIP1, TNFAIP3*) have been identified.[3,9]

There are numerous identified triggers and aggravating factors for psoriasis including: infections, particularly bacterial (e.g. Streptococci) for guttate psoriasis, human immunodeficiency virus (HIV), stress, drugs (e.g. lithium, antimalarials, beta-blockers), smoking, high alcohol intake, obesity and a high glycaemic diet.[5,12,15] Severe rebound psoriasis has been recorded after discontinuing systemic or potent topical corticosteroids.[5]

Comorbidities

Psoriasis is now recognised as a systemic disease mediated via T-cells and the inflammatory processes involved are associated with the development of a number of comorbidities.[9] These comorbidities impact significantly on quality of life and include psoriatic arthritis, hypertension, cardiovascular disease, stroke, obesity, diabetes mellitus, hyperlipidaemia and psychological disorders (including depression).[5,9,16] Comorbidities were present in 551 out of 753 (73%) psoriasis patients in a US university dermatology practice, and in a large US study involving over 100,000 individuals with psoriasis, the incidence rates of hyperlipidaemia, hypertension, depression, diabetes and cardiovascular disease were 27.3%, 25.4%, 9.2%, 8.7% and 8.6%, respectively.[16,17] Such comorbidities have been estimated to contribute to a 3- to 4-year reduction in life expectancy in individuals with severe psoriasis.[9] Furthermore, obesity

acts as a parameter for pharmacological interactions and may serve to increase the inflammatory load.[9]

In addition, severe psoriasis appears to be an independent risk factor for myocardial infarction and patients with psoriasis are more likely to smoke and drink alcohol excessively.[5]

It is also recognised that individuals with psoriasis exhibit an increased risk for other immune-mediated disorders with overlapping pathology, including rheumatoid arthritis (RA) and inflammatory bowel disease (IBD).[3,9] Health promotion and assessment for these risk factors is therefore a very important aspect of psoriasis management.[5]

The prevalence of psoriasis

Psoriasis is estimated to affect 125 million people worldwide.[18] Approximately 2% of the Western population is affected, with the disease exhibiting varying prevalence among different ethnic groups.[5,9,19] Samoans, Australian Aborigines and South American Indians do not appear to be affected by the condition.[20,21] Prevalence estimates from Asia indicate that psoriasis is less prevalent in Japan (0.05%) and China (1.23%) than in the US (3.15%).[22]

The burden of psoriasis

The extent of skin involvement in psoriasis does not always correlate with the levels of symptoms experienced by the individual. Surveys have demonstrated that even patients with relatively limited affected body surface area (BSA) can experience significant impact on quality of life and that the detrimental effects of the condition can go far beyond the skin.[5,23] It is the psychosocial impact of the disease that leads to a more significant reduction in quality of life for many sufferers than itching or pain.[5] A systematic literature review of 17 studies investigating quality of life in patients with psoriasis, found that the illness is associated with: impaired emotional functioning, physical discomfort, a negative body- and self-image, and limitations in daily activities, work, social contacts and (skin-exposing) activities.[24] Psoriasis sufferers bear the humiliation of constantly shedding scales and often bear the brunt of public rejection due to misunderstanding surrounding the disease.

Psoriatics may isolate themselves; a fact evident in a recent survey undertaken in NZ involving 308 individuals with the condition, revealing that 77% of sufferers hide their psoriasis from other people.[8] Almost one-third reported that their psoriasis had prevented them from taking a job where they were unable to cover their body. A recent Canadian study found higher rates of unemployment and reduced income in psoriasis patients and the levels of these parameters increased with increasing disease severity.[25] Children and adolescents in particular with chronic, visible psoriasis may require substantial family and professional support to cope with the social and psychological sequelae of psoriasis and are at increased risk of psychiatric disorders.[26,27]

A US study comparing health-related quality of life (HRQL) in individuals with psoriasis and those with other major chronic health conditions, revealed that individuals with psoriasis reported a reduction in mental and physical functioning comparable to that seen in arthritis, cancer, heart disease, hypertension, diabetes and depression.[23]

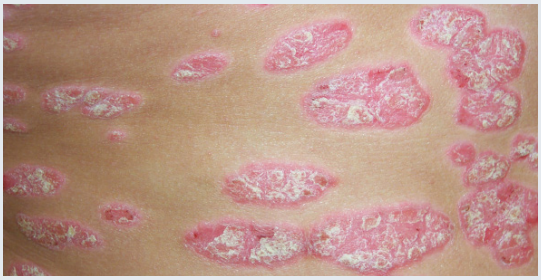
A survey undertaken by the US National Psoriasis Foundation involving 405 patients with psoriasis (66% had moderate-to-severe disease), revealed that 29% spent 30 minutes per day caring for their disease and 24% spent ≥1 hour doing so.[28] In a European survey of 17,000 patients with psoriasis, half reported that the time-consuming nature of therapy for their disease was the most troublesome part of their treatment.[29]

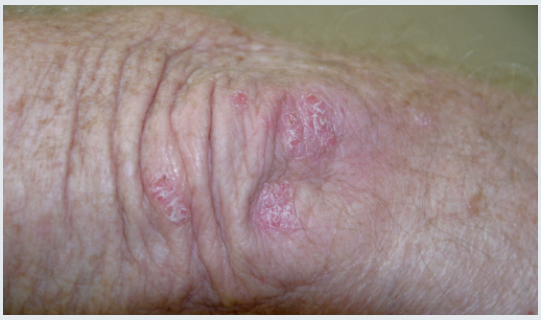
The economic burden of psoriasis is large for both the individual and the community. A NZ survey revealed that for those who reported lost wages due to their psoriasis, the average income lost during the previous year was NZ$1,753.[8] Almost one-third of those in the NZ survey revealed that they had cut back on other spending in order to afford medical treatment for their condition. A US study reported that about 60% of psoriatics missed on average 26 days of work each year due to their illness, while another study reported that work loss costs attribute to 40% of the total cost burden.[30,31]

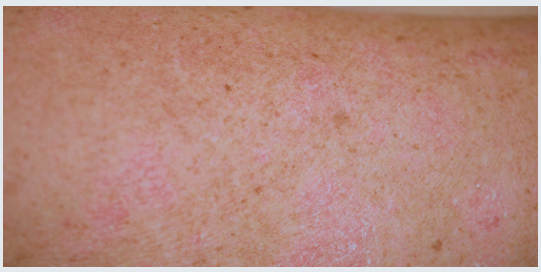
Diagnosis: recognising the psoriatic variants

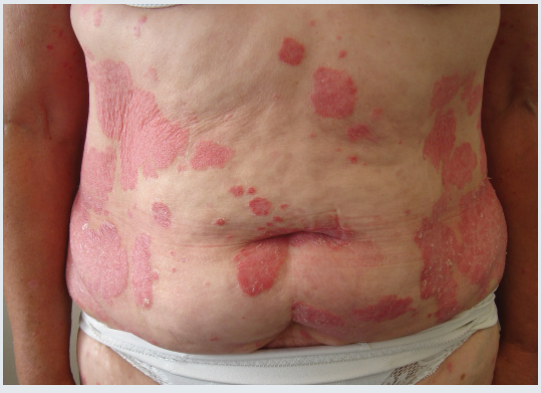
Plaque psoriasis

- Psoriasis is usually a clinical diagnosis. Psoriasis typically presents as small to large, clearly demarcated, erythematous plaques covered by silvery white scales, predominantly on the elbows, knees, scalp, lumbar and umbilical regions, and often affects the body symmetrically.[4,5,32]
- Scales may be less obvious if the patient is using emollients.[32] In highly pigmented skin, scales may appear grey. Hyperpigmentation may develop post-inflammation.[5,32]
- Physical trauma may trigger plaque psoriasis at the site of injury, be it a scratch, surgical wound or infection: this type is referred to as koebnerised psoriasis.[4,5]



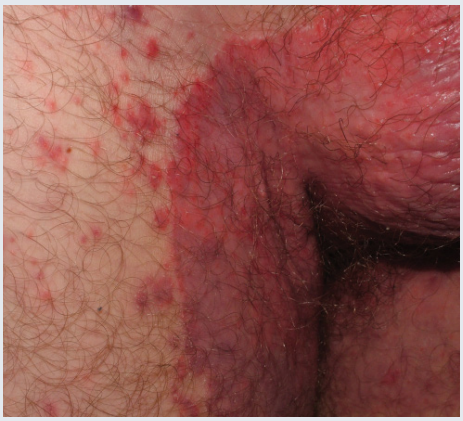
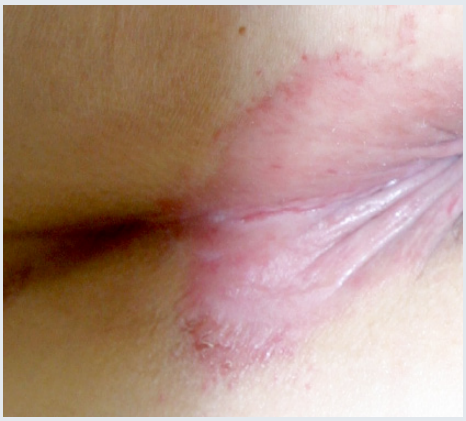
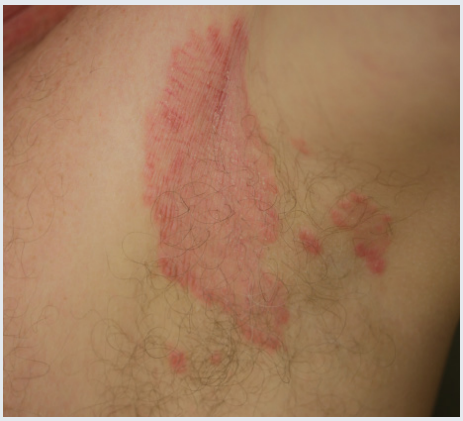






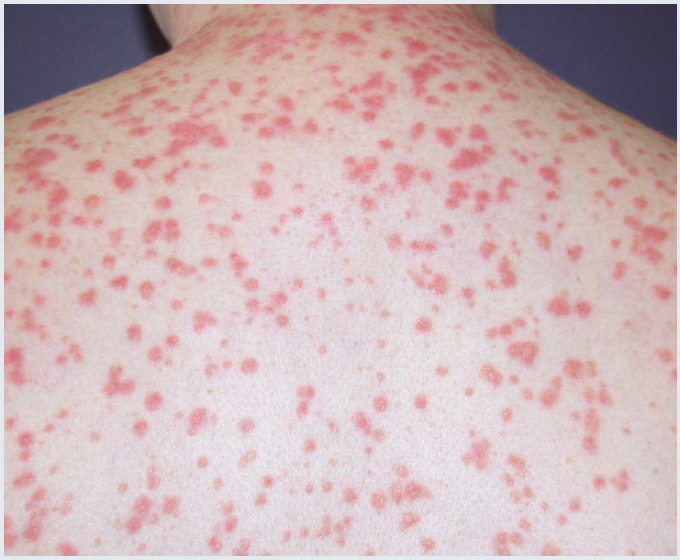
Flexural psoriasis

- Flexural psoriasis occurs in the folds of the skin such as the axillae, umbilicus, inframammary, perianal, groin, genital and natal cleft regions.[4,5,32]
- This type of psoriasis usually lacks scales (although they may be seen in lesions around the edge). Skin fissures may be present.
- Some patients only have flexural involvement and this type is sometimes mistaken for eczema, especially seborrheic eczema (see Page 4 for tips on differentiating psoriasis from eczema).[5]

Guttate psoriasis

- This type of psoriasis is often preceded (2-3 weeks) by a Streptococcal infection.[4]
- It presents as small, disseminated erythematous papules and plaques.[4,32]
- Guttate psoriasis is usually concentrated over the trunk and proximal limbs, but may coalesce in some regions.[5,32]
- If the face and scalp are affected, lesions are usually mild.[32]
- This type of psoriasis sometimes spontaneously clears completely.

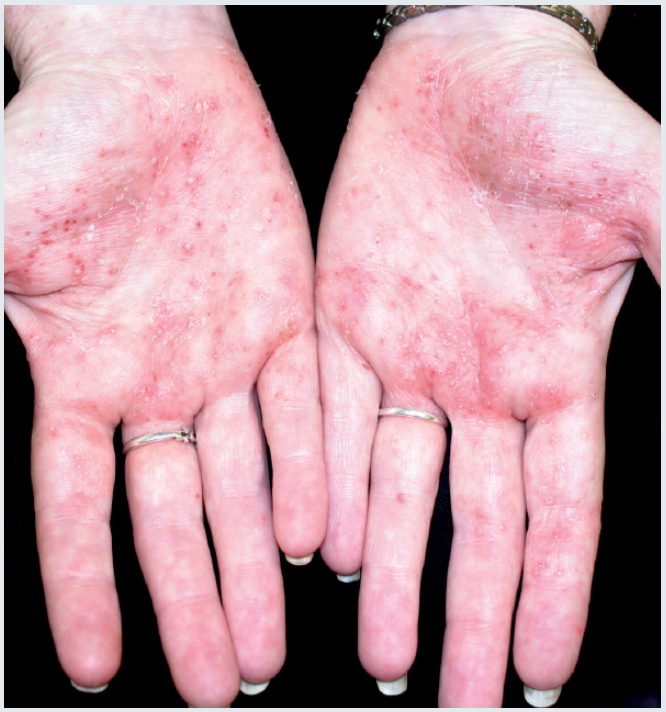


Erythrodermic psoriasis

- Erythrodermic (>90% BSA) psoriasis usually arises due to worsening of unstable psoriasis (any type), but occasionally is the first presentation of psoriasis.[32]
- This condition is considered a dermatological emergency as it is potentially fatal.[4,32]
- Triggers include sudden withdrawal of oral corticosteroids or potent topical corticosteroids.

Palmoplantar psoriasis

- Palmoplantar psoriasis (palmoplantar pustulosis) affects the palms of the hands and the soles of the feet, but is composed of sterile pustules.
- There is some debate as to whether palmoplantar pustulosis is a form of psoriasis or a separate disease in its own right.



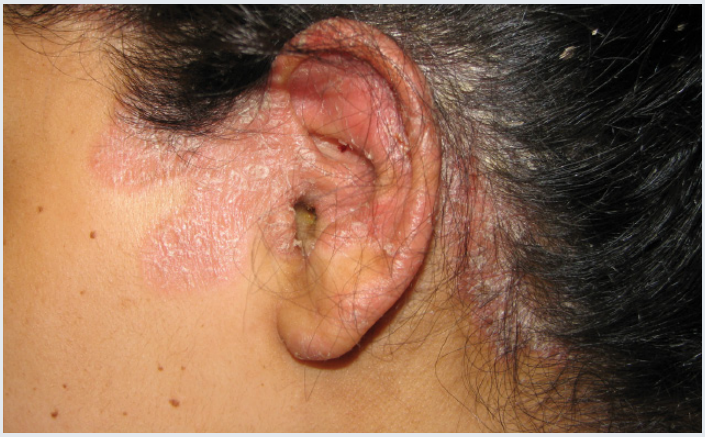
About Research Review

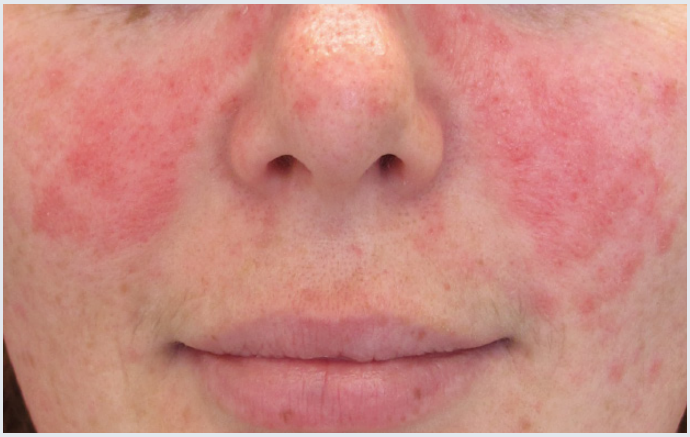
Research Review is an independent medical publishing organisation producing electronic publications in a wide variety of specialist areas.

Research Review publications are intended for New Zealand medical professionals.

Scalp and facial psoriasis

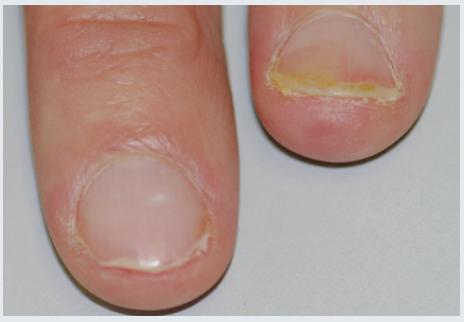
- Any region of the scalp may have psoriasis.
- It may occur in isolation or along with one of the other forms of psoriasis and the lesions may extend a short distance beyond the hairline.
- It is found in 50% of those with plaque psoriasis.[4,32]
- Facial psoriasis ranges from mild to severe and appears as thickened, red and dry patches.[32]
- It may overlap with seborrheic eczema, sometimes known as sebopsoriasis.
- Most patients with facial psoriasis will also have scalp psoriasis.[32]

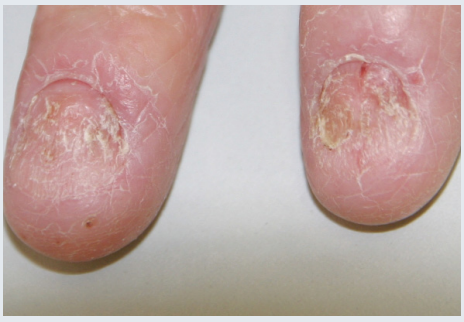




Nail psoriasis

- Nail involvement is frequent in psoriasis, but fingernails are more commonly involved than toenails.[33]
- Most commonly, small pits are seen in the nail plate and the nail may detach from the bed at its lateral or distal attachments (onycholysis).[33]
- Other symptoms include discolouration and thickening of the nail (subungual hyperkeratosis), and orange-yellow deposits underneath the nail plate (oil spots), and rarely, nail plate crumbling.[4,5,32,33]
- Onycholysis, yellow discoloration and hyperkeratosis are frequently misdiagnosed as fungal infection.
- Submit nail and skin scrapings for mycology before treating with antifungal agents.





Examples of other rare types of psoriasis can be found on the DermNet NZ website: http://dermnetnz.org/.

Psoriasis in children

The same clinical variants of psoriasis seen in adults occur in children, though the lesions may be paler, thinner and less scaly.[34] In those under 2-years of age, the first presentation of psoriasis may be in the nappy region, which appears as a bright red, well demarcated, glazed rash.[7,35] Guttate psoriasis is more frequent in children than in adults and emotional stress is more of a trigger of psoriasis in the paediatric population than in the adult population. The paediatric presentation of psoriasis may be atypical, thus making diagnosis difficult.[7] The Auspitz's sign (pinpoint bleeding upon removal of scales) and nail changes are useful features to help with diagnosis.[7]

Differentiating psoriasis from eczema

- Eczema is almost always intensely itchy, whereas most psoriasis suffers do not experience itch[5]
- Psoriatic plaques are clearly defined – eczema is usually not[5]
- Extensor involvement is more typical of psoriasis[5]
- In psoriasis the umbilicus may be involved[5]
- Discoid eczema can mimic psoriasis, but does not have silvery scales[5]
- In flexural areas, scaly plaques around the periphery may be present in psoriasis.[5] The lesions are typically clearly demarcated, in contrast to eczema or thrush or tinea, which are typically less defined and rather faded at the edges
- Look for the Auspitz's sign - pinpoint bleeding upon removal of psoriatic scales[7]
- Look for nail involvement – this is uncommon in eczema unless there is eczema of the distal digits and nail folds

Assessing the severity of psoriasis

The severity of psoriasis is assessed by the degree and extent of the body surface area (BSA) affected as well as its impact on the individual's quality of life.[36] Psoriasis affecting less than 5% of the BSA is considered mild, that affecting 5-10% of the BSA is considered moderate and that affecting >10% of the BSA may be considered severe.[37] As a reference, the palm of the hand is equal to approximately 1% of the skin surface.[36] It must be kept in mind that a patient with facial psoriasis, but a low BSA involvement may be considered as having more severe disease than a patient without facial psoriasis with a larger BSA involvement.[38] It is therefore paramount to establish how much his or her psoriasis affects an individual patient. A patient with minimal disease for whom it has little impact may not need or want any therapy.

The Psoriasis Area and Severity Index (PASI) is considered the current gold standard for the assessment of extensive psoriasis and is used in clinical trials.[39] The PASI takes into account the degree (nil – very severe, scored 0–4) of redness, thickness, scaliness multiplied by the surface area involvement for each of four body areas (head and neck, upper limbs, lower limbs, trunk) which have been mathematically proportioned and then totalled. The PASI takes a few minutes and requires some experience to calculate it accurately. DermNet NZ is an excellent resource with instructions on undertaking the PASI http://www.dermnetnz.org/scaly/pasi.html. Links to a downloadable PASI form and calculator are available from: http://pasi.corti.li/

In NZ, Pharmac requires that this measurement be submitted when applying for special authority for specific medications for a patient, but the PASI is not normally calculated in primary care. Quality of life assessment tools better capture how their disease affects patients than the more objective PASI score. Several such tools, including the Dermatology Life Quality Index (DLQI), which is a simple and reliable assessment method that is sensitive to changes in quality of life following treatment, can be used without specific training and are suitable for use in primary care.[40] These tools are available from: http://www.dermatology.org.uk/quality/quality-life.html

Assessing for comorbidities

Given the increased incidence of comorbidities in this patient population and the overlap with IBD (especially Crohn's disease) and RA, it is advisable to check for other possible symptoms when assessing a patient, including measuring blood pressure, body mass index (BMI), fasting blood lipids, fasting blood glucose and assessing the status of their joints.[9] Of concern, only 39% of patients with a comorbidity in a recent NZ psoriasis survey reported that they were taking measures to manage their associated condition/s.[8]

Approach to therapy

While many therapeutic options exist for the treatment of psoriasis, including topical preparations, phototherapy, systemic therapy and biologic agents, none offer a cure.[32,41] Successful disease management relies on tailoring therapeutic strategies to the particular needs of the patient. It should be explained to patients that psoriasis is a chronic disease (except for guttate psoriasis which often spontaneously resolves) and that treatment is aimed at control rather than cure, with complete clearance possibly not being achievable.[5]

When choosing a treatment, the likes and dislikes of the patient must be considered along with personal time constraints with regard to time available to apply topical therapy.[5] In some cases, patients with mild disease will prefer coping with psoriasis (i.e. not require therapy) than applying creams every day.[5] Sunshine may be enough to clear psoriasis in some cases, but can flare it in some, and fair skinned patients should be warned about the risks of sunburn and long-term overexposure accelerating skin cancers.[15] If patients do desire treatment, they will appreciate a simple regimen. Keep in mind that the best cream to prescribe is the one the patient will actually apply![5]

Most individuals with psoriasis have limited mild-to-moderate disease, (<5% of their BSA).[1] These individuals tend to respond well to topical agents such as limited corticosteroids (which have anti-inflammatory, immunosuppressive and antiproliferative properties), vitamin D analogues, tar products and moisturisers, which generally exhibit a high efficacy-to-safety ratio.[1,15] 70-80% of patients will respond adequately to topical therapy alone.[4]

With regard to managing their condition between flare-ups, patients should be encouraged to continue to optimise lifestyle factors (e.g. maintain sleep hygiene, employ stress management strategies, undertake regular vigorous exercise and ensure weight management through a healthy diet rich in fresh fruit and vegetables) and regularly apply emollients for dry/scaly-prone skin.

Topical therapies

Topical therapies are appropriate for all degrees of psoriasis as well as patients with severe psoriasis receiving systemic therapy. Emollients help reduce itch, facilitate movement in thick scaly areas and reduce the appearance of scales (e.g. Emulsifying ointment, Sorbolene cream, Vaseline, 10% urea cream, salicylic acid-based preparations, coal tar/pine tar solutions and bath oils).[15,32]

First-line therapy may start with the cautious use of topical corticosteroids of varying potency (hydrocortisone, mometasone furoate, betamethasone or clobetasol), topical vitamin D analogues such as calcipotriol [Daivonex®], tar products or calcipotriol in combination with betamethasone dipropionate [Daivobet®], which has proven to be a safe, efficacious and cost-effective option for plaque psoriasis.[42-45] Daivobet® gel is also approved for use for scalp psoriasis.[47] Daivonex® ointment is approved for use in children.[47]

Topical agents can be used both intermittently or continuously.[1] Dermatologists advocate breaks from topical steroid therapy because tachyphylaxis and rebound are frequently seen in psoriasis, and to avoid skin atrophy. Topical vitamin D can be irritating on delicate skin (e.g. intertriginous and facial areas). Calcium levels will need to be monitored if large quantities of topical vitamin D are used.

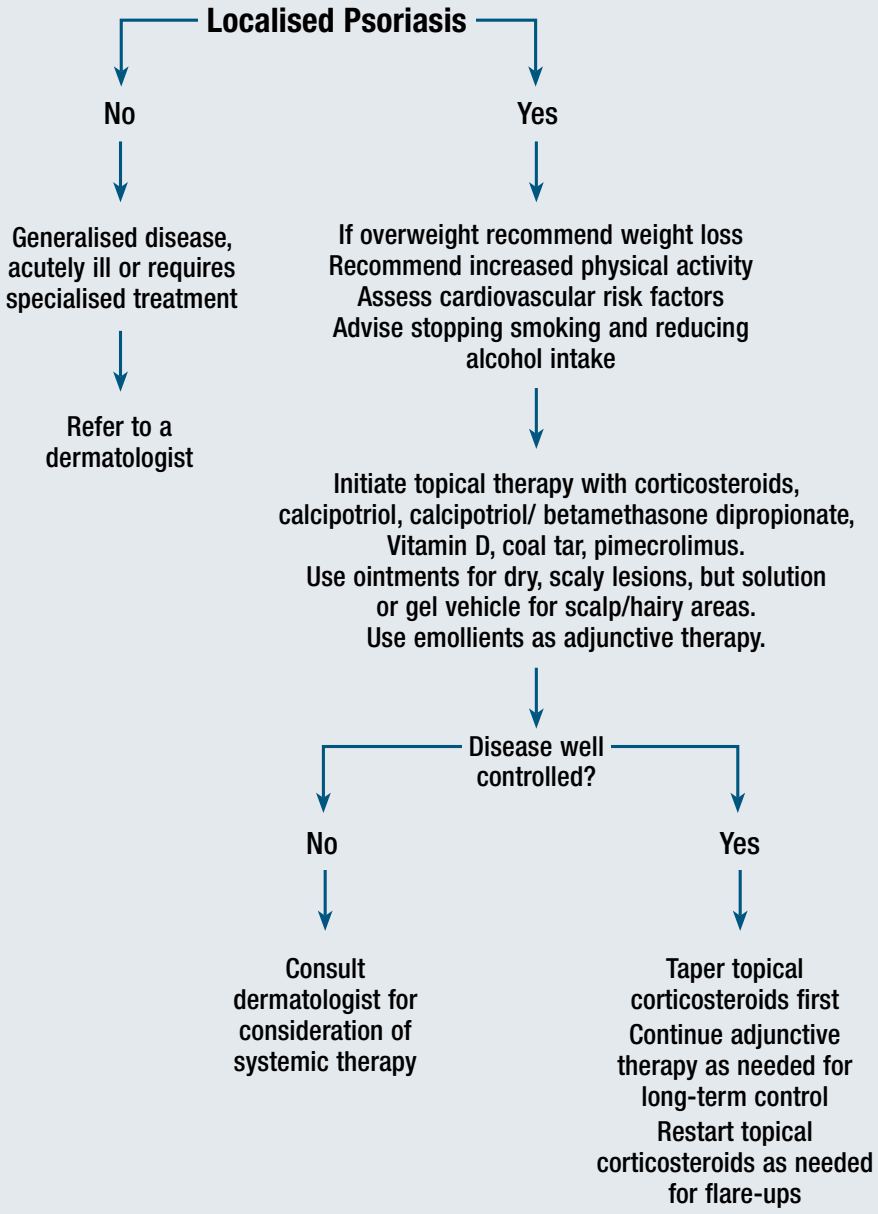
Patients should be advised to use the least potent topical steroid that allows for disease control.[1] Lower potency steroids are usually used for short periods in infants and on the face, while in other areas and in adults, moderate to high-potency corticosteroids are generally recommended as initial therapy.[15] Coal tar 1% solution is less messy than other tar formulations.[5]

For scalp psoriasis, coal tar in combination with salicylic acid and sulphur [Coco-scalp®] is approved for use in NZ, as is Daivobet® gel.[46,48] For psoriasis on the face, genitalia and in the flexures, most dermatologists prescribe mild topical steroids (e.g. hydrocortisone) as short-term treatment only or intermittently (e.g. weekly).[49] Pimecrolimus cream is another alternative for mild disease on the face and genitals and avoids steroid atrophy risk. For nail psoriasis, topical steroid lotions can be applied under the affected nail or referral for systemic therapies may be indicated.[32] Patients should be offered a follow-up appointment within six weeks of initiating or changing topical therapy, in order to assess treatment efficacy and acceptability.

Moving on to more intensive therapy

If topical therapy fails, other treatments available in secondary care include phototherapy, oral agents (methotrexate, acitretin, cyclosporin) and injectable biologics (infliximab, adalimumab) may be prescribed, and these agents are being increasingly used in patients with joint involvement.[1] Most of these therapies need to be prescribed by a dermatologist.

Treatment algorithm for psoriasis in primary care (Adapted from Pardasani et al. 2000[50])



Specialist referral

Specialist referral should be undertaken when there is diagnostic uncertainty, extensive disease (>10-20% BSA involvement), significant impact on quality of life, involvement of difficult-to-treat sites (face, genitalia, palms and soles), failure of appropriately used topical therapy for 2-3 months, acute erythroderma, generalised pustular psoriasis, unstable psoriasis, unexpected adverse reactions to treatment, the patient is pregnant, has psoriatic arthritis, or there is a need for further counselling or patient education.[4,5]

Other appropriate specialist referrals include cardiology, for those with heightened cardiovascular risk factors, rheumatology for those with significant joint symptoms, gastroenterology for those with significant bowel symptoms (there is an increased risk of Crohn's disease with psoriasis), counselling and psychiatry.

Expert commentary on key studies on the management of psoriasis

Effective treatment and improvement of quality of life in patients with scalp psoriasis by topical use of calcipotriol/betamethasone (Xamiol®-gel): results[51]

Authors: Mrowietz U et al

Summary: This prospective, German trial involved 721 patients with scalp psoriasis treated with topical calcipotriol 50 µg/g /betamethasone 0.5 mg/g [Xamiol®] gel for 4 weeks. A significant improvement from baseline in the Physician's Global Assessment and quality of life indices (assessed via a scalp-specific questionnaire) was seen at 4 weeks; mean 4.26 to 2.49 (-41.8%) and 10.57 to 3.22 (-69.5%); both $p < 0.0001$. Among patients who had received previous treatment for their scalp psoriasis, 89.5% (and 87.9% of dermatologists) reported that calcipotriol/betamethasone treatment was better or much better than their previous therapy. Furthermore, 98% of patients and dermatologists reported the tolerability of the agent to be good or very good and 90.4% of patients found it to be easy or very easy to use.

Comment: Scalp psoriasis may cause significant morbidity secondary to pruritus and cosmetic embarrassment from visible scaly erythema, and showers of scales marking clothing. Making a point of examining the scalp of every patient presenting with possible psoriasis not only helps clinicians diagnostically, but also is an important initial step towards improved patient quality of life. Compliance with historical remedies has been hampered by messy, smelly products requiring inconvenient, frequent application and disappointing outcomes. Combination calcipotriol/betamethasone (marketed as Daivobet® in NZ) requires only once daily application, is non-smelly, almost clear, alcohol free, well tolerated and effective (improved quality of life and clinical signs) for medium to long-term use as described in this study. Steroid added to calcipotriol minimises irritation seen with calcipotriol-only products and in turn, calcipotriol reduces (*but does not eliminate*) steroid-induced problems such as atrophy, tachyphylaxis and rebound.

Similar benefits for non-scalp, body plaque psoriasis were seen in the trial by Kragballe et al on Page 7.

A 52-week randomized safety study of a calcipotriol/betamethasone dipropionate two-compound product (Dovobet®/Daivobet®/Taclonex®) in the treatment of psoriasis vulgaris[42]

Authors: Kragballe K et al

Summary: This study randomised 634 patients with psoriasis to one of three treatment regimens: 52 weeks use of calcipotriol/betamethasone dipropionate (two-compound group); 52 weeks of alternating 4-week periods of calcipotriol/betamethasone dipropionate and calcipotriol (alternating group); 4-weeks' use of calcipotriol/betamethasone dipropionate followed by 48-weeks' use of calcipotriol (calcipotriol group). The agents were applied once daily as required. Rates of adverse drug reactions (ADRs) and ADRs of concern associated with topical corticosteroid use in the two-compound, alternating and calcipotriol groups were 21.7% and 4.8%, 29.6% and 2.8% and 37.9% and 2.9%, respectively; odds ratio for an ADR 0.46 (95% CI 0.30-0.70) in the two-compound group compared with the calcipotriol group. Skin atrophy occurred in four patients in the two-compound group, in one patient in the alternating group and in two patients in the calcipotriol group. Folliculitis occurred in three two-compound recipients and one alternating therapy recipient, but did not occur in the calcipotriol group.

Comment: What is relevant for clinicians managing chronic disease is that the combined treatment was used by patients "as required", so mimicking real life adherence to therapy, and though the incidence was small, skin atrophy was seen. Intermittent use when the psoriasis is well controlled is not only what most patients intuitively do, but perhaps should be encouraged by clinicians to facilitate safe long-term use of combined calcipotriol/betamethasone. Moreover, full skin checks at follow-up visits are recommended not only to assess efficacy, but with a high level of vigilance for steroid atrophy, as the sooner culpable therapy is discontinued, the better long-term progression can be avoided and chances of resolution enhanced.

A psoriasis-specific model to support decision making in practice – UK experience[44]

Authors: Freeman K et al

Summary: With the aim of developing an interactive psoriasis model to compare the 2-year outcomes of topical treatment strategies in patients with moderately severe disease in a real-world setting, these UK researchers analysed 1-year economic data on calcipotriol/betamethasone and other topical plaque psoriasis therapies and undertook a literature review and interview programme. The model estimated that topical treatment with high-efficacy first-line therapies in this patient group is a cost effective treatment strategy and predicted potential savings of £126 million in psoriasis care over 2 years in the UK if all psoriasis patients received calcipotriol/betamethasone in a community setting.

Comment: Using effective and well-tolerated products early, mitigates need for referral to secondary care (typically resulting in more expensive therapies), so generating significant socioeconomic health savings.

Topical treatments for chronic plaque psoriasis[52]

Authors: Mason AR et al

Summary: This Cochrane systematic review and meta-analysis included 131 randomised clinical trials, involving 21,448 individuals, comparing the efficacy, tolerability and safety of topical treatments (including vitamin D analogues) for chronic plaque psoriasis against each other and/or placebo. Analysis revealed that vitamin D was significantly more effective than placebo, however, a wide variation in effective size was seen, with the standardised mean difference (SMD) ranging from -0.82 (95% CI -1.34 to -0.29) to -1.90 (95% CI -2.09 to -1.71). In general, all corticosteroids were more effective than placebo: potent corticosteroids SMD -0.95 (95% CI -1.11 to -0.80); very potent corticosteroids SMD -1.29 (95% CI -1.45 to -1.13). While no significant differences in efficacy were seen between vitamin D and potent or very potent corticosteroids, vitamin D in combination with corticosteroids was significantly more effective than either agent alone. Vitamin D was more likely to cause local adverse effects than potent corticosteroids. Topical agents did not differ significantly in their rates of systemic adverse effects.

Comment: Topical corticosteroids and vitamin D were found to be effective, and combined topical corticosteroid/vitamin D superior to either product alone, supporting other studies. It was interesting that there were no suitable study designs supporting the significant efficacy of topical tar products, which is at variance with what is seen in clinical practice. Topical tar has been used for many decades, but the early references were often anecdotal. Cochrane analysis requires double-blind studies and quality of life assessments, among other features. Those were not the criteria used last century when most of the academic work was undertaken with topical tar. Because tar products do typically have an odour to varying degrees, it would be difficult to truly double blind its use. Tolerance for potentially messy or malodorous products has lessened and when there are cosmetically better options, patients would understandably prefer to use those. However, we do not see tachyphylaxis or rebound with topical tar, although we may see irritation if used in flexural areas and folliculitis in hairy regions. As stated in the Cochrane review, head-to-head quality studies (e.g. tar products compared to Daivobet®) are required amongst the topical agents. The current place for topical tar products in psoriasis would be as second-line agents.

The association between physical activity and the risk of incident psoriasis[53]

Authors: Frankel HC et al

Summary: This US study analysed data from 86,655 female nurses participating in the Nurses' Health Study II between 1991 and 2005. During the study period, 1026 incident cases of reported psoriasis diagnosis were documented during 1,195,703 person-years of follow-up. Analysis, adjusting for age, alcohol use and smoking revealed a decreasing risk of psoriasis with increasing physical activity. The most physically active quintile of women exhibited a significantly lower multivariate relative risk (RR) of psoriasis compared with the least physically active quintile; 0.72 (95% CI 0.59-0.89). The risk of psoriasis was reduced in those in the most active quintile undertaking vigorous physical activity; multivariate RR 0.66 (95% CI 0.54-0.81), and a similarly reduced risk was seen in a subgroup of 550 patients with confirmed psoriasis.

Comment: Not only was the incidence of psoriasis inversely associated with increasing levels of vigorous exercise, but in a subgroup of those with confirmed psoriasis the risk of psoriasis was also reduced with increasing levels of vigorous exercise. This finding supports the need for all practitioners involved with psoriatic patients to educate and encourage patients on the benefit of becoming physically active and maintaining high levels of physical activity long-term, as an important way of self-managing psoriasis and furthermore, mitigating psoriasis-associated cardiovascular risk factors.

Diet and weight loss as a treatment for psoriasis[54]

Authors: Gelfand JM and Abuabara K

Summary: This study investigated whether moderate weight loss improves the therapeutic response to low-dose cyclosporin in obese patients with moderate-to-severe psoriasis. A total of 61 adults with a PASI ≥10 and a BMI between 30 and 45 kg/m^2 received cyclosporin 2.5 mg/kg/day and were randomised to receive a dietary intervention (n = 30; caloric restriction of 500 kcal below calculated resting energy expenditure) or no such intervention (n = 31). All subjects were encouraged to exercise for 40 minutes or more per day, four or more times per week. At week 24, the mean weight loss in the calorie-restricted group was 7kg, compared with 0.2kg in the non-calorie restricted group. At that time point, a PASI 75 and PASI 50 response was achieved by significantly ($p < 0.001$) more individuals in the calorie-restricted group than in the non-calorie restricted group; 67% vs 29% and 87% vs 48%, respectively.

Comment: An improved response to cyclosporin was seen in the weight loss-achieving dietary intervention group. Although one could postulate that weight loss would effect a higher blood level of cyclosporin, so explaining the change, a 7kg weight loss in those with a BMI ≥30 but ≤45 would have little impact on cyclosporin 2.5 mg/kg/day, and so the statistically different PASI improvement is more likely related to weight loss. Improved response to therapy is an added incentive for overweight and obese psoriatics to actively reduce calorific intake to achieve weight loss.

Concluding remarks

Psoriasis is now viewed as more than a skin disease, rather, a multisystem disease. The more severe the clinical disease, the higher the risk of comorbidities. So it is important to screen for cardiovascular risk factors and to address obesity, underactivity, smoking and excess alcohol consumption. These will impact on the patient's general wellbeing, psoriasis and other comorbidities. Gaining insight to the impact psoriasis is having on the patient's quality of life will aid how aggressively the disease needs to be treated, choice of topical agents and whether or when referral to appropriate specialities is required.

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