Guidance on the diagnosis and clinical management of psoriasis

S. N. Cohen, S. E. Baron and C. B. Archer, on behalf of British Association of Dermatologists and Royal College of General Practitioners

British Association of Dermatologists, London, UK
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Summary

This article discusses the effects of psoriasis, how to diagnose it confidently, and the options available for treatment, especially in primary care. We also suggest when referral to dermatology should be considered, and try to anticipate some frequently asked questions.

Introduction

Psoriasis is a common inflammatory skin disease, which affects around 2% of the population in the UK. Onset may occur at any age, although two peaks in incidence have been described, one in early adulthood, and the other between the ages of 50–60 years. The disease tends to run a chronic course, with remissions and exacerbations.

The aetiology of psoriasis seems to be multifactorial. Twin studies suggest that approximately two-thirds of the variation in psoriasis risk is heritable. Disease concordance is higher in monozygotic than in dizygotic twins, and the incidence is increased in family members of affected individuals. Various susceptibility genes have been identified, but the precise mode of inheritance is poorly understood.1,2

A triggering factor may be evident at the time of presentation, or when an exacerbation occurs. Infections, particularly bacterial, are often responsible. Streptococci are the most commonly implicated pathogens, classically leading to guttate psoriasis (Fig. 1). Human immunodeficiency virus infection is associated with more severe psoriasis, although it does not increase the likelihood of developing the disease. Psychogenic stress is another well-established factor in triggering or exacerbating the condition. Several drugs are also known to have detrimental effect on psoriasis, notably lithium, antimalarials and, less predictably, beta-blockers. Sudden cessation of systemic or potent topical corticosteroids can also lead to a severe rebound phenomenon, even resulting in erythroderma or generalized pustular psoriasis.

It has been recognized for some time that psoriasis is associated with systemic abnormalities, in particular the metabolic syndrome, involving obesity, hyperlipidaemia and insulin resistance. It also seems that psoriasis, particularly when severe, represents an independent risk factor for myocardial infarction.3 Patients with psoriasis are also more likely to drink alcohol excessively and to smoke. This makes the psoriasis population a good target for assessment of other risk factors and health promotion.4

Effect on quality of life

It should be noted that the extent of skin involvement by psoriasis does not always correlate with the level of symptoms in the individual. Some patients report marked pruritus, whereas others report pain or soreness. Fissures, especially those on the hands and feet, may give rise to severe pain and to difficulty in performing daily activities. Nail disease can also be painful if the nails become very thick or protrudent. Psoriatic arthritis, which occurs in around 5–30% of patients with psoriasis,5 causes further morbidity.

Yet it is the psychosocial impact of psoriasis that, for many patients, leads to a more significant reduction in quality of life. Psoriasis can lead to relationship difficulties, problems finding employment, and low self-esteem.6 Patients often report feeling stigmatized by people staring at their psoriasis, leading them to try to avoid uncovering the affected areas of skin. Social
avoidance and isolation can result, especially if the disease affects exposed areas such as the face or hands. Further detriments to quality of life also result from treatment, due to the mess, smell and the time required for application. The impairment in quality of life due to psoriasis has been found to be equivalent to that from major systemic diseases such as arthritis, chronic lung disease and myocardial infarction. Doctors, including dermatologists, often fail to recognize the extent of psychosocial impairment. It is an important factor to take into account when deciding on treatment, and some patients may be suitable for psychological intervention such as cognitive behavioural therapy.

Presentation and Diagnosis

Psoriasis is usually a straightforward clinical diagnosis. The classic description is of clearly demarcated, erythematous plaques with overlying silvery scales (Fig. 2a). In deeply pigmented skin, the plaques usually have a grey colour, and can give rise to marked postinflammatory hyperpigmentation. Extensor involvement predominates, and scalp involvement is common. Flexural plaques may be seen: here, the scale is often lost due to occlusion. There may be a fissure in the skin crease itself (Fig. 2b).

Atypical presentations can occasionally mimic eczema, discoid lupus erythematosus, dermatophyte infection or mycosis fungoides (cutaneous T-cell lymphoma). Sometimes, signs may be subtle, as with flexural erythema, localized genital psoriasis or mild scaling of the scalp. In such circumstances, careful clinical examination may reveal corroborating features of psoriasis elsewhere. If the diagnosis is in doubt, it is worth specifically checking certain sites which are typically involved. Look for scaling on the scalp and inflammation just behind the ears; erythema and scaling in and around the umbilicus is highly suggestive, and sacral and natal cleft involvement are relatively common. If present, nail changes of pitting, onycholysis and subungual hyperkeratosis can be diagnostic (Fig. 2c).

Another suggestive feature is the isomorphic (Koebner) phenomenon, in which psoriasis affects sites of trauma. This usually becomes evident 10 days to a few weeks after the injury. Linear plaques of psoriasis often represent previous excoriations.

Although most psoriasis is of the chronic plaque variety, some patients exhibit only flexural (inverse) disease; others have the condition confined to the genital area or scalp. Palmoplantar pustulosis may be another variant, or perhaps a distinct disease. It is characterized by sterile pustules, often with associated hyperkeratosis.

Psoriasis can become unstable. This can be due to stress, commencement of exacerbating drugs or cessation of active treatment. The rebound that occurs on suddenly stopping corticosteroids is a relatively common cause. The plaques become much more inflamed and sore, and new plaques can erupt widely over just a few days. This can evolve into generalized pustular psoriasis (Fig. 2d) or erythroderma (> 90% of body surface area is affected). In either of these cases, an urgent dermatological opinion is recommended. Such patients may be systemically unwell, and uncontrolled disease can result in multiorgan failure and even death, particularly in the elderly.

Treatment of psoriasis in primary care

The first step in treatment should involve patient education. It is important to explain that psoriasis is a chronic disease that often waxes and wanes (with the exception of guttate psoriasis, which often responds fully to treatment and may not recur). Most patients have relatively mild disease, but the course is very unpredictable. Patients should understand that treatment is aimed at control rather than cure, and that complete clearance may not be achievable. Some patients will be worried that the rash is contagious or a sign of cancer, and such myths should be dispelled. In conjunction with arriving at a plan of treatment, it is also important to educate patients on how to apply topical therapies.
Treatment selection can be more of an art than a science, or perhaps an art based on science. Although psoriasis severity can be measured fairly objectively with instruments such as the Psoriasis Area and Severity Index (PASI), the true symptomatology is a much more complex function of extent, skin symptoms, patient perception and level of psychosocial impairment. Additionally, more experienced patients may have strong likes and dislikes of certain therapies. There may be other constraints such as time available to apply treatment, whether there is someone to help with application (especially if the patient is frail or disabled), and willingness to use messy or unpleasant-smelling treatments. For a successful outcome, it is essential that all these factors are taken into account. As it is said, the best cream to prescribe is the one the patient will actually apply!

Before discussing specific treatment options, it is worth noting that not all patients require treatment. Indeed, some will prefer coping with psoriasis to the inconvenience of applying creams every day. If treatment is desired, most patients will still appreciate a simple regimen.

**Topical therapies**

The main topical treatment options and their advantages and disadvantages are shown in Table 1.

Because it is a dry skin condition, psoriasis can generally be helped by the application of an emollient. Most of these can also be used as a soap substitute. Soap avoidance is particularly helpful for flexural and genital psoriasis, where soap is especially likely to irritate the inflamed skin.

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**Figure 2** (a) Typical chronic plaque psoriasis. (b) Typical flexural psoriasis. Note that scaling is mainly absent, but can be seen on some of the more peripheral lesions. (c) Psoriatic nail disease, with prominent pitting and onycholysis. (d) Generalized pustular psoriasis. In this case, there is marked inflammation with multiple pustules, but relatively little scale.
Active treatment for typical chronic plaque psoriasis often starts with a topical vitamin D analogue. Calcitriol and tacalcitol are less irritant than calcipotriol, and may be used with care in the flexures and other sensitive sites. However, some patients will still develop irritation. Calcipotriol in combination with a potent once-daily topical steroid is a particularly effective option. There are reassuring safety data for up to 12 months’ continuous use,10 but many dermatologists advocate breaks from steroid treatment to circumvent potential tachyphylaxis (where efficacy decreases over time). This can be achieved by rotating the combination with calcipotriol alone, changing every 4 weeks. Combining a topical steroid with salicylic acid 3% can be useful for hyperkeratotic psoriasis, such as occurs on the palms and soles. The topical retinoid tazarotene offers another clean option for psoriasis generally, but its use is limited by irritation.

Coal tar has been used for many years, and there are many different preparations on offer. Crude coal tar was traditionally used in hospital: it is extremely messy, and the concentration must be increased gradually to avoid irritation. It still has a role in selected patients, although it is less favoured now that systemic treatment options have widened. Coal-tar solution is potentially manageable at home, but requires a motivated patient. Coal tar extract 1% is relatively easy for patients to use at home. It has much less odour than stronger preparations, and less potential to stain clothing. A lotion formulation allows rapid coverage of a wide area.

Table 1 Options available for topical treatment.

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Indication</th>
<th>Main advantages</th>
<th>Main disadvantages</th>
</tr>
</thead>
<tbody>
<tr>
<td>Emollient</td>
<td>Any psoriasis</td>
<td>Helps to lift scale and reduce fissuring; safe</td>
<td>Some patients will find certain products too greasy</td>
</tr>
<tr>
<td>Vitamin D analogue</td>
<td>Most chronic plaque psoriasis</td>
<td>Well-tolerated; easy to use</td>
<td>Can cause irritation, especially in flexures; should not exceed maximum weekly amount</td>
</tr>
<tr>
<td>Mild to moderate potency steroid</td>
<td>Psoriasis at sensitive sites (face/flexures/genitalia)</td>
<td>No irritation; easy to use</td>
<td>Atrophy with long-term use; tachyphylaxis; possible increased risk of cataracts and glaucoma when used on eyelids</td>
</tr>
<tr>
<td>Potent to very potent steroid</td>
<td>In combination with vitamin D analogue at most sites; monotherapy on thick-skinned sites (palms, soles, scalp)</td>
<td>Effective even where there is poor absorption</td>
<td>Atrophy with long term use; tachyphylaxis; rebound on stopping; adrenal suppression if long-term use on wide area</td>
</tr>
<tr>
<td>Salicylic acid</td>
<td>Effective keratolytic; useful for hyperkeratotic disease</td>
<td>Reduces scaling to allow other treatment to penetrate</td>
<td>Irritative to non-hyperkeratotic skin</td>
</tr>
<tr>
<td>Retinoid</td>
<td>Chronic plaque psoriasis</td>
<td>Easy to use</td>
<td>Irritation common; teratogenic</td>
</tr>
<tr>
<td>Coal tar</td>
<td>Any psoriasis</td>
<td>Safe and effective</td>
<td>Smelly; stains skin and clothing; high concentrations can be irritant</td>
</tr>
<tr>
<td>Dithranol</td>
<td>Large plaques</td>
<td>Effective on thick plaques</td>
<td>Severely irritant to normal skin; stains skin and clothing</td>
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this method, a vitamin D analogue may be tried (see above). A low concentration of tar (e.g. the 1% extract) can also be helpful.

Treatments options in secondary care
Where topical therapy fails, other treatments available in secondary care include phototherapy, oral agents and injectable biological therapies. Phototherapy does not suit everyone, as it requires patients to attend hospital 2–3 times per week for up to 3 months or longer. Oral treatments include ciclosporin (which produces a rapid response, but carries a risk of nephrotoxicity and hypertension); methotrexate (more suitable for long-term treatment, but is hepatotoxic, requiring patients to abstain from alcohol); acitretin (a retinoid that is particularly effective in palmoplantar pustular disease, but less so elsewhere) and fumaric acid esters (a treatment widely used in Europe, but which can cause gastrointestinal upset and cramps, as well as flushing – the dose is gradually increased to minimize these adverse events, and blood sampling is also required).

Currently approved injectables comprise the antitu- mour necrosis factor-α agents etanercept, adalimumab and infliximab, and the interleukin (IL)-12 and IL-23 inhibitor ustekinumab. To qualify for these, patients must exceed severity thresholds on both PASI assessment and the Dermatology Life Quality Index; they must also have failed or be unsuitable for standard therapy. The lack of long-term safety data on these drugs was highlighted by the withdrawal from the market of another biological drug, efalizumab, following a small number of deaths from progressive multifocal leucoencephalopathy caused by JC virus reactivation (further information on the use of these drugs in psoriasis is available at http://www.bad.org.uk).

Nail psoriasis poses a particular challenge. A very potent steroid can be tried, applied as a cream to the nail folds, or a scalp application may be used (off-licence), as this can be dripped beneath the nail. Injection of steroid beneath the nail can be effective, but the procedure requires local anaesthetic, and can still be painful despite this. Some nail disease responds well to systemic agents.

Frequently asked questions
How do you differentiate eczema from psoriasis?
Eczema is almost always intensely itchy, whereas many patients with psoriasis do not report itch. Doctors should look for the typical extensor distribution of psoriasis. Specific sites can provide clues (e.g. umbilicus; see above). Psoriatic plaques are very clearly defined, whereas eczema is usually not. Discoid eczema can mimic psoriasis, but does not produce the relatively large, silvery scales seen in psoriasis.

My patient with facial psoriasis complains that whenever she stops the topical steroid for more than a couple of days, the rash returns. What do I do?
This is a difficult scenario. It is worth trying a topical vitamin D analogue or coal tar extract either as monotherapy or in between bursts of topical steroid, bearing in mind that both might irritate. Either of these would be safe for long-term, uninterrupted treatment. A topical calcineurin inhibitor could be tried (off-licence), but this should be initiated by a specialist.

My patient has bad nail psoriasis. What should I advise?
Nails should be kept short, and care should be taken to avoid traumatizing them. Topical treatment as described above can be tried. If symptoms are marked, consider referral.
My patient with psoriasis presents with an exacerbation of chronic obstructive pulmonary disease. Is it safe to prescribe oral steroids?

Problems arise with oral steroids as they are withdrawn. In this case, if the patient requires them for chest disease, they should be given. Rather than stopping them suddenly after a week, they should be weaned down slowly (e.g. by 5 mg decrements each week), which reduces the chances of a rebound of psoriasis.

Further information

Further information and guidelines on psoriasis treatment are available on the website of the British Association of Dermatologists (http://www.bad.org.uk). Patients with psoriasis may wish to contact The Psoriasis Association, Dick Coles House, 2 Queensbridge, Northampton NN4 7BF; Tel.: 08456760076; http://www.psoriasis-association.org.uk. For further reading, see the chapter in *Rook’s Textbook of Dermatology.*

References

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