Psoriasis

Eleanor Higgins

Abstract

Psoriasis is a common, chronic inflammatory skin disease affecting 2 -3% of the UK population. A family history of psoriasis occurs in approximately 30% of patients, and usual age of onset is 20-35 years. Psoriasis is predominantly an immunological T lymphocytedriven disease, involving both the innate and T-cell-mediated immune systems. Chronic plaque psoriasis accounts for 85% of cases. Commonly affected sites include the scalp, extensor surfaces of the knees and elbows, umbilicus, genitalia, anterior lower legs and nails. Psoriasis can significantly impact on a patient's quality of life. Associated co-morbidities include psoriatic arthritis, obesity and the metabolic syndrome, cardiovascular disease and fatty liver disease. Treatment is stratified by disease severity, impact on quality of life, patient preference, relevant co-morbidities and treatment efficacy. Topical treatment such as emollients, tar, vitamin D analogues and corticosteroids are first line for localized/mild disease. In the UK, up to 30% of patients may require specialist referral for phototherapy or systemic therapy (methotrexate, ciclosporin, acitretin, apremilast). Recent therapeutic advances with targeted biological therapies have revolutionized the management of patients with severe disease. Despite this, erythrodermic and pustular forms may still present as life-threatening dermatological emergencies.

Keywords Biological therapies; ciclosporin; methotrexate; palmoplantar pustulosis; psoriasis; psoriatic arthritis; pustular psoriasis; treatment: tumour necrosis factor inhibitor

Epidemiology

In the UK, psoriasis occurs in about approximately 2–3% of the population. Male and female patients are equally affected. The age of onset is usually before 25–30 years, but there is a second peak with some people developing psoriasis age 50–60. Around 75% of cases occur before age 40 years. Most cases are mild and are managed in primary care with topical therapy. Evidence suggests that up to 30% of UK patients require second-line therapy with either phototherapy or systemic therapy.

Socioeconomic burden

Psoriasis represents a significant burden in terms of its effect on quality of life. This has been found to be similar to the impact of ischaemic heart disease, chronic obstructive airways disease and diabetes mellitus.

Eleanor Higgins MB BCh BAO MRCP MSc MEd is a Consultant Dermatologist at St John's Institute of Dermatology, London, UK. Competing interests: Eleanor Higgins has received speaker's honoraria from Leo and educational support for conference attendance from AbbVie UK.

Key points

- Psoriasis is a clinical diagnosis. It is characterized by red plaques with scale at typical sites including the extensor surfaces of the elbows, knees, scalp, natal cleft and umbilicus. Psoriatic arthritis occurs in up to 30% of patients with psoriasis. Patients with moderate/severe disease or who fail to respond to first-line topical therapy should be referred to a dermatology department for second-line therapy
- Newer biological agents have revolutionized the management of severe psoriasis and psoriatic arthritis
- Co-morbidities associated with psoriasis include psoriatic arthritis, metabolic syndrome, inflammatory bowel disease, non-alcoholic steatohepatitis and cardiovascular disease
- Psychological distress, anxiety and depression are common in patients with psoriasis. The Hospital Anxiety and Depression Scale, Patient Health Questionnaire 9 and Generalized Anxiety Disorder 7-item scale are useful screening tools

Pathogenesis

The pathogenesis of psoriasis is a complex interplay between genetic and environmental factors, adaptive and innate immune responses and key inflammatory and epidermal cells that drive the disease. Epidermal and capillary proliferation appear to be stimulated by the release of cytokines from lymphocytes. The mediators involved include interferon- γ , interleukin (IL)-2, tumour necrosis factor (TNF)- α , IL-17, IL-12, IL-23 and IL-8.

Genetic factors

Affected relatives are found in up to 30% of patients with psoriasis. Family and twin studies have indicated an important genetic component to psoriasis.

Two types of chronic plaque psoriasis have been identified, based on age of onset, disease course and association with human leukocyte antigen (HLA) Cw6:¹

- type 1 young onset, positive family history, more severe disease; 80% have HLA-Cw6
- type 2 older onset, peak age of incidence 50—60 years, family history less common, tend to have milder disease; 20% have HLA-Cw6.

Weaker associations are seen with HLA B13, B17 and DR7. Recent key genetic advances include chromosomal localization of the major psoriasis genetic locus, psoriasis susceptibility 1 (*PSORS1*), to 6p21.3. *PSORS1* contributes up to 50% of the genetic risk. Several other genes have also been discovered that point to specific biological pathways involved in epidermal barrier and adaptive and innate immune responses, and may represent future therapeutic targets.

Environmental factors

Infection: upper respiratory tract infections, particularly with streptococci, are associated with disease flares. In 60% of

patients with guttate psoriasis, there is a preceding systemic infection, usually from the upper respiratory tract. Psoriasis can be more severe in individuals with untreated HIV.

Medications: psoriasis can be worsened by several drugs (notably lithium, β -adrenoceptor blockers, antimalarial agents related to chloroquine), although these drugs are often taken without any effect on the patient's psoriasis. Withdrawal of systemic corticosteroids sometimes results in a flare-up.

Both smoking and alcohol excess are more common in individuals with psoriasis than the rest of the population. Excess alcohol is associated with more severe disease, may limit systemic treatment options and can make the psoriasis less stable and more difficult to treat.

Other factors: many patients say that stress induces flares in disease activity. Ultraviolet light worsens psoriasis in some patients. Physical trauma such as surgical trauma, burns and abrasions can also induce psoriasis.

Clinical features of psoriasis - overview

Plaque psoriasis (Figure 1) is the most common presentation, accounting for 80–90% of cases. Psoriatic plaques usually demonstrate three characteristic features of scale, thickening (induration) and redness (inflammation). Plaques are usually symmetrical and typical sites include the scalp, extensor surfaces of the elbows, anterior aspects of the lower legs, umbilicus and natal cleft.

The colour of plaques varies from a dark, dusky red to a paler 'salmon pink' (Figures 2 and 3). The plaque surface is usually scaly, ranging from a fine silvery scale to a thick, adherent scale. Psoriatic plaques may vary from small/guttate (<1 cm) to large plaques. Treated plaques can resolve to leave post-inflammatory erythema, which can persist for several months. Post-inflammatory hypo- or hyperpigmentation is common, especially in darker skinned individuals.

Disease activity fluctuates over a variable timescale of months or years, and larger or smaller areas may be affected at different times. Prolonged remission can occur spontaneously or be brought about by treatment.



Figure 1 Chronic plaque psoriasis of the elbow.



Figure 2 Dusky red plaques with thick scale.



Figure 3 Paler thin plaques.

Diagnosis

Psoriasis is a clinical diagnosis. Clues include a history of long-standing scalp scale/dandruff, scaling in the ears, pruritus in the genital area or arthralgia. The Koebner phenomenon is the development of psoriatic lesions at sites of trauma (surgical wounds, trivial scratches, abrasions, burns). If present, koebnerization may be a helpful clue but can also be seen in other inflammatory dermatoses such as lichen planus. There is no specific blood test for psoriasis.

Psoriasis is associated with several other conditions (Table 1).

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