key points

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Seborrhoeic dermatitis usually starts at puberty with a peak incidence at 40 years of age and is more common in males. Patients develop symmetrical, well demarcated, dull or yellowish red patches and plaques with overlying adherent, yellowish greasy scales. Seborrhoeic dermatitis has a distinctive distribution in areas rich in sebaceous glands – the scalp, eyebrows, glabella, nasolabial and nasofacial folds, cheeks, peri-auricular skin, pre-sternal and interscapular areas. It may occur in flexures, especially the axillae, groin, anogenital skin, infra-mammary skin and the umbilicus. Some patients may develop blepharitis with erythematous eyelids and destruction of eyelash follicles.

Infantile seborrhoeic dermatitis is a similar condition usually starting in the first month of life and resolving by four months. It may be caused by sebaceous glands being active at birth due to stimulation by maternal androgens or nutritional factors. There is no evidence of a genetic role. Mild cases of infantile seborrhoeic dermatitis do not require treatment, for more severe scalp disease softening and removing scales by massaging vegetable/mineral oils into the scalp before bathing in bath water containing a dispersing bath oil is effective.

Seborrhoeic dermatitis appears to be dependent on the presence of the yeast *Malassezia furfur.* At puberty, sebaceous glands become more active and colonisation with *M. furfur* increases. It is hypothesised that *M. furfur* then induces seborrhoeic dermatitis by activating complement through an alternative lipase-producing pathway. Patients with HIV infection, neurological diseases, including parkinsonism and cranial nerve palsies, have a higher incidence of seborrhoeic dermatitis. Patients presenting with sudden onset severe seborrhoeic dermatitis should be screened for risk factors for HIV.

Patients with seborrhoeic dermatitis have a good prognosis, particularly infantile seborrhoeic dermatitis, which usually remits within a few weeks or months and does not recur. The differential diagnosis includes psoriasis, atopic or allergic contact dermatitis, dermatitis secondary to pediculosis or tinea, pityriasis versicolor and drug eruptions. For non-scalp disease first-line treatment includes ketoconazole 2% cream and hydrocortisone 1% cream. For scalp disease, ketoconazole 2% shampoo and zinc pyrithione 1% shampoo are recommended.

Patients should be referred in the following situations: diagnostic uncertainty – consider other differential diagnoses; failure to respond to first-line treatment after four weeks – consider secondary changes e.g. bacterial infection, flexural intertrigo, lichenification, otitis externa; and severe/widespread disease.