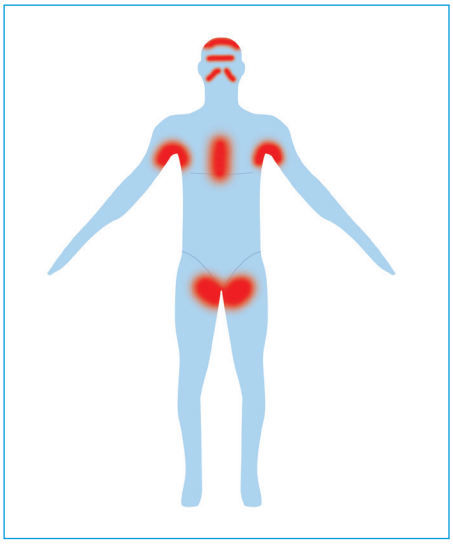
Seborrhoeic dermatitis

Seborrhoeic dermatitis, also known as seborrhoeic eczema, is a common chronic inflammatory skin disorder with a prevalence between 1 and 10% of the general adult population (Naldi, 2010; Bukvic et al, 2012). Men are more likely to be affected than women, with the peak incidence in the third and fourth decades of life.

Seborrhoeic dermatitis is characterized by a red scaly rash affecting the sebaceous (oil producing) areas of the face, scalp, upper chest and back. The axillae and genital region may also be involved in some cases (*Figure 1*, *Table 1*). An infantile form also exists and is often self limiting, although if this is persistent it can develop into a more chronic and diffuse form of the condition (Berk and Scheinfeld, 2010; Del Rosso, 2011). This article provides a stepwise approach to the assessment, diagnosis and management of seborrhoeic dermatitis with a particular focus on the adult form of the disease.

Figure 1. Body sites commonly affected by seborrhoeic dermatitis.



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Aetiology

Numerous risk factors have been implicated in the development of seborrhoeic dermatitis. It is important to note that it is the interaction between multiple factors that determines an individual's susceptibility to the development of seborrhoeic dermatitis.

Malassezia yeasts

There is a correlation between *Malassezia* spp. and the development of seborrhoeic dermatitis (Naldi, 2010; Del Rosso, 2011). *Malassezia* spp. are a type of fungus naturally found on the skin surfaces of humans. It is thought that specific species are implicated in the development of certain conditions including pityriasis versicolor, *Malassezia* folliculitis and possibly atopic dermatitis. Species associated with the development of seborrhoeic dermatitis include *M. restricta* and *M. globosa* (Levin, 2009). *Malassezia* spp. are lipophilic and are often distributed (but not limited to) the sebum (lipid) rich areas of the scalp, face and trunk. The production of lipase by these organisms and the subsequent release of arachidonic acid is thought to cause cutaneous inflammation (Kim, 2009).

The link between sebum overproduction and the hyperproliferation of *Malassezia* is tenuous at best, as individuals with normal sebum production can still manifest seborrhoeic dermatitis. This suggests alternative factors such as sebum composition or immunological predisposition may be to blame (Kim, 2009). What is clear is that cases of seborrhoeic dermatitis often respond well to antifungal therapy, leading to the notion that these commensal yeasts play an important role in its development (Kim, 2009; Del Rosso, 2011).

Immune response

Immune dysfunction is thought to play a role in the development of seborrhoeic dermatitis by allowing the hyperproliferation of *Malassezia* (Amado et al, 2013). Seborrhoeic dermatitis

Table 1. The clinical features of seborrhoeic dermatitis and dandruff in adults

Scalp	From mild desquamation to honey-coloured crusts attached to the scalp and hair, leading to a non-scarring alopecia. May reach into forehead as a scaly erythematous border known as 'corona seborrheica'
Face or retro-auricle area	Forehead, eyebrows, glabella or nasolabial folds. May spread to malar regions and cheeks in butterfly distribution
	Eyelids: yellowish scaling between eye lashes. Can lead to blepharitis with honey-coloured crusts on free margin
	Retro-auricular area: crusting, oozing and fissures. May expand to external canal, with marked itching on occasionally secondary infection (otitis externa)
Upper chest	Petaloid type (common): small, reddish follicular and peri-follicular papules with oily scales at onset that become patches resembling a medallion (flower petals)
	Pityriasiform type: widespread 5–15 mm oval-shaped, scaly macules and patches. Distributed along the skin tension lines (similar to extensive pityriasis rosea). New eruptions can continue for >3 months. Commonly on face and intertriginous areas
Body folds	Moist, macerated appearance with erythema at the base and periphery on axillae, umbilicus, breast fold, genital or inguinal areas. May progress to fissures and secondary infection

From Borda and Wikramanayake (2015)

is more prevalent in patients with lymphoma and organ transplant recipients (Borda and Wikramanayake, 2015). The strongest evidence comes from patients infected with the human immunodeficiency virus (HIV) who have developed acquired immune deficiency syndrome (AIDS). Approximately 30–85% of patients with AIDS are affected with seborrhoeic dermatitis (Amado et al, 2013). While seborrhoeic dermatitis is not an AIDS-defining illness per se, the British HIV Association (2008) suggests offering routine HIV tests to individuals with 'severe or recalcitrant seborrhoeic dermatitis'.

Neurological disease

Seborrhoeic dermatitis has an increased prevalence in a number of neurological and psychiatric conditions. Approximately 52–59% of patients with Parkinson's disease are affected by seborrhoeic dermatitis (Arsenijevic et al, 2014). The pathophysiology of this is complex, including facial immobility contributing to increased sebum accumulation. One suggestion, however, is that patients with Parkinson's disease have increased plasma levels of α-melanocyte-stimulating hormone (Arsenijevic et al, 2014). α-melanocyte-stimulating hormone is responsible for increasing sebum production and is under the direct control of melanocyte-stimulating hormone-inhibiting factor. In patients with Parkinson's disease, there is a lack of melanocyte-stimulating hormone-inhibiting factor because of inadequate dopamine levels. Therefore, patients with Parkinson's disease are likely to have increased sebum levels and are more likely to develop seborrhoeic dermatitis. Furthermore, anti-parkinsonian drugs such as L-dopa are effective in reducing sebum secretion and treating the seborrhoeic dermatitis found in these patients (Arsenijevic et al, 2014; Borda and Wikramanayake, 2015).

There also appears to be a link between neurological damage, for example traumatic brain and spinal cord injury, and seborrhoeic dermatitis (Borda and Wikramanayake, 2015). It is speculated that this may be caused by an accumulation of sebum and scale on inadequately scrubbed skin. Furthermore, dysregulation of sympathetic outflow in areas below a spinal cord injury may lead to an altered immune response and therefore more favourable conditions for the growth of *Malassezia* spp. (Han et al, 2015).

Figure 2. Severe seborrhoeic dermatitis of the scalp.

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Figure 3. Erythematous papules of mild seborrhoeic dermatitis affecting the nasolabial fold.

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Alternative factors

Diet, in particular zinc deficiency, emotional stress and winter months are also implicated in the development of seborrhoeic dermatitis (National Institute for Health and Care Excellence, 2013; Borda and Wikramanayake, 2015).

The presentation of seborrhoeic dermatitis varies depending on the body site affected, age of the patient and skin colour. Seborrhoeic dermatitis usually presents as symmetrical erythematous greasy patches with yellowish scaling. There is a predilection for areas rich in sebaceous glands, namely the scalp (presenting as dandruff; *Figure 2*), hairline, ears, eyebrows, eyelids (blepharitis), nasolabial folds (*Figure 3*), upper lip, upper chest and back, umbilicus and groin (National Institute for Health and Care Excellence, 2013; Oakley, 2014; Borda and Wikramanayake, 2015). Pruritus is not uncommon, but is more often seen when the scalp and ears are affected.

Two types of seborrhoeic dermatitis can appear on the chest, termed either petaloid or pityriasiform (National Institute for Health and Care Excellence, 2013).

- Petaloid: common, red-brown follicular and perifollicular papules with greasy scales. The papules can expand to form patches resembling the shape of flower petals or medallions
- Pityriasiform: less common, thought to be a more severe form of petaloid, presenting as generalized macules and patches that resemble extensive pityriasis rosea.

Generalized seborrhoeic dermatitis is often found in immunocompromised patients and can lead to the development of erythroderma (erythema affecting >90% of body surface area) (National Institute for Health and Care Excellence, 2013; Borda and Wikramanayake, 2015). Genital involvement can manifest as scaly red plaques, which can present a diagnostic challenge particularly if it is the

only area affected. The differential diagnoses of balanitis, psoriasis or even Bowen's disease should be considered in such cases (Salava, 2015).

In infants, scalp disease or 'cradle cap' is the most common clinical manifestation. However, it can also spread to involve the face, armpit and groin folds. The rash consists of salmon pink patches which may be associated with scaling and with little or no pruritus (Oakley, 2014).

Differential and workup

The diagnosis of seborrhoeic dermatitis is usually clinical and often does not require any formal investigation (*Table 2*). However, if the diagnosis is in doubt, a skin biopsy may be needed. Biopsy of seborrhoeic dermatitis lesions show parakeratosis (retention of nuclei of keratinocytes) in the epidermis, plugged follicular ostia (the opening of hair follicles) and spongiosis (oedema) (Clark et al, 2015). Parakeratosis is not exclusive to seborrhoeic dermatitis and can also be found in conditions such as psoriasis (one of the main differentials). It is therefore pertinent that a thorough history and examination should be performed.

When assessing a patient's skin, the clinician should take into account the patient's age, gender, affected sites, distribution and onset of lesions, past medical history (taking particular notice of any immunosuppressive conditions), drug history, family history as well as everyday habits (Bukvic et al, 2012). The diagnosis can be challenging in individuals with darker skin, but the same principles apply (Clark et al, 2015).

Table 2. Differential diagnoses in seborrhoeic dermatitis per body site

Scalp	Psoriasis	Sharply demarcated plaques. Thick, with a silvery scale. Features such as pitting and onycholysis may aid the diagnosis
	Sebopsoriasis	Overlap between seborrhoeic dermatitis and psoriasis
	Tinea capitis	Leading edge (active border), scaly, red, slightly raised with a central clearing. There may be vesicles at the active border. Can lead to a scarring alopecia
	Infected eczema	Eczema which is weeping, crusted and can have pustules
	Systemic lupus erythematosus	Scarring alopecia with disc-like lesions
Face or retro-auricle area	Systemic lupus erythematosus	Photosensitive, maculopapular eruption with fine scaling and a butterfly distribution with sparing of the nasolabial folds. Can be disc-like (discoid)
	Rosacea	Erythematous, eruptions of papules and pustules on forehead, cheeks, nose, and eyes
	Impetigo	Superficial skin infection caused by streptococci and/or staphylococci; begins as vesicles with thin, fragile roof
	Lichen simplex chronicus	Eczematous eruption caused by habitual scratching of single localized area; more common in adults, but possible in children
Upper chest	Atopic dermatitis	Typically affects the antecubital and popliteal fossae. Associated with pruritus and over time, lichenification through repeat scratching
	Pityriasis rosea	Typically a herald patch or 'mother patch'. Salmon pink plaques over the trunk and proximal extremities
	Pityriasis versicolor	Flatter, extensive, less symmetrical than lesions of petaloid seborrhoeic dermatitis. Often noticed after a holiday where the person has been exposed to the sun
	Psoriasis	Numerous subtypes, including plaque, flexural, guttate and inverse. Distinctive red, scaling round-to-oval plaques are common
Body folds	Candidiasis	Often found in the intertriginous regions

From National Institute for Health and Care Excellence (2013); Clark et al (2015)

Management

The main goal of treatment is to reduce the psychological distress that seborrhoeic dermatitis may cause. Patients frequently have concerns about the cosmetic effects of the condition. Any concerns regarding the psychological impact of the disease, should be highlighted through the use of the Dermatology Life Quality Index (Araya et al, 2015). Furthermore, it is important to mention that the condition is chronic, and control rather than cure is the aim. Treatment options include the use of antifungals, keratolytics, corticosteroids and immunomodulators (Clark et al, 2015).

Scalp and beard

Initially, mild seborrhoeic dermatitis of the scalp can be treated with over the counter anti-dandruff shampoo containing selenium sulfide, zinc pyrithione (e.g. Head and Shoulders), or coal tar. Results improve if left in for at least 5–10 minutes before rinsing. Any thickening from scales can be removed before shampooing by applying descaling preparations containing coconut oil and salicylic acid (e.g. Neutrogena T/Gel, L'Oréal Elvive, Selsun and Capasal shampoo). Over the long term, shampoos containing ketoconazole 2% (Nizoral) or ciclopirox 1% (Loprox) can be used two or three times a week. This regimen should be followed for at least 1 month after which it can be reduced to once weekly (as a maintenance therapy) depending on symptom control. Severe scalp inflammation (*Figure 2*) can be treated with potent topical corticosteroids such as betamethasone valerate 0.1%, hydrocortisone butyrate 0.1%, or mometasone furoate 0.1% (National Institute for Health and Care Excellence, 2013; Oakley, 2014; Borda and Wikramanayake, 2015; British Association of Dermatologists, 2015; Clark et al, 2015).

Face and body

Imidazole creams combined with low or mid-potency topical corticosteroids are first line. Examples of imidazole creams include ketoconazole 2%, clotrimazole 1%, econazole 1% and miconazole 2%. Creams are applied twice or three times a day (depending on the type of cream) and are used for a total of 4 weeks. Shampoos used for the scalp can also be used as a body wash alongside the creams. Topical corticosteroids such as hydrocortisone 0.5%, hydrocortisone 1% or clobetasone butyrate 0.05% (Eumovate) can be used

alone, but are more efficacious when used in combination with the creams. Intermittent use of steroids is preferable to continuous use, for the reasons stated previously.

Combination creams containing both an antifungal and steroid are widely available and examples include Daktacort, Lotriderm and Trimovate (the latter is particularly effective in seborrhoeic dermatitis in the groin). Particular care should be taken when treating patients with rosacea as steroids will make this worse. In such cases, patients should be advised to carefully apply steroids to only those areas affected by seborrhoeic dermatitis (National Institute for Health and Care Excellence, 2013; Oakley, 2014; Borda and Wikramanayake, 2015; British Association of Dermatologists, 2015; Clark et al, 2015).

Blepharitis should be managed with good eye lid hygiene, avoiding eye makeup and contact lenses. A warm (but not hot) compress should be applied twice daily with gentle wiping along eyelid margins to help clear debris.

Topical calcineurin inhibitors such as tacrolimus 0.1% ointment (Protopic) and pimecrolimus 1% (Elidel) are alternative second-line (off licence) agents which can be used instead of topical corticosteroids. These agents work by inhibiting T lymphocyte-driven cytokine production and consequently reducing cutaneous inflammation (Cook and Warshaw, 2009). Randomized controlled trials have shown calcineurin inhibitors to be equally as effective as topical corticosteroids and are often used as a maintenance therapy to reduce steroid burden. There is debate about whether long term use should be avoided because of the theoretical risk of skin cancer and lymphoma (Clark et al, 2015). Clinicians are therefore advised to use them on a case by case basis, taking into account their own experiences of using these therapies.

Protopic is only available as an ointment while Elidel only comes in a cream form. Steroids can come in both cream and ointment form. Patient and clinician preference should be considered before deciding which is best to use. As a guide, creams are emulsions of water and oil and are more effective at treating large areas. Ointments contain a higher proportion of oil and are therefore more effective when used on dry scaling skin.

Severe or widespread

The management of severe or widespread seborrhoeic dermatitis should initially focus on treatment of any underlying immunocompromise (if any). This may involve starting individuals on antiretrovirals, for example, if they are HIV positive. Phototherapy, in particular narrow band ultraviolet B, improves symptoms in those with severe seborrhoeic dermatitis. Usually 20–30 treatments are required for it to be beneficial. However, there are a number of disadvantages, including rapid disease relapse and a theoretical risk of skin cancer (usually seen between 250 and 300 treatments) (Bukvic et al, 2012). For these reasons it is not a practical option.

Alternatively, oral antifungals such as itraconazole may be tried, but studies have not shown any significant therapeutic effect (Bukvic et al, 2012), so their use should be balanced against their risk of hepatotoxicity and the cost–benefit ratio. Both phototherapy and systemic antifungal treatment can be used alongside topical agents.

Conclusions

Clinicians should attempt to make a prompt and accurate diagnosis in suspected cases of seborrhoeic dermatitis. This should involve performing a thorough history and examination. An emphasis should be placed on the chronicity of the condition and therefore a control rather than a cure approach is encouraged. Antifungal agents and mild topical steroids are the cornerstones to managing seborrhoeic dermatitis and preventing significant morbidity. **BJHM**

Conflict of interest: none.

KEY POINTS

- Seborrhoeic dermatitis is a common, chronic, inflammatory skin disorder that can affect both adults and children.
- The aetiology is multifactorial, but there is a strong correlation between the presence of *Malassezia* yeasts and the development of seborrhoeic dermatitis.
- Clinical features may vary depending on the body site affected, age and skin colour. Commonly, there is a predilection for areas rich in sebaceous glands.
- The main differential to exclude is psoriasis, but an overlap syndrome termed 'sebopsoriasis' can be present.
- Treatment options include the use of topical antifungals, keratolytics, corticosteroids and immunomodulators. Maintenance therapy is often required.
- Severe or widespread seborrhoeic dermatitis may require the use of systemic agents or phototherapy, depending on clinician and patient preference.

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