Seborrhoeic dermatitis is a common chronic papulosquamous dermatosis with distinct infantile and adult forms, easily recognised clinically. Owing to its frequent recurrence in patients with HIV and AIDS, a careful evaluation of this pathology is warranted. The disease varies from mild to severe exhibiting a variety of forms including psoriasiform, pityriasiform and erythroderma.

Definition and Epidemiology of Seborrhoeic Dermatitis

Seborrhoeic dermatitis is a chronic papulosquamous dermatosis characterised by scaling and poorly defined erythematous patches. This is due to increased sebum production (seborrhoea) in areas of scalp and follicle rich areas of face and trunk.

Even though seborrhoeic dermatitis is one of the most common skin infections, data on its prevalence is limited owing to lack of clearly defined diagnostic criteria or grading scales. It is, however, found to affect nearly 11.6% of the general population with infants being the most vulnerable population. About 70% of infants are affected within the first three years of their lives as opposed to 3-5% of adults being affected, primarily in the third to fourth decade of their lives.
There appears to be an **ethnic predilection** with only few cases seen in African Americans; this claim has however been contended by other researchers supporting an unbiased ethnic predisposition. It is found to be commonly associated with patients having **Parkinson’s disease** or those under **psychotropic drugs treatment** like haloperidol.

**Pathophysiology of Seborrhoeic Dermatitis**

The exact pathophysiology behind seborrhoeic dermatitis remains unknown with a number of theories propounded. Here, we discuss the most popular ones.

**Microbial effects**

Unna and Subauraud first described this disease with an etiology based on **bacteria**, **yeast** or both. The theory did not receive much support in relation to **Candida albicans**, **Staphylococcus**, **Propionibacterium** but a strong correlation seems to exist between seborrhoeic dermatitis and a yeast called **Malassezia furfur**.

There are reasons to implicate this yeast in the causation of seborrhoeic dermatitis such as **positive response to antifungal treatments** and **seborrhoeic-like lesions** in animal models inoculated with this yeast.

These yeasts are said to induce **keratinocyte production of proinflammatory cytokines** causing **skin eruptions**. The final metabolites are also thought to react with triglycerides from **sebaceous glands** leading to inflammatory changes.

**Seborrhoea**

Seborrhoeic dermatitis is not a disease of the salivary gland nor can increased production of sebum be found in all patients. Seborrhoea, however, may be considered a **predisposition** owing to the high incidence of this disease in newborns with large sized and highly active sebaceous glands.

In **childhood**, sebum production and seborrhoeic dermatitis correlate well. In **adults**, however, rates of sebum production are increased during puberty while the disease appears much later. The **commonly affected sites** – scalp, ear, face, and upper parts of the trunk that are rich in sebaceous follicles and glands.

**Miscellaneous**

- **Drugs**: incidence increased in patients exposed to arsenic, gold, neuroleptics
- **Neurotransmitter abnormalities**: found to be associated with certain neurological conditions like **epilepsy**, Parkinsonism, facial paralysis, poliomyelitis, **syringomyelia**
- **Nutritional deficiencies**: zinc deficiency and biotin deficiency may mimic the condition but studies have found administration of zinc and biotin no more effective than placebo.
- **Aberrant epidermal proliferation**: similar to **psoriasis**, epidermal proliferation is increased.
- **Physical factors**: Cutaneous blood flow and skin temperature may be responsible for the distribution seen. Low temperatures and humidity in centrally heated rooms are known to worsen the condition.
Clinical Features of Seborrhoeic Dermatitis

Seborrhoeic dermatitis is characterised by the development of pruritic, erythematous patches with easily detachable greasy large scales. Prominent follicular openings and mild to severe pityriasisiform scales are seen.

Seborrhoeic dermatitis in infants

![Image: "Baby with cradle cap." License: CC BY-SA 3.0]

**Scalp (cradle cap)**
- Frontal and parietal regions involved, may involve retro auricle folds, ear pinna and neck later
- Oily looking thick fissured crust (*crustalactea*)
- No hair loss
- Complications include otitis externa and opportunistic infection with *C. albicans*, *S. aureus*.

**Leiner's disease** (erythroderma desquamatium)
- Non familial or familial C5 dysfunction
- Complication of seborrhoeic dermatitis in infants
- Sudden confluence of lesions
- Universal scaling and redness of the skin (erythroderma)
- Severely ill patients with anemia: diarrhea, vomiting

**Trunk**
- Including flexures and napkin areas

Seborrhoeic Dermatitis in Adults
Scalp

- **Seborrhoeic eczematoid** - mildest form of disease
  - Involves scalp, eyebrows, nasolabial folds, retroauricular area, sternum, shoulder
  - Scaling, mild redness, pruritis

- **Pityriasis sicca**
  - Asymptomatic fluffy white dandruff of scalp

- **Patchy seborrhoeic dermatitis**
  - Classic well known disease with chronic recurrent lesions
  - Involves scalp, temples, glabella with nasolabial folds, retroauricular areas, external ear canal, V shaped areas of chest and back

**Face** (may involve blepharitis)

In some cases, blepharitis is seen as honey-coloured crusts along the rims of eyelids and casts of horny cell debris around the eyelashes

**Trunk**

- **Petaloid**: lesions form clearly outlined round to circinate patches.
- **Flexural**
- **Follicular**
- **Eczematous plaques**
- **Pityriasiform**: oval scaly lesions whose long axis is parallel to the ribs. Mimics pityriasis rosacea.

**Generalised** (may be erythroderma)

**Investigations for Seborrhoeic Dermatitis**

**Histopathology**

**Acute lesions**
- Folliculocentric scale crust composed of orthokeratosis and focal parakeratosis with scattered neutrophils
- Focal spongiosis
- Sparse superficial perivascular infiltrate of lymphocytes and histiocytes

**Subacute lesions**

- Numerous yeast species in the stratum corneum
- Mild psoriasiform hyperplasia
- Features of acute lesion

**Chronic lesions**

- Prominent psoriasiform hyperplasia
- Crusting scales in a folliculocentric distribution (supports seborrhoeic dermatitis when needs to be differentiated from psoriasis)
- Superficial dilation of capillaries and venules
- Minimal spongiosis

**Serology**

In adults with severe, atypical widespread or treatment resistant forms, seborrhoeic dermatitis acts as a cutaneous marker for the presence of HIV/AIDS. Thus, an ELISA test for antibodies may be done when CD4+ counts are between 200 and 500 cells/mm³.

**Differential Diagnosis**

While the diagnosis is usually easily established on clinical grounds, a number of conditions may lead to some confusion.

- **Rosacea**: owing to similarities in distribution
- **Tineacapitis**: can be differentiated from seborrhoeic dermatitis of the scalp on examination. The superficial skin scraping prepared with potassium hydroxide are used to confirm the diagnosis.
- **Wiskott-Aldrich syndrome**: mimics seborrhoeic dermatitis in young patients but purpura and petechiae are additionally seen in the syndrome.
- **In infants**: Most common entity leading to confusion is atopic dermatitis followed by scabies and psoriasis. Langerhan’s cell histiocytosis must be ruled out before making a definitive diagnosis.
- **Other differentials include**
  - Contact dermatitis
  - Erythrasma
  - Dermatomyositis
  - Vitamin B deficiency
  - Zinc deficiency
  - Drug eruption

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<thead>
<tr>
<th>Site</th>
<th>Differential Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Scalp</td>
<td>Psoriasis, dandruff, atopic dermatitis</td>
</tr>
<tr>
<td>Face</td>
<td>Psoriasis, impetigo, contact dermatitis</td>
</tr>
<tr>
<td>Ear canal</td>
<td>Psoriasis, contact dermatitis</td>
</tr>
<tr>
<td>Eyelids</td>
<td>Atopic dermatitis, Demodex follicularum infestation</td>
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<tr>
<td>Chest and trunk</td>
<td>Pityriasis rosacea, pityriasis versicolor</td>
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Management of Seborrhoeic Dermatitis

The goals of therapy are:

- Loosening and removal of scales and crusts
- Inhibition of yeast colonisation
- Control of secondary infection
- Reduction of erythema and itching

In **adults**, the condition is usually **chronic** and therapy is directed at controlling the disease rather than curing it, while in infants the disease being benign and self limited carries a good prognosis.

Treatment for infants

**Scalp**

- Removal of crusts with 3% salicylic acid in olive oil
- Application of low potency glucocorticoids in a cream or lotion for few days
- Topical **antifungal agents** like imidazoles
- Mild baby shampoo
- Proper skin care with soft pastes, creams etc.

**Intertriginous areas**

- Drying lotions such as 0.2-0.5% clioquinolin zinc lotion or zinc oil
- Nystatin or amphotericin B for candidiasis followed by soft and stiff pastes
- 0.1-0.25% gentian violet in cases where oozing matitis seen

Treatment for adults

**Scalp**

- Frequent shampooing with shampoos containing 1-2.5% selenium sulfide, imidazoles, zinc pyrithione, benzoyl peroxide, salicylic acid, coal or juniper tar
- Overnight application of glucocorticoids or salicylic acid to remove crusts or scales
- Avoid tinctures, alcoholic solutions, hair tonics etc. as they aggravate the condition

**Face and trunk**

- Avoid greasy ointments and reduce or omit use of soaps
- Alcoholic solutions are not recommended.
- Low potency glucocorticoids helpful in early stage

**Seborrhoeic otitis externa**
Low potency glucocorticoids based cream are treatment of choice.
- Otic preparations that contain neomycin should be avoided.
- Once under control, discontinue glucocorticoids and apply aluminium acetate containing solution
- Topical pimecrolimus is effective.

Seborrhoeic blepharitis
- Hot compresses with gentle debridement with cotton tipped applicator and baby shampoo once or twice daily
- Resistant cases may use topical antibiotic such as sodium sulphacetamide ophthalmic ointment
- If Demodex folliculorum mites occur in large numbers, antiparasitic drugs like permethrin and benzoyl benzoate may be used.

References
Fitzpatrick's Dermatology in General Medicine

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