Seborrhoeic Dermatitis (Seborrhoeic Eczema) — Causes and Treatment

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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 (seborrhea) in areas of scalp and follicle rich areas of face and trunk.

Epidemiology of Seborrhoeic Dermatitis

Seborrhoeic dermatitis is a common dermatological disorder in the United States. Three age-groups were identified by epidemiologists that show a peak in the incidence of seborrhoeic dermatitis.
The first peak in the incidence of seborrhoeic dermatitis is seen in **infants up to 3 months of age**. The incidence of seborrhoeic dermatitis in this age group can be as high as 42%. The second peak in the incidence of the disease is observed in **adolescents**, while the third is observed in **adults**. The estimated incidence of seborrhoeic dermatitis in adolescents is around 3%, whereas, the incidence in the general adult population is around 1%. The **male to female** ratio of seborrhoeic dermatitis is 1.15 : 1.

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
• **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.

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**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 pityriasiform scales are seen.

### Seborrhoeic dermatitis in infants

**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 oppurtunistic infection with *C. albicans*, *S. aureus*.

**Leiner’s disease** (erythroderma desquamativum)

- 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

**Seborrheic 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)
Seborrhoeic Dermatitis in Patients with Immune-Suppression

Extensive involvement of different areas of the body that are not commonly involved such as the extremities.

The degree of spread of the disease is negatively-correlated with CD4 counts. Meaning, a CD4 count less than 200 cells/mm$^3$ can be associated with more widespread disease than a CD4 count more than 200 cells/mm$^3$. It is commonly associated with rosacea, psoriasis, and acne.

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/mm$^3$.

**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

<table>
<thead>
<tr>
<th>Site</th>
<th>Differential Diagnosis</th>
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<tbody>
<tr>
<td>Scalp</td>
<td>Psoriasis, dandruff, atopic dermatitis</td>
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<td>Face</td>
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<td>Chest and trunk</td>
<td>Pityriasis rosacea, pityriasis versicolor</td>
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<tr>
<td>Intertriginous areas</td>
<td>Psoriasis, candidiasis</td>
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<tr>
<td>All sites, rule out</td>
<td>Secondary syphilis, pemphigus</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% clioquinol 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.

**Systemic Treatment for adults**

Systemic antifungal therapy might be indicated in more severe cases of seborrheic dermatitis.

**Itraconazole:**
- Once daily dose of 200 mg orally for one week followed by a maintenance dose of once 200 mg every other day for one month.
- Liver toxicity has been reported, albeit it is believed to be rare.

**Terbinafine:**
- It is given in an oral formulation in the dose of 250 mg once daily for 4 to 6 weeks.
- It might be associated with a low risk of tachycardia and insomnia.

**References**


Fitzpatrick’s *Dermatology* in General Medicine


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