

# The Pathogenesis and Treatment of Seborrhoeic Dermatitis

## SEBOREİK DERMATİTİN PATOGENEZİ VE TEDAVİSİ

H. Serhat İNALÖZ\*, Necmettin KIRTAK\*\*

\* Assist.Prof., Department of Dermatology University of Wales College of Medicine, Cardiff, UK & Department of Dermatology University of Gaziantep Faculty of Medicine, Gaziantep, TURKEY

\*\* Assist.Prof., Department of Dermatology University of Gaziantep Faculty of Medicine, Gaziantep, TURKEY

### Summary

The importance of seborrhoeic dermatitis has increased again among dermatologists after the AIDS epidemic. Infantile seborrhoeic dermatitis and adult seborrhoeic dermatitis are regarded as different entities. Pathogenesis of infantile and adult seborrhoeic dermatitis are multifactorial. Several mechanisms such as maternal hormones, nutritional factors, atopic diathesis, biotin deficiency, decreased D-6-desaturase activity and *Pityrosporum ovale* play roles in the pathogenesis of infantile seborrhoeic dermatitis. The amount and composition of skin lipids, different strains of *Pityrosporum ovale*, lipase activity, specific immune reactions against *Pityrosporum ovale*, heredity, the work environment and mental stress have an influence on the pathogenesis of adult seborrhoeic dermatitis.

Topical imidazole derivatives and topical lithium ointment are more effective than the other topical remedies in treatment. Systemic treatment such as antihistamines, isotretinoin, ketoconazole, itraconazole, fluconazole or terbinafine can be administered in severe cases.

**Key Words:** Seborrhoeic Dermatitis

T Klin J Med Sci 2002, 22:239-244

### Özet

AIDS'in epidemi yapmasından sonra seboreik dermatitin dermatologlar arasında önemi tekrar artmıştır. İnfantil seboreik dermatit ve adult seboreik dermatit farklı antiteler olarak kabul edilirler. İnfantil ve adult seboreik dermatitin patogenezi multifaktöriyeldir. Anmeden geçen hormonlar, beslenme faktörleri, atopi, biotin eksikliği, azalmış D-6-desaturaz aktivitesi ve *Pityrosporum ovale* gibi çeşitli mekanizmalar infantil seboreik dermatitin patogenezinde rol oynarlar. Derideki lipidlerin miktarı ve yapısı, *Pityrosporum ovalenin* çeşitli tipleri, lipaz aktivitesi, *Pityrosporum ovaleye* karşı spesifik immün reaksiyonlar, heredite, iş çevresi ve mental stres adult seboreik dermatitin patogenezinde etkilidirler.

Topikal imidazol türevleri ve topikal lityum merhemleri diğer topikal ilaçlara göre tedavide daha etkilidirler. Antihistaminler, isotretinoin, ketokonazol, itraconazol, flukonazol veya terbinafinin şiddetli olgularda sistemik tedavi olarak verilebilir.

**Anahtar Kelimeler:** Seboreik Dermatit

T Klin Tıp Bilimleri 2002, 22:239-244

Seborrhoeic dermatitis (SD) was first described in 1887 by Paul Gerson Unna (1). It is a well known chronic dermatitis that involves the areas of the body rich in sebaceous glands. Mostly scalp, face and sometimes body creases are affected. SD is not seen on body sites with no sebaceous glands, such as palms and soles. It consists of a dry form (simple dandruff), with small dry to thick, powdery scales and little to no erythema, and an oily form with greasy or oily scales on an erythematous base (1).

SD consists of the following morphological variants clinically:

#### A- INFANTILE SEBORRHOEIC DERMATITIS

1. Scalp (cradle cap)
2. Trunk (including flexures and napkin dermatitis)
3. Leiner's disease (non-familial, familial C5 dysfunction)

T Klin J Med Sci 2002, 22

#### B- ADULT SEBORRHOEIC DERMATITIS

1. Scalp (dandruff, inflammatory that may extend onto non-hairy areas, e.g. postauricular)
2. Face (may include blepharitis and conjunctivitis)
3. Trunk (pateloid, pityriasiform, flexural, eczematous plaques, follicular)
4. Generalised (may be erythroderma) (2).

#### Pathogenesis

The pathogenesis of SD is unknown. Various factors play roles in the pathogenesis.

#### 1. THE ROLE OF SEBUM

It is obvious that sebum has a role in the pathogenesis of SD. However, no increase in sebum excretion rates or sebum levels has been found in patients with SD. For this reason, "Dermatitis of sebaceous areas" has been suggested as a more accurate term than SD (2,3).

It has been shown that newborns have large sebaceous glands with high sebum excretion rates. Maternal androgen stimulation may have an effect on high sebum in infantile SD. The concurrence of the resolution of lesions with decreasing sebum production during the last six months of infancy supports this suggestion (4,5).

SD seems to be more common in patients with neurological disorders such as Parkinsonism, facial paralysis, supraorbital injury, poliomyelitis, syringomyelia, quadriplegia, unilateral injury to the ganglion Gasser (6). This could be explained by an increased pooling of sebum due to immobility (7). No evidence of neural control of sebaceous glands has been identified (8). Treatment of Parkinsonism reduces sebum excretion and sometimes leads to improve SD in these patients (2).

The amount and composition of skin surface lipids change in SD. The amount of lipid on forehead in patients with SD was found to be significantly higher than in controls (8). The lipid composition was characterised by an increase proportion of cholesterol, triglycerides and paraffin and a decrease in squalene, free fatty acids and wax esters (6). Also alterations in the composition of sebum have a permissive role on the growth of saprophyte lipophilic yeast *Pityrosporum ovale* (*P. ovale*). It was found that treatment of SD with isotretinoin diminished the sebum excretion rate and improved SD significantly (5).

## 2. MICROBIAL EFFECTS

### *Pityrosporum ovale*

Rivolta was first to connect yeasts with skin scaling in 1873 when he described yeast cells present in scales from his beard, which was affected with seborrheic dermatitis and he names the cells "cryptococcus psoriasis" (1). One year later, Malessez considered this fungus responsible for scalp scaling. Castellani and Chalmers introduced the name *P. ovale* in 1913 and succeeded in culturing the organisms in 1925.

In fact, pityrosporum species are part of the normal resident flora in the acroinfundibulum of sebaceous gland (5). However, *P. ovale* is not only a member of human cutaneous flora but also the aetiological agent of several diseases.

In pityriasis versicolor, *P. ovale* changes from the yeast form to mycelial form under the effect of predisposing factors. In pityrosporum folliculitis, it is present in hair follicles and causes itching, follicular papules and pustules. *P. ovale* has been identified significantly in patients with atopic dermatitis, particularly on head and neck and a beneficial effect of antifungal treatment have been reported (9). *P. ovale* has been cultured in confluent and reticulate papillomatosis of

Gougerot and Carteaud and considered to be the aetiological agent of this disease.

No differences in the number of cultured *P. ovale* were noted from the lesions in patients with seborrheic dermatitis, compared to normal skin and controls (8). However, the altered immunological reactions to *P. ovale* is the striking point in the pathogenesis of SD. Total serum IgG and IgA antibody levels were found to be higher than control groups in SD patients (8). This could be the result of a polyclonal activation in connection with skin inflammation. The skin inflammation may be caused by *P. ovale* lipase activity which might produce degradation products. High IgA levels could also be the result of antigen exposure along the gastrointestinal tract. The patients with SD were shown to have low levels of IgG antibodies against pityrosporum protein extracted with several different methods. Furthermore, these patients was noted to have some signs of low T-lymphocyte activation (8-11). It is believed that an impaired T-cell function may facilitate fungal survival in the skin and contribute to a low T-cell dependent antibody response. These findings may explain why SD is so common in AIDS patients. An increased number of pityrosporum yeasts was found in SD lesions of AIDS patients (12).

However, the SD-like eruption in AIDS patients has been suggested as a distinct clinical entity with characteristic histological features. Some other researchers could not find significant cultured *P. ovale* yeasts from HIV positive patients (13). Moreover, HIV seropositive patients with SD treated with topical antifungals did not respond well enough (13).

### *Candida albicans*

*Candida albicans* is usually found in skin lesions and in stool specimens in infantile SD. Even *Candida albicans* has been mentioned as a factor in the pathogenesis of infantile SD, it is not accepted as an important aetiological agent (14). However, a fast and long lasting regression of SD has been reported after vigorous therapy with Nystatin administration orally in adult SD (5).

AIDS patients usually have a history of oral candidiasis before suffering SD (13). It should be borne in mind that SD may develop as an id reaction to a primary candidal infection (5).

### *Staphylococcus aureus*

*Staphylococcus aureus* and *P. ovale* were usually cultured in infantile SD (14). It could be cultured in adult SD patients also. It is believed that *Staphylococcus aureus* is a secondary invader as in atopic dermatitis.

### *Propionibacterium acnes*

*Propionibacterium acnes* counts usually were found to be low in patients with SD. Low free fatty acid levels

could be explained by the low propionibacterium counts from the skin surfaces in patients with SD (6).

### 3. NUTRITIONAL FACTORS

Pyridoxine, biotin and essential fatty acid deficiency have been reported in SD (6). It is believed that they have roles in the pathogenesis of infantile and adult SD. Dramatic responses have also been described in infants with Leiner's disease who were treated with oral and parenteral biotin (2).

There have been no reported cases of spontaneous essential fatty acid deficiency in humans. However, essential fatty acid deficiency may develop after surgical gut resection or after dietary intervention. The skin alterations that develop in adults, even in cases of advanced essential fatty acid deficiency are not disseminated and severe as in essential fatty acid deficiency in infancy (15). On the other hand, altered essential fatty acid levels were found without a nutritional deficiency in infantile SD. The decreased activity of an enzyme was shown in infantile SD. D-6-desaturase is considered to be role limiting enzyme in polyunsaturated fatty acid biosynthesis.

Oleic acid, linoleic acid and arachidonic acid level alterations were noted with impaired function of D-6-desaturase. It has been suggested that a defective function of D-6-desaturase may be important in infantile SD and atopic dermatitis (15,16). Also a decrease in the severity of atopic dermatitis has been shown by supplementation with evening primrose oil which is rich in gammalinolenic acid (17).

### 4. THE ROLE OF ATOPY

A relation between infantile SD and atopic dermatitis is well known. Podmore et al. (18) performed a retrospective study of 92 children from birth to one year of age who were diagnosed as infantile SD. These authors demonstrated that patients earlier diagnosed with infantile SD later have an increased incidence of atopic manifestations. Bergbrant (8), demonstrated that patients with adult SD had a history of asthma or hay fever and childhood dermatitis.

### 5. THE ROLE OF MINERALS

The serum levels copper and magnesium were found to be high in patients with SD. As copper is a component of a caeruloplasmin, a scavenger of oxygen intermediates, this elevation may be a reflection of this anti-inflammatory plasma protein (5).

In patients with transient neonatal zinc deficiency and acrodermatitis enteropathica, erythematous skin lesions mimicking SD were investigated (6). In this disease, prostaglandin E<sub>2</sub> is considered to be important in the transport of zinc across the intestinal mucosa (15).

### 6. THE ROLE OF GENETICS

An increased incidence of SD has been found among identical twins. Moreover, a positive correlation between the patients with SD and presence of SD in their parents was found (8).

### 7. THE ROLE OF HUMIDITY AND TEMPERATURE

Most of the SD patients have severe skin problems in winter and less in summer. Low winter temperatures and low humidity in centrally heated rooms may worsen the SD (6). It has been shown that local occlusion aggravates the previous SD (5). Sweating and overgrowth of *P. ovale* have effects on this condition.

There was a striking correlation between the warm areas of the face and the distribution of lesions in SD. This extended to hair-bearing scalp, which becomes cool when hair is lost. Of interest is that SD clears in hair-bearing areas that become bald. The reason that high skin temperature seems to favour the appearance of SD lesions is unknown. Perhaps *P. ovale*, the organism thought to be an aetiological agent in the pathogenesis of SD, grows best at higher temperatures (19).

### 8. THE ROLE OF DAYLIGHT AND STRESS

Sun exposure has a positive effect on SD. It has been reported that UV light can damage *P. ovale*. A relationship between melatonin secretion and sebum output is estimated (20). Stress has a negative effect of SD and patients with mood depression have been observed to have a high prevalence of SD (20). It is known that depressed and neurologic patients tend to live indoors and avoiding sun exposure. Increased sebum production has been found in patients who spend much of their life indoors. SD is common in patients who have a job indoors. The explanation for the aggravation given by these patients included stress, dry air and pollution.

### 9. SEBORRHOEIC DERMATITIS AND OTHER SKIN DISEASES

SD has been associated with other skin diseases such as pityriasis versicolor and pityrosporum folliculitis. However, the idea that patients with one *P. ovale* related disease are susceptible to other *P. ovale* related disease is not supported. The suggestion that different patients susceptible to different strains of *P. ovale* is more accurate.

SD patients have a psoriatic diathesis and seborrheic dermatitis is a term used for overlapping conditions. SD of face has been observed rarely during the PUVA treatment that can be avoided by masking the face on PUVA therapy (6).

### 10. SEBORRHOEIC DERMATITIS AND AIDS

Recent studies have demonstrated a high prevalence of SD in AIDS patients (5). The high prevalence of SD may be explained by an overgrowth of *P. ovale* and some authors suggest that the term SD is a misnomer and "dysseborrhoeic dermatitis" is more accurate (10). SD is more severe in patients with HIV seropositive than in patients with HIV seronegative.

## 11. SEBORRHOEIC DERMATITIS AND CUTANEOUS DRUG ERUPTIONS

Some drug eruptions particularly due to methyl dopa, cimetidine and chlorpromazine may mimic SD (2).

## 12. SEBORRHOEIC DERMATITIS AND MALIGNANCY

SD is known to occur in association with malignancy (21).

### Investigations and Differential Diagnosis

#### A- INFANTILE SEBORRHOEIC DERMATITIS

Infantile SD may be mixed up with atopic dermatitis and napkin dermatitis. The distribution of rashes are the crucial point for diagnosis. Infantile SD usually affects both napkin area and the other flexures as well. Napkin dermatitis always affects napkin areas. The sparing of the folds is a clue for diagnosis. However, atopic dermatitis usually affects the forearms and shins. Commonly sparing of the napkin area is a striking point.

Sometimes RAST IgE to egg white and milk antibodies helps in diagnosing atopic dermatitis and distinguishing from infantile SD. Skin swabs helps in diagnosing for secondary infection. Stool samples help to identify bowel carriage of candida. Intestinal candidiasis should be eliminated for treatment.

SD should always make one consider histiocytosis X (1). Histiocytosis X has a characteristic petechial component, extracutaneous features and histology. A skin biopsy should be undertaken in any suspicion of histiocytosis (2).

#### B- ADULT SEBORRHOEIC DERMATITIS

Skin swabs can help for identifying the secondary infections. Skin scrapings are sometimes necessary for distinguishing from pityriasis versicolor. Biopsy is not recommended except when it is necessary to exclude other diagnostic possibilities (1). Pathologic features of SD are not diagnostic.

The presence of spongiosis in SD is a differentiating point between psoriasis and SD. The histology of SD in patients with AIDS tends to show more follicular involvement with more plasma cells (2).

## Treatment

SD is one of the most responsive of eczematous dermatoses to treat (5). Treatment differs according to severity of disease, morphological variants and response to previous medicaments.

### A- INFANTILE SEBORRHOEIC DERMATITIS

#### 1. Topical treatment

The condition usually improves over a few weeks even in quite florid eruption effects most of the skin. It should be mentioned to mother that the infant is otherwise perfectly healthy and not distressed by the rash. The basic treatment is keeping the skin dry and relieving the inflammation. Napkins should not be used in warm weather. Frequent change of light clothing is helpful. Soap and talc should be avoided. Tar preparations should be avoided for irritating the eyes in infants.

##### 1.1. Scalp

Arachis oil and olive oil are helpful for removing crusts. Salicylic acid preparations should be avoided. High absorption at this site could lead to salicylism. Low potency steroid lotions are useful for erythema. A water dispersible emollient could be applied. It would be more effective if it was applied an hour or two hours before a bath (2). Occlusive or moist dressings on the head should be avoided because of possible hyperthermia and aggravation.

##### 1.2. Body

The infant should be bathed at least once daily with bath oil. Affected areas should be cleaned with emollients during the bath (2). Topical steroids should be limited between 0.5 % to 1 % hydrocortisone. Topical steroids can be combined with clioquinol for secondary infections. An effective new treatment for infantile SD with Borage oil was suggested (16). Borage oil contains approximately 24% Gammalinolenic acid (GLA) and is obtained from seeds of a plant, *Borago Officinalis* (Borage). It was postulated that all children were free from skin lesions by applying 0.5 ml of Borage oil within 10-12 days to the nappy areas twice daily. No recurrence was observed with intermittent therapy 2-3 times per week. After stopping the treatment at about 6-7 months of age, no relapses were observed (16). Topical ketoconazole cream should be applied when the other treatments failed.

#### 2. Systemic Treatment

Antihistamines can be administered for severe pruritus in syrup. Intestinal candidiasis should be eliminated.

### B- ADULT SEBORRHOEIC DERMATITIS

#### 1. Topical treatment

### 1.1. Scalp

Tinctures, alcoholic solutions, hair tonics and similar products should be avoided. Dandruff responds to frequent washing with shampoos containing selenium sulphide, zinc pyrithione (22), benzoyl peroxide (6) and salicylic acid. Tar extract shampoos are helpful but additional topical therapy is usually required (5). Imidazole derivative shampoos with ketoconazole or fluconazole 2% (23) and bifonazole (24) are more effective than the others. Pityriasis amiantacea may develop in severe cases. Coccois oil and cade oil are helpful for this condition.

### 1.2. Face

Alcoholic solutions or pre or after shave lotions should be avoided (6). Creams are usually preferred cosmetically. Ointments may aggravate the condition due to occlusion of follicles. Corticosteroid creams can be applied to the face. However, potent and long term use of corticosteroids have side effects of atrophy and hyperpigmentation. Low potent 1% hydrocortisone cream is very effective. Imidazoles such as 2% ketoconazole cream (25) and metronidazole gel (6) may be administered also.

Lithium succinate ointment (Efalith) was conducted in patients with AIDS-associated facial SD. Twice daily applications of the ointment brought a rapid improvement (26). Efalith ointment has a potent antiviral effect and was first developed as a possible treatment for oral and genital herpes. A dramatic effect on SD was noted accidentally Efalith alone is sufficient to control SD for all over the skin except from the scalp. Shampoos are required for scalp SD. Lithium blocks the release of free fatty acids from tissues. We know that pityrosporum yeasts are unable to grow without free fatty acids (27).

### 1.3. Trunk

It is difficult to treat this morphological variant. Imidazole derivatives 2 % ketoconazole cream and its combination with steroids are effective (24). Narrow-band UVB phototherapy appears to be a very effective and safe treatment option for patients with severe SD. Treatment was given three times weekly until complete clearing or to a maximum of 8 weeks (28).

### 1.4. Seborrhoeic Intertrigo

The treatment of seborrhoeic intertrigo differs according to the severity. In minor variants, low potent steroids usually help topical antibacterial and anti-candidal therapy should be added in secondary infections. Sometimes soaks and potent topical steroids are required in quite inflamed lesions. Systemic antibacterials are helpful in this type.

## 2. Systemic treatment

Systemic steroids and isotretinoin can be administered in severe cases. Isotretinoin reduces sebum production, drying and erythema. It is suggested in very low dosages ranging from 10 mg daily to 10 mg three times weekly. A short course of oral itraconazole or fluconazole may be tried in severe cases. Oral ketoconazole is effective but inappropriate. Oral terbinafine (250 mg daily), an antimycotic allylamine compound, may be useful in the treatment of SD (29,30).

## REFERENCES

1. White JW. Seborrhoeic dermatitis. In: Sams WM., Lynch PJ. (Eds), Principles and Practice of Dermatology, 2<sup>nd</sup> ed., New York: Churchill Livingstone, 1998: 441-5.
2. Burton JL, Holden CA. Seborrhoeic dermatitis. In: Champion RH, Burton JL, Burns DA, Breatnach SM, (Eds). Textbook of Dermatology. 6<sup>th</sup> ed., Oxford: Blackwell Scientific Publication; 1998: 638-43.
3. Burton JL, Pye RJ. Seborrhoea is not a feature of seborrhoeic dermatitis. Br Med J 1983; 286: 1169-70.
4. Agache P, Blanc D, Barrant C, Laurent R. Sebum levels during the first year of life. Br J Dermatol, 1980; 103: 643-9.
5. Marks R, Schopf R. Seborrhoeic dermatitis. In: Eczema. Martin Dunitz Publications, London: 1992; 129-145.
6. Plewig G, Jansen T. Seborrhoeic dermatitis. In: Freedberg IM, Eisen AZ, Wolff K, Austen KF, Goldsmith LA, Katz SI, Fitzpatrick TB, (Eds). Dermatology in General Medicine, 5<sup>th</sup> ed. New York: McGraw Hill, 1999: 1482-9.
7. Cowley NC, Farr PM, Shuster S. The permissive effect of sebum in seborrhoeic dermatitis: an explanation of the rash in neurological disorders. Br J Dermatol 1990; 122: 71-6.
8. Bergbrant IM. Seborrhoeic dermatitis and Pityrosporum ovale: cultural, immunological and clinical studies. Acta Derm Venereol Suppl (Stockh) 1991; 167: 1-36.
9. Faergemann J, Bergbrant IM, Dohse M, Scott A, Westgate G. Seborrhoeic dermatitis and Pityrosporum (Malassezia) folliculitis: characterisation of inflammatory cells and mediators in the skin by immunohistochemistry. Br J Dermatol 2001; 144: 549-56.
10. Vidal C, Girard PM, Domp Martin D, et. al. Seborrhoeic dermatitis and HIV infection. Qualitative analysis of skin surface lipids in men seropositive and seronegative for HIV. J Am Acad Dermatol 1990; 23: 1106-10.
11. Bergbrant IM, Andersson B, Faergemann J. Cell-mediated immunity to Malassezia furfur in patients with seborrhoeic dermatitis and pityriasis versicolor. Clin Exp Dermatol 1999; 24: 402-6.
12. Schectmen RC, Midgley G, Ray RJ. HIV disease and Malassezia yeasts: a quantitative study of patients presenting with seborrhoeic dermatitis. Br J Dermatol 1995; 133: 694-8.
13. Wikler JR, Nieboer C, Willemze R. Quantitative skin cultures of Pityrosporum yeasts in patients seropositive for the human immunodeficiency virus with and without seborrhoeic dermatitis. J Am Acad Dermatol 1992; 27: 37-9.
14. Broberg A. Pityrosporum ovale in healthy children, infantile seborrhoeic dermatitis and atopic dermatitis. Acta Derm Venereol Suppl (Stockh) 1995; 191: 1-47.

15. Tolesson A, Frithz A, Berg A, Karlman G. Essential fatty acids in infantile seborrheic dermatitis. *J Am Acad Dermatol* 1993; 28: 957-61.
16. Tolesson A, Frithz A. Borage oil, an effective new treatment for infantile seborrheic dermatitis. *Br J Dermatol* 1993; 129: 95.
17. Gehring W, Bopp R, Rippke F, Gloor M. Effect of topically applied evening primrose oil on epidermal barrier function in atopic dermatitis as a function of vehicle. *Arzneimittelforschung* 1999; 49: 635-42.
18. Podmore P, Burrows D, Eedy DJ, Stanford CF. Seborrheic eczema: A disease entity or a clinical variant of atopic eczema? *Br J Dermatol* 1986; 115: 341-50.
19. Hale EK, Bystryn JC. Relation between skin temperature and location of facial lesion in seborrheic dermatitis. *Arch Dermatol* 2000; 136: 559-60.
20. Marietta G, Rongioletti and Rebora A. Seborrheic dermatitis and daylight. *Acta Derm Venereol* 1991; 71: 538-9.
21. Cliff DC, Dodd HJ, Kirby DT, Midgley G, Noble WC. Seborrheic dermatitis and malignancy. *Acta Derm Venereol* 1988; 68: 48-52.
22. Marks R, Pearse AD, Walker AP. The effects of shampoo containing zinc pyrithione on the control of dandruff. *Br J Dermatol* 1985; 112: 415-22.
23. Hay RJ, Graham-Brown RA. Dandruff and seborrheic dermatitis: causes and management. *Clin Exp Dermatol* 1997; 22: 3-6.
24. Segal R, David M, Ingber A, Luire R, Sandbank M. Treatment with bifonazole shampoo for seborrhea and seborrheic dermatitis. A randomised, double-blind study. *Acta Derm Venereol* 1992; 72: 454-5.
25. Katsambas A, Antoniou C, Frangouli E, et al. A double-blind trial of treatment of seborrheic dermatitis with 2 % ketoconazole cream compared with 1 % hydrocortisone cream. *Br J Dermatol* 1989; 121: 353-7.
26. Langtry JA, Rowland Payne CM, Staughton RC, Stewart JC, Horrobin DF. Topical lithium succinate ointment (Efalith) in the treatment of AIDS-related seborrheic dermatitis. *Clin Exp Dermatol* 1997; 22: 216-9.
27. Efalith Multicentre Trial Group. A double-blind, placebo-controlled, multicenter trial of lithium succinate ointment in the treatment of seborrheic dermatitis. *J Am Acad Dermatol* 1992; 26: 452-7.
28. Pirkhammer D, Seeber A, Honigsmann H, Tanew A. Narrow-band ultraviolet B (ATL-01) phototherapy is an effective and safe treatment option for patients with severe seborrheic dermatitis. *Br J Dermatol* 2000; 143: 964-8.
29. Braun-Falco O, Plewig G, Wolff HH, Burgdorf WHC. Seborrheic dermatitis. In: *Dermatology*. 2<sup>nd</sup> ed. Berlin: Springer Verlag 2000: 487-91.
30. Scaparro E, Quadri G, Virno G, Orifici C, Milani M. Evaluation of the efficacy and tolerability of oral terbinafine (Daskil(R)) in patients with seborrheic dermatitis. A multicentre, randomized, investigator-blinded, placebo-controlled trial. *Br J Dermatol* 2001; 144: 854-7.

---

**Geliş Tarihi:** 19.06.2001

**Yazışma Adresi:** Dr. H. Serhat İNALÖZ

Gaziantep Üniversitesi Tıp Fakültesi Dekanlığı,  
Kilis Yolu Üzeri, 27310 GAZİANTEP