Review Article

The management of seborrheic dermatitis 2020: An update

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Abstract

Background: Seborrheic dermatitis (SD) is a chronic relapsing dermatitis manifesting in the seborrheic area, affecting infants or adults. In Indonesia, the prevalence of SD is 0.99–5.8% of all dermatology cases from 2013 to 2015. SD has been known to be a prominent manifestation among HIV patients, but there is an increasing trend in the general population. Therefore, in 2017, the Indonesian Society for Dermatology and Venereology proposed a consensus for the management of SD in Indonesia based on the discussion from 12 dermatological centers. Concurrent with the development of new drugs, this study aims to evaluate and develop a guideline for the treatment of seborrheic dermatitis in Indonesia to update the previous guidelines in 2017.
Methods: Systematic review was based on evidence-based methods, and scientific evidences were acquired through systematic search. Evidence analysis was in accordance with the level of evidence. The available evidences were evaluated, and conclusion was based on the grade of recommendation. Critical appraisal was conducted by experts in dermatology and venereology.
Results: Severity of SD can be determined by using the Seborrheic Dermatitis Area Severity Index. The principle of scalp SD management is controlling the scalp condition in a cost-effective manner to make patients comfortable. The recommendations for treatment of adult SD are topical agents, such as antifungals, nonsteroidal anti-inflammatory agents with antifungal properties, corticosteroids, and calcineurin inhibitors.
Conclusion: We have updated and added newer agents for the treatment of SD. The approach is divided into scalp or nonscalp and also adult or infantile SD.

Keywords: *seborrheic dermatitis, management approach, diagnosis, Seborrheic Dermatitis Area Severity Index, guidelines*

Background

Seborrheic dermatitis (SD) is a chronic relapsing dermatitis manifesting in the seborrheic area. The clinical manifestations range from poorly defined erythematous patch to plaque with flaky white or yellow scales. SD can occur in infants or adults with male predominance in the third to fourth decade of life.[1]

In Indonesia, the prevalence of SD is 0.99–5.8% of all dermatology cases in 2013–2015. On the other hand, the prevalence of human immunodeficiency virus (HIV) infection keeps increasing in Indonesia, resulting in an increase in the overall prevalence of SD and more recalcitrant cases.[1]

The management of SD is targeted to reduce or diminish clinical signs and symptoms, mainly scale and pruritus, and also to maintain remission.[1]

Cheong et al. have developed a guideline for the treatment of SD in the Asian population. Initial treatment for SD can be performed by general practitioners, dermatologists, or a collaboration of both since mild SD can be treated by the former.[2]

In 2017, the Indonesian Society for Dermatology and Venereology had published a National Clinical Practice Guideline for the Treatment of SD in Indonesia. We proposed an update for this guideline due to the increasing incidence of SD and availability of medications in Indonesia.[1]

Methods

This review was based on the data of a 2016–2017 systematic review[1] and an additional review from July 2019 to December 2019. Literature review was conducted based on the principle of evidence-based methods, and scientific evidences were acquired through systematic search. Three databases were used: Cochrane Library, Google Scholar, and PubMed. The keywords used were seborrheic dermatitis, seborrheic eczema, outcome measures, quality of life, management, and therapy.

The level of evidence (LoE) is determined based on the classification developed by Oxford Centre for Evidence-Based Medicine (Table 1).[3] In this study, modifications were made if no high-level evidence was noted. Based on the LoE, the recommendation can be made (Table 2).[4]

Results

Assessment of SD severity

The severity of SD can be determined using Seborrheic Dermatitis Area Severity Index (SDASI), as shown below.[1]

- Determination of the local area score on each area, which is the percentage of SD symptom on the affected area compared to the surrounding area
  1: ≤10%
  2: 11%–30%
  3: 31%–50%
  4: 51%–70%
  5: >70%
- Assessment of the degree of erythema (E), papules (P), and scales (S) on each area:
  0: None
  1: Mild
  2: Moderate
  3: Severe
- Calculation of SDASI score of each area
  (E+P+S) × Local Area Score
- Quantification of all three SDASI scores
- Classification of SD severity according to the SDASI score
  Mild : 0–7.9
  Moderate : 8–15.9
  Severe : >16

Table 1. Level of evidence based on Oxford Centre for Evidence-Based Medicine[4]

Level	Description
1	Meta-analysis or systematic review of randomized controlled trials (RCTs) Individual RCT
2	Nonsystematic review of RCTs Systematic review of cohort studies Individual cohort study
3	Non-RCT study Systematic review of case-control studies Individual case-control study
4	Serial cases and cohort study and case-control study with low validation
5	Expert's opinion without well-defined critical appraisal or only based on physiology

Table 2. Grade of Recommendation Based on Level of Evidence (LoE)[4]

Grade	Definition	LoE
A	Very recommended to be applied	Level 1
B	Recommended to be applied	Level 2, 3 or extrapolation of level 1
C	Can be applied	Level 4 or extrapolation of level 2, 3
D	Not recommended to be applied	Level 5 or other studies not included in levels 1–4

Management

The principle of scalp SD management is controlling the scalp condition in a cost-effective manner to make patients comfortable. The medication for SD should be accepted aesthetically, meaning it can be used with daily hair care products, which leads to an increase of compliance and successful management. Several factors considered prior to determining treatment for SD are age, comorbidities, the potency of agents, efficacy of topical agents, comfortability and compliance, and drug's safety. Patients should also be advised to avoid predisposing factors (use of air conditioner, low humidity, scratching, irritating agents, high-fat food).[1]

Indonesia has implemented several policies regarding management of diseases in primary care facilities. Mild SD without comorbidities can be treated in primary care, while moderate-to-severe SD with and without comorbidities are referred to as secondary or tertiary care facilities. Therefore, we developed an algorithm for mild and moderate-to-severe SD. Different agents were used for scalp and nonscalp SD. Figures 1 and 2 show algorithms for the management of SD. Tables 3, 4 and 5, on the other hand, show the recommendations for management.[1]

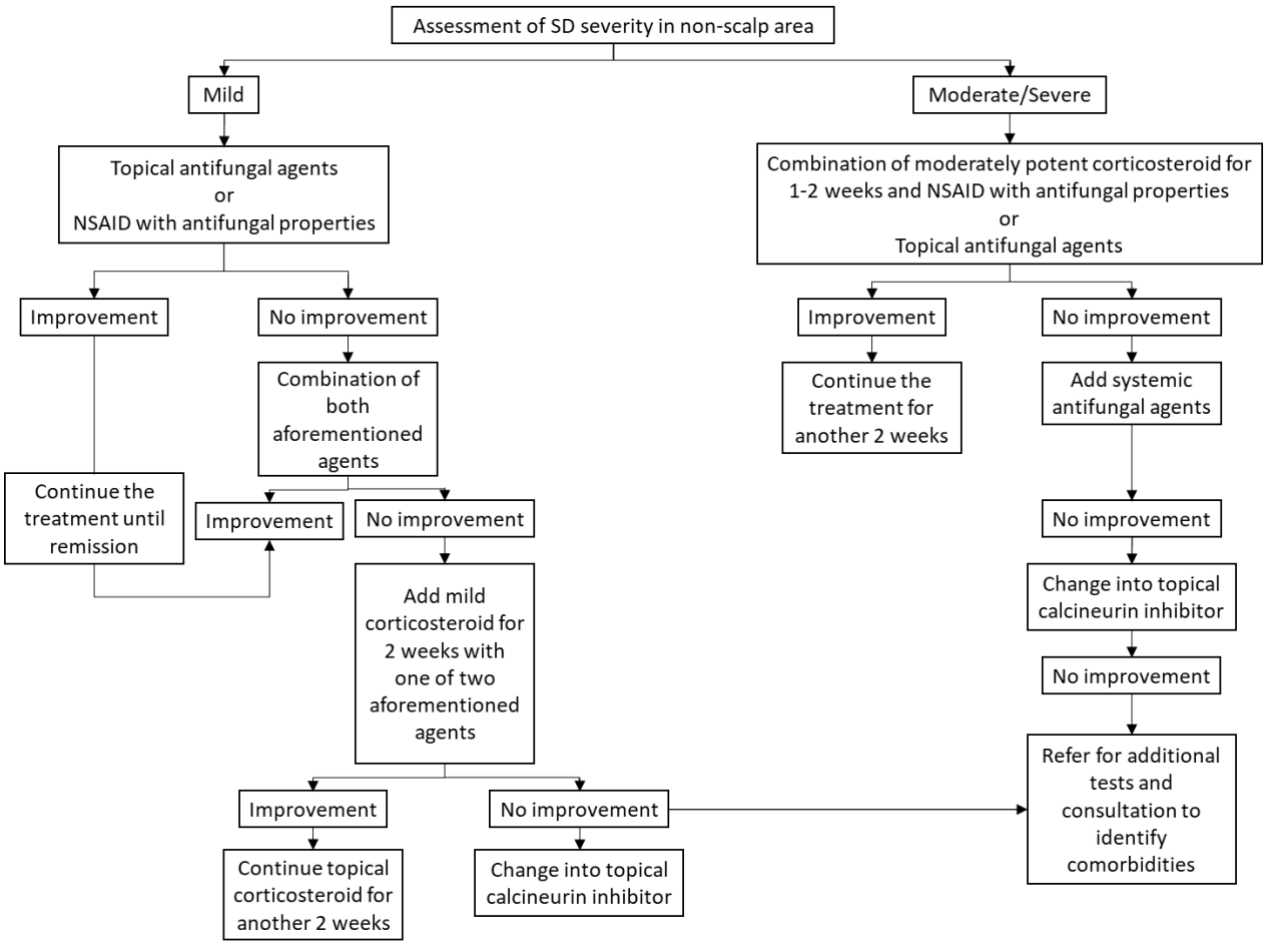

Figure 1. Algorithm for the Management of Adult Seborrheic Dermatitis (SD) in the Nonscalp Area Based on Severity.[1] NSAID: nonsteroidal anti-inflammatory drug
cited with modifications from reference 1

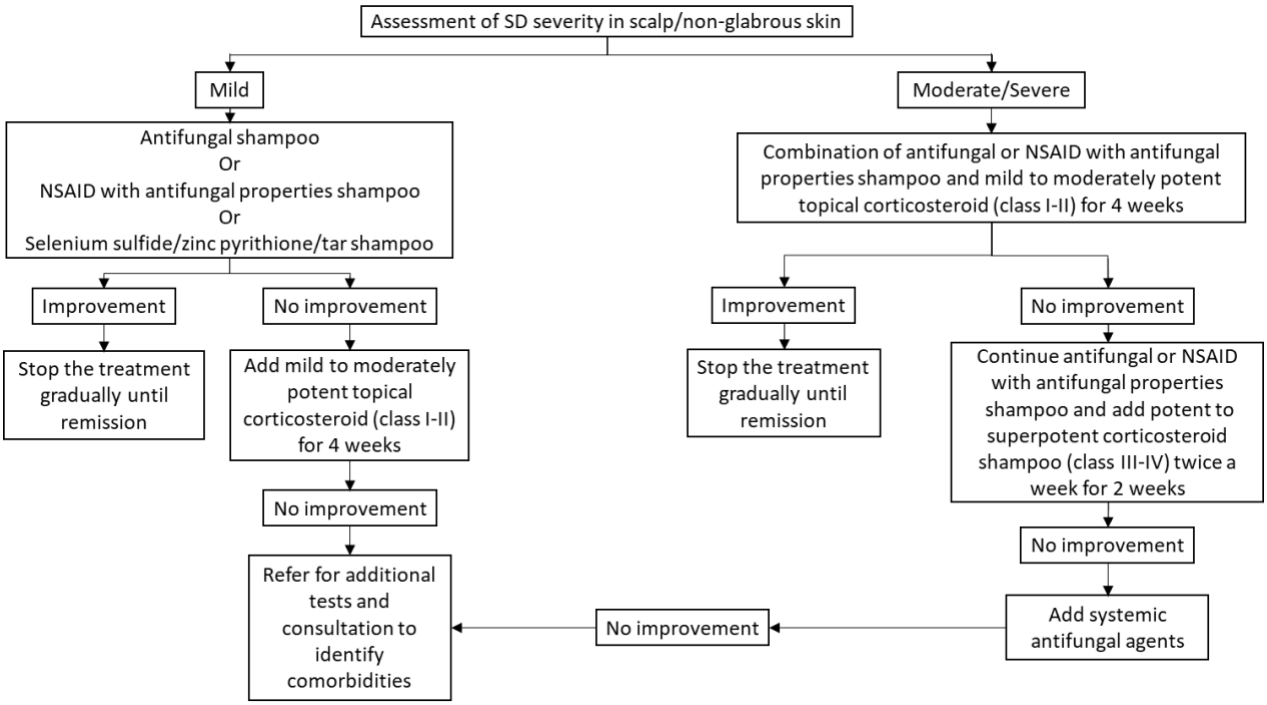

Figure 2. Algorithm for the Management of Adult Seborrheic Dermatitis (SD) in Scalp and Nonglabrous Skin Based on Severity.[1] NSAID: nonsteroidal anti-inflammatory drug

Table 3. Management of Nonscalp Seborrheic Dermatitis[1]

Product	Formula	Dosage	LoE	GoR
Mild SD				
Topical antifungal agents	Ketoconazole cream 2%	Twice a day for 4 weeks	1	A
	Terbinafine cream		1	A
AIAFp (nonsteroidal anti-inflammatory agent with antifungal properties)	Piroctone olamine/algycera or bisabolol cream		1	A
Topical corticosteroid	Hydrocortisone 1% cream and ointment		1	A
Topical calcineurin inhibitor	Pimecrolimus 1% cream		1	A
	Tacrolimus 0.1% ointment		1	A
Moderate-to-severe SD				
Topical corticosteroid (class 2)	Aclometasone 0.05% ointment*	Twice a day for 4 weeks	1	A
	Desonide 0.05% cream		1	A
Systemic antifungal agents	Itraconazole 100 mg capsule	First month: 200 mg/day for 1 week continued with 2 days/month until 11 months	1	A
	Terbinafine 250 mg capsule	Continuous regiment: 250 mg/day for 4-6 weeks Intermittent regimen: 250 mg/day for 12 days per month for 3 months	1	A
	Fluconazole 50 mg capsule	50 mg/day for 2 weeks or 200-300 mg/week for 2-4 weeks	1	A
Others	Phototherapy	Narrowband ultraviolet B 3 times a week for 8 weeks	4	C

GoR: grade of recommendation, LoE: level of evidence, SD: seborrheic dermatitis, *not available in Indonesia, cited with modifications from reference 1

Table 4. Management of Scalp Seborrheic Dermatitis[1]

Medication	Preparation	Dosage	LoE	GoR
Mild SD				
Topical antifungal agents	Ketoconazole 1%–2% shampoo	2–3 times a week	1	A
AIAFp (nonsteroidal anti-inflammatory agent with antifungal properties)	Piroctone olamine/bisabolol/glycyrrhetinic acid/lactoferrin shampoo	2–3 times a week	1	A
Keratolytic and tar	Preparation containing salicylic acid 3%, tar 1%–2% and combination	Salicylic acid shampoo 1–3 times a week	1	A
Other agents	Selenium sulfide 2.5% shampoo Zinc pyrithione 1%–2% shampoo	2–3 times a week	1	A
Topical corticosteroid				
Class I	Hydrocortisone 1% cream	Once a day for 4 weeks	1	A
Class II	Aclometasone 0.05% ointment Desonide 0.05% cream		1	A
Moderate-to-severe SD				
Topical corticosteroid				
Class I	Hydrocortisone 1% cream	Once a day for 4 weeks	1	A
Class II	Aclometasone 0.05% ointment Desonide 0.05% cream		1	A
Topical corticosteroid				
Class III	Mometasone 0.1% solution or cream	Once a day for 4 weeks	1	A
Class IV	Clobetasol propionate 0.05% shampoo	Twice a week, leave on for 5 minutes, for 2 weeks	1	A
Systemic antifungal agents	Itraconazole capsule 100 mg	First month: 200 mg/day for 1 week, followed by 2 days/month for 11 months	1	A
	Terbinafine capsule 250 mg	Continuous regiment: 250 mg/day for 4–6 weeks	1	A
	Fluconazole capsule 50 mg	50 mg/day for 2 weeks or 200–300 mg/week for 2–4 weeks	1	A

GoR: grade of recommendation, LoE: level of evidence, SD: seborrheic dermatitis

Table 5. Management of Infantile Seborrheic Dermatitis[1]

Medication	Preparation	Dosage	Notes	LoE	GoR
Scalp and hairy area					
Topical antifungal agents	Ketoconazole 2% shampoo	Twice a week for 4 weeks	A study in 13 patients (<1-year-old) showed no systemic absorption or liver function disorder after 11-month use	1	A
Emollient	White petrolatum ointment	Daily use	Softens the scales to ease manual release	1	A
AIAFp	Piroctone olamine/alglycera/bisabolol cream	Every 12 hours	Effective for cradle cap	1	A
Nonscalp area					
Topical antifungal agents	Ketoconazole 2% cream	Once a day for 7 days	Can be used alone or in combination with topical corticosteroid	1	A
Topical corticosteroid (class I)	Hydrocortisone 1% cream	Once a day for 7 days	Limited area	1	A

GoR: grade of recommendation, LoE: level of evidence, SD: seborrheic dermatitis

Antifungal agents

The development of antifungal agents is not as progressive as the development of antibacterial agents. Each drug has a specific site of action that causes the efficacy, duration of treatment, and adverse effect to differ from one another.[5]

Systemic antifungal agents

Systemic antifungal agent is the treatment of choice for deep mycosis (subcutaneous mycosis and systemic mycosis) and some superficial mycoses. The choice of agents is determined by several considerations, such type of fungi, cure rate, cost-benefit analysis, adverse effect, drug interaction, comfortability, age, general condition, and patient's medical history. Several things that can cause drug interaction, adverse effect due to insufficient organ function, and teratogenic adverse effect should be taken into account prior to drug administration.[5]

a. Azoles

Azoles are classified into imidazole and triazole based on the number of nitrogen atoms on the azole chain. Ketoconazole is an example of imidazole, while itraconazole, fluconazole, voriconazole, and posaconazole are examples of triazole. Its mechanism of action is the inhibition of lanosterol 14α-demethylase, and cytochrome P450 (CYP450), leading to the disruption of ergosterol synthesis, causing instability and hyperpermeability of the cell membrane, which disrupts fungal growth and life sustainability. The most common adverse effect is gastrointestinal disorder, whereas the less common adverse effects are liver function disorder, cardiac toxicity, hypertriglyceridemia, edema, urticaria, anaphylaxis, neuropathy, hypertension, impotence, and leukopenia. Azole interacts with CYP3A4, so the use of azole with drugs metabolized by these enzymes can cause changes in both drugs' plasma concentrations. Absorption of azole is also disrupted if being consumed with antacid, H2 blocker, and proton pump inhibitor.[5]

b. Allylamines

Allylamines' mechanism of action is inhibition of squalene epoxidase, a non-cytochrome P450 enzyme, leading to the disruption of ergosterol biosynthesis. This class has fungicidal and fungistatic activities. The fungicidal activity is acquired through the accumulation of squalene, which weakens fungi's cell membrane, while fungistatic activity is acquired through ergosterol deficiency, which leads to the disruption of membrane function in fungal growth. The most common adverse effect is gastrointestinal disorder, whereas the less common adverse effects are headache, exanthematous eruption, pustular psoriasis, chest pain, malaise, fatigue, liver disorder, and toxic epidermal necrolysis. This class is metabolized by CYP450, so the drugs inducting or inhibiting this enzyme can disrupt the clearance of allylamines. Other interactions are also reported with the use of cyclosporine, warfarin, and drugs metabolized by CYP2D6. Terbinafine is a systemic allylamine.[5]

c. Terbinafine

Terbinafine is absorbed in the gastrointestinal tract. The preparations are in the forms of tablet and granules. It is lipophilic and keratophilic, so it is widely distributed in the skin, nail, and fat. The bioavailability of terbinafine is 40%, with a half-life of 22 hours. Metabolism occurs in the liver through oxidation by CYP2D6. Elimination occurs mainly through urine (80%) and feces. Rifampin increases the clearance of terbinafine, while cimetidine decreases the clearance of terbinafine. Terbinafine increases plasma concentration of several drugs, such as tricyclic antidepressants, beta blocker, selective serotonin reuptake inhibitor, and monoamine oxidase type B inhibitor.[5]

Corticosteroid

Topical corticosteroid with low-to-medium potency is indicated in severe SD. This agent can be used alone or with an antifungal agent to manage the inflammation, mainly erythema and pruritus. Prolonged use is not advised due to potential adverse effects, such as hypertrichosis, skin atrophy, telangiectasia, perioral dermatitis, etc. Several studies have shown that monotherapy with topical corticosteroid was not superior to topical antifungal agent. Therefore, antifungal agents are still considered first-line treatment for mild SD, but topical corticosteroids are the first-line treatment for moderate-to-severe SD.[6]

Topical AIAFp (nonsteroidal anti-inflammatory agent with antifungal properties)

a. Piroctone olamine

Piroctone olamine is an ethanolamine salt derived from hydroxamic acid. It has antifungal properties and can be found in cosmetic products with 0.5% or 1% concentration. Its mechanism of action is by penetrating the cell membrane, binding to iron ions and making complexes, and then inhibiting the metabolism of energy in the protein mitochondria. This agent is often combined with climbazole, an antifungal agent that binds fungal P-450, leading to the inhibition of P-450-mediated effects. Several studies on the application of climbazole/piroctone olamine cream and shampoo showed effectiveness in reducing erythema and sebum on the face and scalp.[7]

b. Bisabolol
Bisabolol is a monocyclic sesquiterpene alcohol with antioxidant and anti-inflammatory properties. Its mechanism of action is by downregulating polymorphonuclear neutrophil and releasing reactive oxygen species. It is found to be protective of the gastric epithelium from nitric oxide and prostaglandins. This effect is thought to be also applied on the skin due to the similarities between skin and gastric epithelia. For SD, bisabolol is shown to be effective, but it should be used in combination with other agents, such as piroctone olamine, alglycera, and temesteine.[7]

c. Glycyrrhetinic acid
It originates from black licorice. It has anti-inflammatory, anti-irritation, anti-allergy, and antivirus properties. The mechanism of action is by inhibiting 11-β-hyroxysteroid hydroxygenase, leading to the inhibition of hydrocortisone conversion in steroid metabolism, exerting its anti-inflammatory properties. A randomized, comparative study showed that a shampoo containing 18β-glycyrrhetinic acid in ciclopirox olamine and zinc pyrithione had significant clinical improvement, marked by a decrease of erythema, dandruff, and amount of *Malassezia spp.* on the skin surface 2 weeks post-therapy.[7]

d. Lactoferrin
Its mechanism of action is hypothesized by modulation of function, maturation, and migration of immune cells, as well as binding and interaction of iron with other compounds. A previous study measuring the effect of a topical gel containing lactoferrin, ciclopirox olamine, aloe vera, and glycerol-phosphoinositol showed that it caused significant reductions of erythema, itch, and desquamation.[7]

e. Promiseb®
Promiseb® is combination of lycyrrhetinic acid and piroctone olamine cream with antifungal and anti-inflammatory properties. It has been shown to be equally effective as desonide cream in reducing SD signs and symptoms. It is effective in controlling flares with less probability of relapse without any potential serious adverse effects.[7]

Topical calcineurin inhibitors (TCIs)
The mechanism of action for calcineurin inhibitors is by inhibiting T cell-induced cytokine secretion, which will lead to a decrease in inflammation. TCIs are rarely associated with adverse effects. However, prolonged use is not advised due to a report of malignancy in patients using topical calcineurin inhibitors for long-term use. Several studies comparing pimecrolimus cream and topical corticosteroids (betamethasone 17-valerate 0.1% cream or hydrocortisone acetate 1% cream) have shown that they are equally effective in SD. Pimecrolimus cream was also found to demonstrate a milder relapse and longer duration of remission. For tacrolimus ointment, it was shown to have better efficacy than topical corticosteroids but was not superior to zinc pyrithione shampoo. The application of tacrolimus ointment is generally not well accepted by patients due to its greasiness.[7,8]

Tar and keratolytics
a. Coal tar
This agent has keratoplastic properties, which is useful in SD. The previous study showed that the use of coal tar extract 8.75% in liquid detergent daily prior to shampooing and two times per week for 3 weeks was found to improve SD signs and symptoms in 86.4% of the subjects.[7]

b. Salicylic acid/lipohydroxy acid (LHA)
LHA is found to induce stimulation of re-epithelization and desquamation and has antimicrobial properties against *Malassezia ovalis*. Previous study showed that the use of shampoo containing LHA 0.1% and salicylic acid 1.3% was found to reduce erythema, scale, pruritus, and dryness following topical application every 2 days for 4 weeks.[7]

c. K301
K301 or Kaprolac is a mixture of lactic acid, urea, propylene glycol, and a small amount of water and glycerol. It has keratolytic, hydrating, exfoliating, and antifungal properties. Significant clinical improvement can be seen following 2 weeks of treatment. The possible adverse effects are erythema, rash, pruritus, burning sensation, eczema, and ulceration.[7]

Selenium sulfide
This topical agent has antifungal properties by promoting shedding of the stratum corneum in the infected site. Selenium sulfide 2.5% shampoo is found to be effective in treating moderate-to-severe dandruff, especially in the reduction of pruritus and irritation. Unfortunately, it is less tolerated than ketoconazole shampoo due to its residual odor and hair discoloration.[7]

Phototherapy

Ultraviolet has been shown to inhibit *Malassezia* growth on the skin. Ultraviolet B (UVB) can be considered for severe or recalcitrant SD. Previous study showed an improvement in patients treated with NB-UVB three times a week for 2 months. However, relapse occurred in 2–6 weeks following the discontinuation of therapy. On the other hand, the studies of psoralen plus ultraviolet A (PUVA) are still contradictory. Not to mention, a substantial risk of carcinogenic effects in fair-skinned individuals is noted.[6]

Other agents

1. *Anethum graveolens*
   It is a plant from the *Apiceae* or *Umbeliferae* family. Based on a randomized, double-blind comparative study in 115 DS patients on facial skin, the use of *Anethum graveolens* can prevent recurrence. Following an 8-week use, the recurrence rate (21%) was lower in the control group (40%), which could be explained by the regulation effects of *toll-like receptor* (TLR).[9]

2. Emu oil
   Emu oil originates from emu bird's fat tissue (*Dromaius novohollandiae*) with antioxidant and anti-inflammatory properties. A randomized, controlled study in 126 SD patients on facial skin showed reduction in pruritus and erythema as much as 20%. However, its effectivity was lower than that of 1% clotrimazole and 1% hydrocortisone.[9]

3. Hyaluronic acid
   Hyaluronic acid is a non-sulfate anionic glycosaminoglycans, abundant in connective, epithelial, and nerve tissues. A prospective study administering hyaluronic acid sodium salt gel 0.2% in SD patients on facial skin twice a day following facial wash reported a reduction of scales (76%), erythema (64%), and pruritus (50%).[9]

4. Lithium gluconate
   It is a monovalent cation with suspected anti-inflammatory properties. Based on a multicenter, randomized study in 290 DS patients, the effectivity of lithium gluconate 8% ointment (52%) was higher than ketoconazole 2% cream (30.1%). The safety of both drugs is similar. A randomized study showed 90.9% remission in the group using lithium gluconate 8% ointment compared to the control group (54.7%).[9]

5. Nicotinamide
   Nicotinamide is a water-soluble amide, which is also soluble in nicotinic acid. The mechanism of action is by inhibiting sebocyte secretion and anti-inflammatory properties with a dose-dependent manner. In an open-label, randomized study of 48 SD patients administered with nicotinamide 4% cream, a 75% reduction of the total score was shown.[9]

6. Propylene glycol
   It is a non-aromatic substance, acting as a humectant with hygroscopic and absorbability properties. It has been used as an alternative of corticosteroid in the treatment of scalp SD. It can also significantly reduce the colony of *Pityrosporum orbiculare*.[9]

7. Quassia Amara
   It is a substance rich of *triterpenoid quassinoids* with antimicrobial, antifungal, and anti-inflammatory properties. In a close-label, randomized, comparative study, the Quassia extract 4% gel was shown to be effective and safe and can be tolerated for facial SD.[9]

8. Tea tree oil
   It is originated from *Melaleuca alternifolia* with antimicrobial and anti-inflammatory properties. A close-label, randomized study in scalp SD patients showed that this agent can exert better clinical improvement than placebo. However, the use of tea tree oil is limited due to the possibility of estrogenic and anti-androgenic effects.[9]

9. Other natural agents
   Other natural agents, such as *Allantoin, Aloe vera, Borrago officinalis, Arctium lappa, Echinacea purpurea, Incense, Lactoferrin, Potassium alum, Retinyl palmitate, salicylic acid, Tarassaco*, and vitamin E can be used in the management of SD or similar conditions through their roles as moisturizers, keratolytics, anti-inflammation, antioxidant, immunology, antimicrobial, antifungal, sebum and itch modulatory. However, the study on their efficacies and mechanisms of action has not been conducted.

Conclusion

The principles of management in SD are eliminating or improving clinical signs and symptoms, maintaining remission, and avoiding predisposing and precipitating factors. The

recommendations for mild nonscalp adult SD are topical agents, such as antifungals, non-steroidal anti-inflammatory drugs (NSAID) with antifungal properties, class I corticosteroids, and calcineurin inhibitors. **(LoE 1A)**, while the recommendations for moderate/severe nonscalp adult SD are class II corticosteroids and systemic antifungals **(LoE 1A).** Moreover, phototherapy may be recommended **(LoE 4C).** For mild adult scalp SD, the recommendations are the same as nonscalp areas, with the addition of class II corticosteroids and other formulations with keratolytic properties, or mild antifungals. **(LoE 1A).** For moderate/severe adult scalp SD, the recommendations are the same as nonscalp areas, with the addition of corticosteroids ranging from class I to IV **(LoE 1A).** There is no recommendation for phototherapy. The recommendations for infantile scalp SD are antifungal shampoo and NSAID with antifungal properties, with the addition of white petrolatum to ease scaling **(LoE 1A)**, while the recommendations for infantile nonscalp SD are antifungal cream and class I corticosteroids **(LoE 1A).**

This review is an update of the 2017 National Guidelines on the Management of Seborrheic Dermatitis in Indonesia. As an update, several newer agents have been proved effective for the management of SD based on the current literatures.

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