Medical Scope Journal 2026;8(1):110-116
DOI: https://doi.org/10.35790/msj.v8i1.59549
URL Homepage: https://ejournal.unsrat.ac.id/index.php/msj

Coexistence of Alopecia Areata and Seborrheic Dermatitis: A Case Report

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Received: December 17, 2024; Accepted: August 1, 2025; Published online: August 7, 2025

Abstract: Alopecia areata is an autoimmune hair disorder that causes bald patches. AA occurs at all ages with a slightly greater tendency in women. Diagnosis is usually clinical but in atypical cases such as ophiasis, supporting examinations such as trichoscopy or skin biopsy can be performed. Seborrheic dermatitis (SD) is an inflammatory skin disease with scales in areas full of sebaceous glands such as the scalp. Factors that play a role in the pathogenesis of SD include decreased immunity and the role of Malassezia. The diagnosis of SD is usually based on clinical. SD and AA can occur alone or together. In this report, a 44-year-old man came with complaints of patchy hair loss on the scalp for four years accompanied by itchy and scaly red patches on the scalp in the last one month. Based on the history and physical examination, the diagnosis of alopecia areata and seborrheic dermatitis was established. Intralesional corticosteroids, a combination cream of clobetasol and ketoconazole, and loratadine is given. After one week, improvement was seen with the disappearance of scales redness, and hair growth within four weeks of treatment. In conclusion, the treatment performed showed good results to overcome both conditions.
Keywords: alopecia areata; intralesional corticosteroid; seborrheic dermatitis

INTRODUCTION

Alopecia areata (AA) is a hair disorder that causes patchy baldness due to autoimmune disorders.[1] This AA appears as oval or round patches with sharp boundaries in hairy areas, especially the scalp,[2] occurs at all ages, with a slightly greater gender predominance in women than men.[3] The severity can range from slight to total baldness (Alopecia Universalis).[4] Its pathogenesis is thought to be due to a combination of autoimmune responses, especially by cytotoxic T lymphocytes, genetic factors, and environmental factors that contribute to its occurrence.[5]

Typical clinical characteristics of AA include sharply demarcated, round-oval patches of hair loss, with hair forming an exclamation mark, i.e. short hair with a tapered base, often found at the edge of baldness indicating active disease.[3] Nail abnormalities, such as pitting or trachynonychia.[4] AA can appear in various forms such as localized AA, ophiasis, alopecia totalis (AT), and alopecia universalis (AU).[5] AA diagnosis can usually be easily established through anamnesis and physical examination. However, diagnosis is quite difficult in atypical forms such as ophiasis or diffuse forms, which require examinations such as trichoscopy to scalp biopsy.[2,3]

Meanwhile, seborrheic dermatitis (SD) is a skin disease with a typical picture of reddish scaly patches in areas with sebaceous glands such as the scalp and face. The scales that appear can be just peeling like dandruff to oily crusts. Several factors play a role in the occurrence of SD such as immunodeficiency, the role of Malassezia, keratinocyte hyperproliferation, and other factors such as weather.[6] The diagnosis of SD is usually made clinically, dermoscopy and skin biopsy examinations can help in establishing the diagnosis of SD. [7]

Both AA and SD can cause psychological effects and social disorders in individuals such as depression, anxiety, and lack of self-confidence due to visible hair loss and the appearance of reddish patches. The social stigma of baldness also affects a person's quality of life. Stress factors and these two diseases have a reciprocal relationship that influences each other.[4,5,6]

CASE PRESENTATION

A 44-year-old male online driver came to the DV Polyclinic – outpatient department Sumber Waras Hospital – with a primary complaint of uneven hair loss on the scalp for the past 4 years. The pattern of hair loss is localized in several spots in the hair. The first spot appeared at the crown of the scalp, followed by other additional spots without following the hair baldness pattern (Figure 1). These symptoms were localized to the scalp and did not affect other areas of the body. At the beginning there was a small bald spot, then it became larger and followed by other spots. The patient only used hair tonic over the counter to treat the baldness, but no improvement. The patient's father also experienced hair loss. Due to his condition, he expressed a lack of confidence.

Approximately a month before the first consultation, the patient experienced itch, redness, and there was dandruff on the scalp. The patient often scratches the scalp to relieve the itchiness. When the patient stressed out, it became itchier. The patient used a cap to cover the baldness and dandruff, also the patient often used a helmet due to the patient's job. The patient had experienced the dandruff for many years since he was a young adult but never got this inflamed. The patient rarely washed his cap or helmet. He takes a bath once a day and wash his hair 4-5 times a week, and he often changes his shampoo.

Physical examination revealed the patient's vital signs were within the normal range. General status and neurological status were stable, with normal weight and a Glasgow Coma Scale (GCS) score of 15. No abnormalities were noted on the nails. The hair-pull test is positive. Dermatological status shows efflorescence of smooth, non-cicatricial, well-defined red patches in several localized places in the parietal region with varying sizes (nummular to plaque), round shape with firm edges and hair that is easily broken, erosion and squama accompanied (Figure 1a).

Based on the anamnesis and physical examination, the patient was diagnosed with alopecia areata and seborrheic dermatitis. The diagnosis was made clinically without supporting tests due to helathcare coverage liitations. Therefore, the differential diagnoses included tinea capitis, psoriasis vulgaris, and psoriatic alopecia.

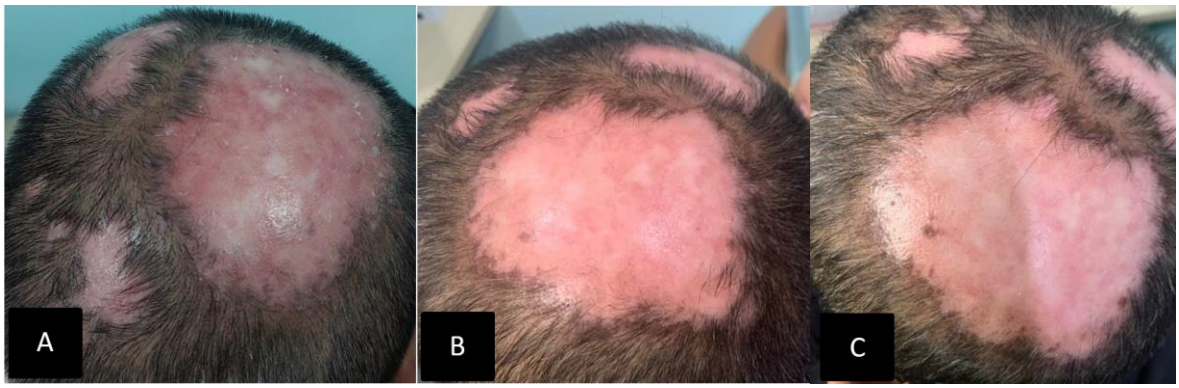

Figure 1. (A) First Consultation showing a redness on the scalp and alopecia areata characteristic. (B) a week after, the redness of the skin is better, the scalling was gone, and several fine hairs appeared (C) a month after, the redness is not seen, more fine hairs growth.

The patient was treated with 0.5 cc of subcutaneous corticosteroids once a week for about four weeks. To cure the SD the patient was given loratadine (second-generation antihistamine) and a topical ointment containing combination of ketoconazole and clobetasol propionate for one week. Education, Information, and Communication were conducted with this patient about his condition, treatment, duration of treatment, prognosis, and side effects of the treatment. After one week, the redness and scaling improved and baby hairs began to grow after four weeks (Figure 1 b,c)

DISCUSSION

Alopecia Areata (AA) is a non-scarring autoimmune disorder in which baldness occurs due to dysfunction of immune system regulation. The etiology is unclear, but there are several contributing factors such as genetics, immunity, and environment. Genetic predisposition is mainly due to variations in the HLA-DR gene which is associated with an increased risk of AA.[4] Other factors such as the environment including trees, viral infections, and trauma help AA conditions in people with genetic potential.[8]

This disease affects 2% of the world's population, with a lifetime incidence of 2.1%. The mean age of onset is 33 years, with a higher incidence in children and young adults.[9] It is slightly more common in women than in men.[9] The condition can be classified based on the extent of hair loss; Alopecia Areata (AA) which is characterized by small and round patches of hair loss, Alopecia Totalis (AT) which is characterized by total scalp hair loss, Alopecia Universalis (AU) which characterized by complete hair loss on the body and scalp, Ophiasis which characterized by Band-like hair loss around the scalp's edge, and Diffuse Alopecia Areata which is more diffuse form resembling telogen effluvium.[9]

The mechanism of AA is caused by an immune-mediated attack on hair follicles. Autoreactive T lymphocytes infiltrate hair follicles, causing follicle dysfunction and hair loss, leading to baldness. Cytokines also act like interferon-gamma (IFN-γ) which triggers inflammation around hair follicles, eventually leading to miniaturization and hair loss.[3,8]

Moreover, AA is usually asymptomatic. Clinically, AA is characterized by clear, well-defined, round, and smooth patches of hair loss without scales. Hair forms an exclamation mark, which is hair that is easily broken with a tapered base and is often seen at the edge of bald patches, indicating active disease. Nail abnormalities can also be accompanied by pitting and onychorrhexis, generally in 10-20% of patients.[10] The diagnosis is usually based on clinical, but in atypical cases, supporting examinations such as trichoscopy and scalp biopsy can be performed. Trichoscopy can show "yellow dots", short hair villi, and "black dots" which help differentiate AA from other hair diseases such as tinea capitis.[11] Scalp biopsy may show different images depending on the phase of the disease.[12,13]

In this case, the patient had chief complain of uneven hair loss in the last 4 years. The patient

reported localized hair loss on the scalp that did not follow a typical pattern of baldness. Initially, the hair loss began as a small patch in the parietal region of the scalp but eventually expanded and was accompanied by additional patches of hair loss in other areas. The lesion's characteristics were consistent with alopecia areata (AA), presenting as localized, non-scarring alopecia with smooth surfaces and well-demarcated borders. A hair pull test was performed, yielding positive results. In this case, a family history of hair loss (the patient's father) may indicate a genetic predisposition. The patient also experiencedincreased scalp pruritus during stressfuk period, supporting a possible role of environmental or psychological stressors. Therefore, the diagnosis of AA was made.

To date, AA can also have a major psychological impact, causing social anxiety, depression, and decreased self-esteem. About 5% of AA can turn into AT or AU.[14] To calculate the impact of AA there is a tool called AASc or Alopecia Areata Scale (Figure 3). In this case, Alopecia Areata Severity Scale was calculated by counting the area of the hair loss. Approximately the score was 22.4%. The psychosocial functioning in this patient was good because the patient didn't have any trouble with his job and social interaction, there were no secondary criteria met. This case indicates a moderate alopecia.

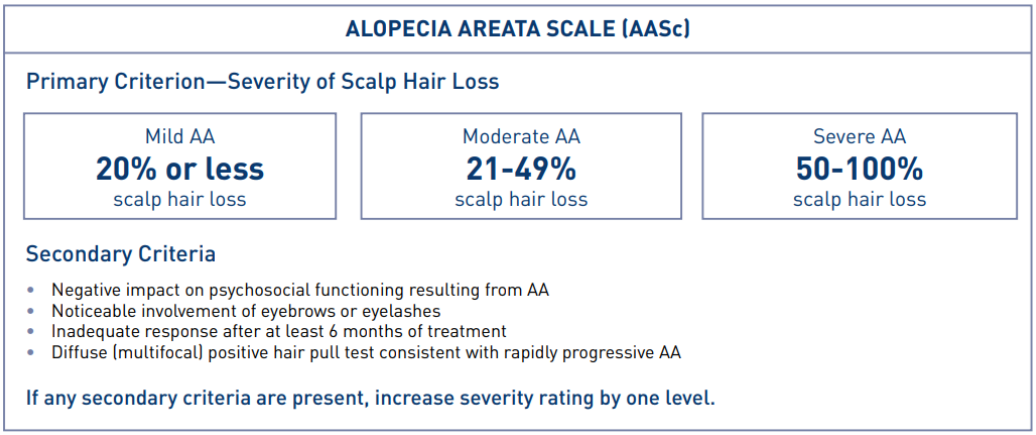

Figure 3. Alopecia Areata Scale Classification[15]

In other hand, Seborrheic dermatitis (SD) is an inflammatory skin disease in areas with many sebaceous glands. The characteristic of this disease is the appearance of reddish patches with thin scales that form an oily crust. SD causes itching, burning, discomfort, cosmetic disorders, and decreased self-confidence. Although anyone can get it, SD is more common in individuals with low immunity such as AIDS or people with neurological disorders such as Parkinson's.[6] In addition, individuals with hypersensitive skin or an allergic tendency (e.g., atopic background) may also be more prone to developing SD due to increased reactivity to Malassezia metabolites and othe environmental triggers.[16]

Seborrheic dermatitis usually appears chronically and recurs, especially in adolescent to young adult populations.[17] The prevalence of SD in the world is around 2.35 - 11.40 % of the general population. It is often associated with changes in season (winter).[18] SD is found in blood rich in sebaceous glands such as the scalp, face, chest, and intertriginous areas. It can also occur in other areas, but the clinical picture is increasingly atypical.[19]

The exact etiology of SD is unknown. The pathogenesis of SD is caused by multiple factors, both endogenous and exogenous. Immunological influences play a role, especially in immunocompromised patients who are more susceptible.[20] The normal human skin flora, *Malassezia*, also plays a role in SD. Although in SD patients the number of *Malassezia* is not significantly different, variations in the types of *M. globosa* and *M. restrica* are thought to play a role in SD. Both species change lipids into irritating free fatty acids so that in susceptible individuals they can cause dandruff-like peeling.[21,22] There is also a hyperproliferation disorder of the epidermis

which has similarities with psoriasis, to distinguish it a skin biopsy can be performed.[23] In Parkinson's disease, SD can occur due to increased sebum levels that allow proliferation of *Malassezia*. Other conditions that worsen SD include; Low humidity and cold temperatures also worsen SD in winter, facial trauma such as scratching, and patients with PUVA therapy.[6]

The diagnosis of SD is usually clinical based on its morphology and characteristics. Dermoscopy examination helps especially in SD on the scalp, the picture that will be seen includes curved circular loops, red dots, red globules, arborizing vessels and atypical red vessels.[7] Scraping of the lesion which is then examined with KOH preparation can be done to confirm the diagnosis of *Pityrosporum folliculitis*. Skin biopsy can also be done in unclear cases.[6]

In this case, the presence of erythema, scaling, and erosion as well as a history of bad hair hygiene, stress-induced itchiness, and a history of recurrent dandruff indicates there's not only AA but also a seborrheic dermatitis. Based on these findings, a clinical diagnosis of alopecia areata and seborrheic dermatitis was made. Alopecia Areata and Seborrheic Dermatitis are two different conditions, both can occur alone or together. In this case, AA coexists with SD.

The AA and SD share similar pathophysiology involving the immune system. However, SD is not an autoimmune disease. In AA the body attacks the hair follicles, while in SD the body's normal flora (*Malassezia*) causes the immune system to react and cause inflammation of the scalp. Both diseases also involve the role of T cells, although the mechanisms are different.[19,24] In this case, SD does not directly cause the AA, but SD can worsen alopecia due to atrophy of the sebaceous glands, chronic inflammation that disrupts the hair growth cycle, and itching that causes repeated scratching.[25,26]

Several treatment options can be given in cases of AA. Many factors influence the choice of treatment such as the severity and extent of AA. Topical corticosteroids are effective for mild to moderate AA. Topical corticosteroids can overcome inflammation and encourage hair follicle growth, the responsiveness rate is 60% for uneven AA.[4] Intralesional corticosteroids are very effective for local AA, with 60-75% of patients experiencing significant hair growth after 6 - 12 weeks.[27] Oral corticosteroids are used in severe AA cases such as AT and AU.[28] Oral corticosteroids cause rapid regrowth, although relapses often occur after treatment is stopped. However, long-term use is not recommended considering the major side effects. Minoxidil can also be used for mild to moderate AA but is less effective in severe AA. Minoxidil can be combined with other treatments.[29] Oral JAK inhibitors (tofacitinib) have shown promising results in the treatment of severe AA.[5] In addition, there are also other immunotherapy agents such as DPCP and SADBE which are used for extensive AA.[29]

Management of SD includes the use of emollients to reduce the side, the use of topical corticosteroids starting from the lowest potency to reduce erythema, scales, and pruritus appropriately, Tacrolimus and Pimecrolimus can be used to inhibit inflammatory cytokines with the same effectiveness as corticosteroids but without the risk of telangiectasis and skin atrophy. In addition, the use of antifungals such as ketoconazole has been proven to be very effective in SD.[6] The use of keratolytic such as ascitic acid and selenium sulfide is also useful for dandruff. Lithium can reduce inflammation and growth of Malassezia. In cases with the extensive distribution of SD can be used short-term systemic glucocorticoids, oral antifungals (Itraconazole, Fluconazole, and Pramiconazole) and Isotretinoin in severe cases.[30] Phototherapy also helps in immunomodulatory and anti-inflammatory functions. [6]

In this case, both AA and SD were treated together at the same time. For AA intralesional corticosteroid injections (0.5 cc of triamcinolone acetonide at a concentration of 10 mg/mL) were conducted (Figure 2). Combined topical corticosteroid (clobetasol propionate 0.05% cream) and antifungal (ketoconazole 2%) is given to treat the SD. Also, topical corticosteroids play a role in AA. This combination demonstrated a synergistic effect in the treatment of alopecia areata (AA) and seborrheic dermatitis (SD). Loratadine twice a day was prescribed as an antihistamine to relieve pruritus. Education, Information, and Communication were conducted with this patient about his condition, treatment, duration of treatment, prognosis, and side effects of the treatment.

After one week of treatment, erythema, pruritus, and scales resolved. (Figure 1b) At the four-week follow-up, fine hair regrowth was observed in the affected areas. (Figure 1c).

The prognosis of AA is still unpredictable, as it depends on factors such as the duration of the condition, the high rate of spontaneous remission, disease progression, and the extent of hair loss. However, timely and appropriate treatment can significantly improve the overall prognosis.[16] Other than that, SD is a chronic condition and can recur especially in adolescence and young adulthood depending on the triggering factors, such as psychological stress, cold or dry weather, hormonal changes, excessive sebum production, or prolonged use of occlusive headwear.[6]

Treatment of SD focuses on controlling symptoms and preventing recurrence. Based on the progress of the treatment, the prognosis in this case was good

CONCLUSION

A case of alopecia areata (AA) and seborrheic dermatitis (SD) was reported in a 44-year-old male patient. The diagnosis of AA and SD was established clinically through history-taking and physical examination. The treatment performed showed good results to overcome both conditions above. Based on the progress of the treatment, the prognosis in this case was good

Conflict of Interest

The authors affirm no conflict of interest in this study.

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