Clinical, Cosmetic and Investigational Dermatology

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CASE SERIES

Alopecia Areata – Effects of Treatment with Upadacitinib – Two Case Reports

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Introduction: Alopecia areata (AA) is a condition in which the immune system attacks the hair follicles, resulting in hair loss. A deeper understanding of AA's underlying mechanisms has contributed to the development of new therapies, such as JAK inhibitors. There are only two EMA-approved medications for AA. Upadacitinib shows much potential, but there are few reports on its effectiveness.

Case Report: The clinical presentation of AA is variable. We report two cases of AA to highlight the varied clinical presentations and diagnostic challenges in our setting and encourage consideration of using upadacitinib as a treatment, which has yielded satisfactory results, with visible regrowth of vellus hair within 3–6 months. In one case, we have a classical form of AA universalis, and in the other one, we have the coexistence of AA with sarcoidosis.

Conclusion: The early inclusion of effective treatment for AA with JAK inhibitors contributes to an improvement in the patient's quality of life, both physically and psychologically, without further aggravating the skin lesions of sarcoidosis.

Keywords: alopecia areata, upadacitinib, JAK1 inhibitor, sarcoidosis

Introduction

Alopecia areata (AA) is an autoimmune disease, one of the non-scarring types of hair loss, characterized by the activation of cytotoxic T cells against hair follicles.[1] AA manifests as patches of bald spots on the scalp and may progress to complete hair loss on the scalp (alopecia totalis) or complete hair loss of all hair of the body (alopecia universalis). Hair loss in AA is associated with lymphocyte infiltration around the hair follicles, and IFN-γ plays a role in the immune response. In addition, AA often co-occurs with other autoimmune diseases or allergic conditions and can be triggered or exacerbated by infections or inflammatory processes.

One of the diseases that can occur in conjunction is sarcoidosis. While sarcoidosis and AA are distinct diseases, their immune-mediated nature and shared cytokine pathways suggest that coexistence, though rare, is plausible.[2] Recognizing this overlap can help guide diagnosis, biopsy decisions, and treatment planning, particularly when considering therapies like JAK inhibitors, which are gaining more and more recognition in treating a variety of skin conditions. Although much of the current evidence comes from isolated case reports or small series, it reflects a growing enthusiasm and the considerable potential of JAK inhibitors in managing previously treatment-resistant or rare skin disorders. Promising responses have been observed in diseases such as cutaneous sarcoidosis, dermatomyositis, necrobiosis lipoidica, hypereosinophilic syndrome, lichen planus, and mastocytosis.[3]

Another group of diseases associated with AA are allergic conditions, such as asthma, eczema, or reactions to pollen, dust, and cats. Each of them contributes to an elevated AA risk, influencing the onset and relapse of AA.[4,5] The risk of developing AA in patients with asthma has increased by 1.86 times.[6]

There are studies showing efficacy in improving the condition of AA with using upadacitinib. The treatment's effect is visible not only in patients with various comorbidities, both skin related and internal, but also in patients without any associated conditions.[1,7]

https://doi.org/10.2147/CCID.S549384
Received: 3 August 2025
Accepted: 11 January 2026
Published: 19 February 2026

Upadacitinib's safety and effectiveness have been noted in recent studies. We can see that upadacitinib administration shows great success in the treatment of AA with comorbidities such as atopic dermatitis and vitiligo, but also without coexisting conditions. Upadacitinib, administered at 15 mg/day, the same dose as our patients, has been an effective and safe treatment option in cases of adolescents with severe AA, as some of the recent studies have concluded.[8,9] The drug inhibits phosphorylation of downstream effector proteins, thereby suppressing cytokine signaling in key pathways associated with inflammatory diseases. The compound reversibly inhibits IL-6 and IL-7 cytokine signaling in vivo in a concentration-dependent manner. As a result upadacitinib is an effective drug for the treatment of several chronic inflammatory diseases such as rheumatic, dermatologic, and gastrointestinal diseases.[10]

Aim of the Study

The case study aimed to demonstrate the potential of treating patients with AA using modern treatments, such as JAK 1 inhibitors, specifically upadacitinib.

Case 1

A 65-year-old female patient was admitted to the hospital in good general condition due to visible worsening of skin lesions associated with sarcoidosis observed over the past month. On the skin of the back, arms, and abdomen, erythematous and papular lesions were visible. Additionally, the patient presented with livedo reticularis on the upper and lower extremities.

In addition, the patient had the following comorbidities: hypertension, type 2 diabetes, bronchial asthma, nodular goiter, hepatic steatosis, left renal cortical cyst, and the presence of maxillary and ethmoidal sinus polyps.

The suspected diagnosis of skin sarcoidosis was made based on histopathological samples taken from a lichenoid rash on the back and hand dorsum. Pulmonary sarcoidosis was not confirmed, even though the patient presents with mediastinal lymphadenopathy, due to the inconclusive, inconsistent histopathological examinations. In the laboratory tests performed, monocytosis, elevated thyroglobulin antibodies, slightly elevated inflammatory exponents could be observed, along with positive lupus anticoagulants.

One year prior, the patient was hospitalized for the diagnosis of generalized alopecia. She reported severe hair loss that had progressed over the previous nine months, with complete hair loss throughout the body. She also reported being lethargic and slowed down with a depressed mood. During hospitalization, scalp biopsies were taken. On histopathological examination, the picture is consistent with AA.

Based on the results from both hospitalizations in two consecutive years, a diagnosis of AA on the scalp with overlapping sarcoidosis on the skin of the torso was concluded. Treatment with 250 mg chloroquine daily was administered, with good results - a slight regrowth of mesquite hair on the scalp was visible.

Over the last two years, the patient has remained under the care of the Dermatology Outpatient Clinic, where treatment with upadacitinib 15 mg/day, topical minoxidil once daily, and a solution of estradiol benzoate, prednisolone, and salicylic acid once daily has been administered on the scalp. After one month of treatment, dermoscopy revealed visible dark hairs, with exclamation hairs also present. After one year of administering upadacitinib treatment hair regrowth approx. 2 cm was visible. Dark hairs and exclamation hairs present in dermatoscopy. The results of the treatment are visible in Figures 1 and 2. Post two-year evaluation shows regrowth on the entire scalp. No visible signs of disease activity. Patient is feeling much better, she did not report any adverse effects during the two-year treatment period.

Case 2

A 17-year-old male patient attended the Dermatology clinic due to alopecia, which started with a single outbreak which can be seen in Figure 3. After one month, diffuse alopecia covered the entire scalp - treated with topical clobetasol ointment and prednisone 40 mg/week with no result.

His medical history consists of early-onset asthma treated with budesonide and formoterol and allergies (Dermatophagoides farinae, canine epidermis, trees, grasses, grain, egg white, duck meat, pork) managed with levocetirizine 5 mg/day.

Figure 1 The 65-year-old female with scalp hair loss after two years of upadacitinib treatment.

The patient was treated with cyclosporine 250 mg/day and prednisone 40 mg/day, resulting in a good response - visible hair regrowth across the entire scalp. In dermatoscopic examination, there are some exclamation hairs, follicular hair regrowth, and black dots. After five months, the patient reported deterioration, including loss of hair in the underarm and eyebrow areas. The decision to discontinue the cyclosporine and include upadacitinib was made.

Treatment with upadacitinib 15 mg/day has brought satisfactory results. After three months, follicular hair regrowth was present throughout the scalp, and short, light eyelashes and light hair regrowth in the chin area were visible as it's visible in Figure 4.

The patient remains under the care of the Dermatology Outpatient Clinic, where treatment is ongoing. Six months after treatment initiation, vellus hair regrowth on the scalp was observed, along with increased eyelash visibility and light hair regrowth in the beard area. The patient reported no complaints throughout the treatment period. At the one-year follow-up, regrowth of dark hair on the scalp was noted. Treatment was completed after 18 months of upadacitinib therapy. Two months after treatment completion, normal hair growth was observed, with no residual areas of alopecia. The patient reports an improvement in well-being and self-esteem.

Discussion

Despite multidisciplinary efforts, the etiology of AA is not fully understood in patients, and its course is unpredictable Some evidence suggests that environmental, immunological, and genetic factors may contribute to the development of the disease or exacerbate its symptoms.

The first case presents concomitant AA with sarcoidosis. Sarcoidosis is an autoimmune, multisystem inflammatory disease characterized by the formation of small clusters of inflammatory cells. Described as the "mimic" of many other pathological processes due to the variety of clinical presentations, it can affect any part of the body. Depending on the location, granulomas

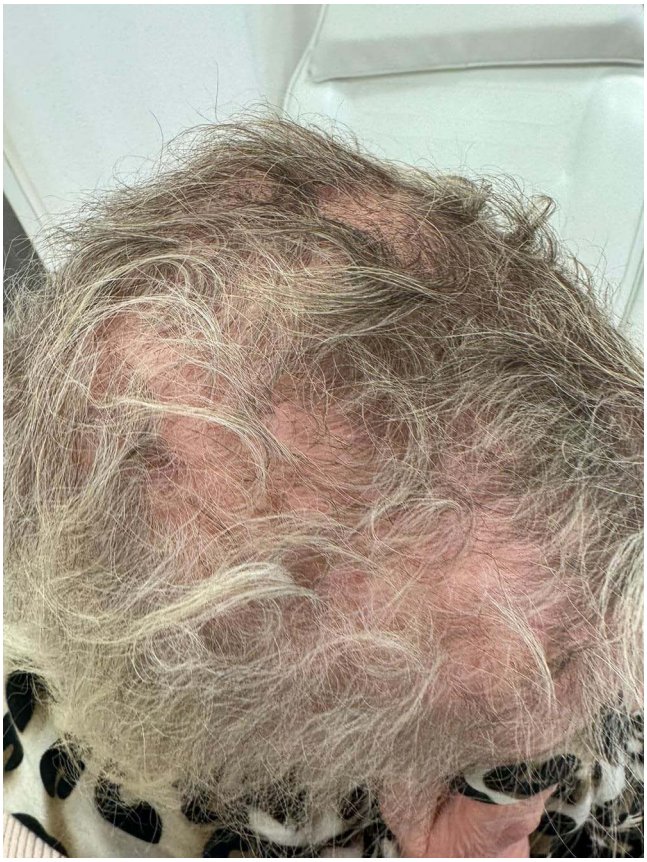

Figure 2 The 65-year-old female with scalp hair loss after two years of upadacitinib treatment. Close-up view.

can cause mild or severe symptoms or no clinical symptoms at all.[11] Lungs, along with the rest of the pulmonary system, are most frequently occupied. The second most common manifestation is cutaneous sarcoidosis. Skin lesions can present in various forms and locations on the body. Clinical presentations may include lupus pernio, nodular sarcoidosis, papular sarcoidosis, plaque sarcoidosis, scar sarcoidosis, or erythema nodosum.[12] In some cases, sarcoidosis may manifest on the scalp as both scarring and non-scarring alopecia. It manifests as diffuse or localized, structureless, orange-yellowish areas. Some focussed linear or branching vessels may also be visible. Whitish lines, follicular plugs, dilated follicles, pigmentation structures, and yellow and/or white scales can also be found.[13] Dermatoscopy is not necessarily considered a reliable tool for differentiating such diseases; consequently, histological assessment is needed. The complex interaction of immunological factors in these diseases, as well as identified cases of their co-occurrence, underscore the need for a multifaceted approach to understanding them and tailoring appropriate treatment.[14]

While sarcoidosis and AA are distinct conditions, their shared autoimmune characteristics and inflammatory mechanisms suggest a potential for co-occurrence in some individuals. Countless reports confirm that sarcoidosis can mimic various other alopecias, including acne keloidalis nuchae, frontal fibrosing alopecia, lichen planopilaris, and AA. In the case of our patient, sarcoidosis not only mimics but also occurs in conjunction with primary AA.[15]

This highlights the role of histopathological studies in diagnosing and differentiating cutaneous sarcoidosis and various types of alopecias. Recognizing and managing both conditions may require a tailored approach to immunosuppressive therapy, along with ongoing monitoring for the development of additional autoimmune symptoms.

A therapy for alopecia that has gained recognition in recent years contains JAK inhibitors. Two of them, baricitinib (JAK ½ inhibitor) for adults and ritlecitinib (JAK 3/TEC inhibitor) for individuals aged 12 or older, have been EMA-approved for treating severe AA. They forever changed the treatment, the chance of recovery, and disease management.[16,17] Although only two drugs are approved, many more are being tested, showing encouraging potential in the treatment of AA. Most of the patients experienced visible hair regrowth.[18]

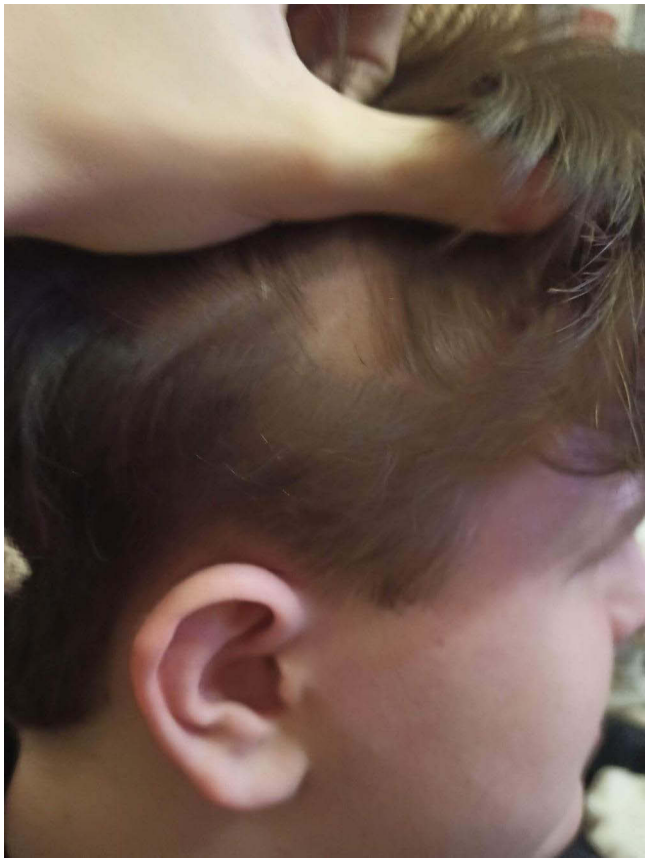

Figure 3 The first outbreak of alopecia in a 17-year-old patient.

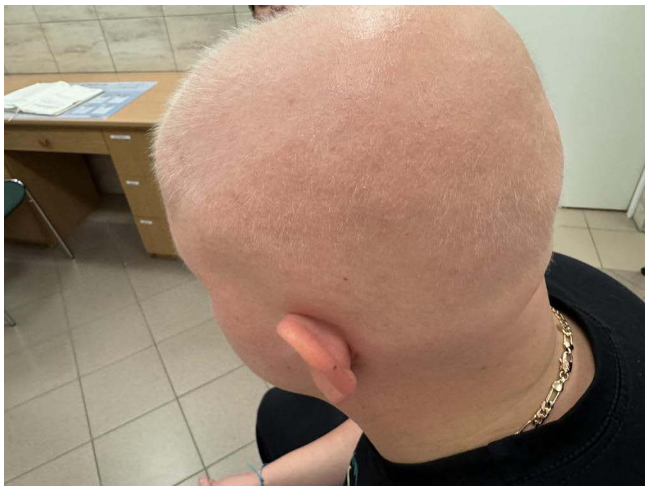

Figure 4 A 17-year-old patient with loss of all hair on the scalp after 4 months of upadacitinib treatment.

JAK inhibitors are gaining increasing recognition in the treatment of dermatological diseases, such as AA, systemic lupus erythematosus, sarcoidosis, and acquired vitiligo.[19]

JAK inhibitors appear to have great therapeutic potential. Research has demonstrated their safety and effectiveness in treating sarcoidosis, especially in patients presenting with cutaneous manifestations of the disease.[20,21] One of the drugs that has shown promise, with more and more evidence of its efficacy, is upadacitinib. Primarily used in the treatment of inflammatory bowel disease, rheumatoid arthritis, or atopic dermatitis, it has shown efficacy in treating both sarcoidosis and AA.[22–25]

Other examples of therapeutic success with upadacitinib include our patient, who has both sarcoidosis and AA. The treatment has not only led to significant hair regrowth but also improved the patient's mental health. Hair loss can have a profound psychological impact, contributing to anxiety, depression, low self-confidence, and social withdrawal.[26] Our patient experienced similar symptoms, prompting a psychiatric consultation. With ongoing treatment and visible results, the patient's mental well-being continues to improve.

The other patient also presented with AA but with a completely different set of circumstances. His case shows the connection of AA with allergic conditions like allergies (for our patient, canine epidermis, trees, grasses, grain, egg whites, duck meat and pork allergies) and asthma. Asthma is a disease characterized by airway inflammation, presenting with a history of dyspnea, shortness of breath, and cough, which vary in duration and intensity.[27] The cause of asthma remains unclear, but it is known to be influenced by a combination of genetic and environmental factors.[28] A recent study shows that the genetic factor of asthma plays a significant role in increasing the likelihood of other diseases. Amongst the diseases that can be linked to asthma, we can distinguish AA.[6,29]

Therapeutic options for AA, previously limited mainly to corticosteroids, sensitizers, and immunosuppressive agents, have recently expanded to include innovative treatments. Among these, JAK kinase inhibitors—such as upadacitinib—appear to be the most promising.[1] Treatment yields satisfactory results in patients with comorbidities without exacerbating the disease cause, as demonstrated in our patients.

The study has potential limitations. The sample size used in this study containing two people's cases, might not be sufficient for statistical measurements. Moreover the time frame of the study is short. It gives the information about results over the span of approximately one year after the treatment introduction. For more insight studies throughout few years should be performed. Even though those two cases give some insight into the matter and gives doctors a potential new way of treatment in cases of AA.

Conclusions

Treatment with upadacitinib for AA brings therapeutic success in patients with comorbidities reflected in both the physical and psychological state of the patient.

Sarcoidosis and AA are two different diseases that may present similar dermatologic symptoms. During the diagnostic process, it is essential to consider the possible similarities and adjust the diagnosis accordingly to ensure a correct diagnosis and, consequently, effective treatment.

The same thing goes for asthma and its connection with AA. Due to the increased likelihood of AA, patients with asthma should be carefully monitored and alerted to the possibility of symptoms.

Abbreviations

AA, alopecia areata.

Consent for Publication

Written informed consent for publication of history and photographs was obtained from the patient for Case 1 and from the legal guardian for Case 2. Institutional approval was not required to publish the case details.

Disclosure

The authors report no conflicts of interest in this work.

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