DOI: 10.7860/JCDR/2026/78279.23126

Case Series

Dermatology Section

Atypical Morphological Patterns of Alopecia Areata with Trichoscopic and Histopathologic Correlation: A Case Series

SAMUEL JEYARAJ DANIEL[1], SARANYA SELVAM[2], BALAJI GANESH JAYARAMAN[3], ROSEMIN JOSE MELETH[4]

ABSTRACT

Alopecia Areata (AA) is an autoimmune disease that affects the hair follicles, clinically characterised by patchy areas of hair loss, commonly affecting the scalp but also involving any hair-bearing area of the body. Various morphological patterns are described till date, the most common being patchy hair loss. Atypical patterns like annular and rectangular AA are also reported. This case series includes three morphological patterns of AA, which differ from classic forms, namely the central sparing pattern characterised by preserved islands of terminal hair within areas of patchy hair loss, the paw pattern in which multiple patches of hair loss cluster together to resemble the shape of a paw, and the punctate pattern marked by numerous small punctate areas of hair loss. Diagnosis of AA was confirmed by trichoscopic and histopathologic features. Awareness of such patterns is essential for dermatologists to aid in proper management. Hereby, the author reports three unusual morphological patterns of AA previously not documented in the literature.

Keywords: Coudability sign, Histopathology, Non-scarring alopecia, Peribulbar infiltrates, Trichoscopy

INTRODUCTION

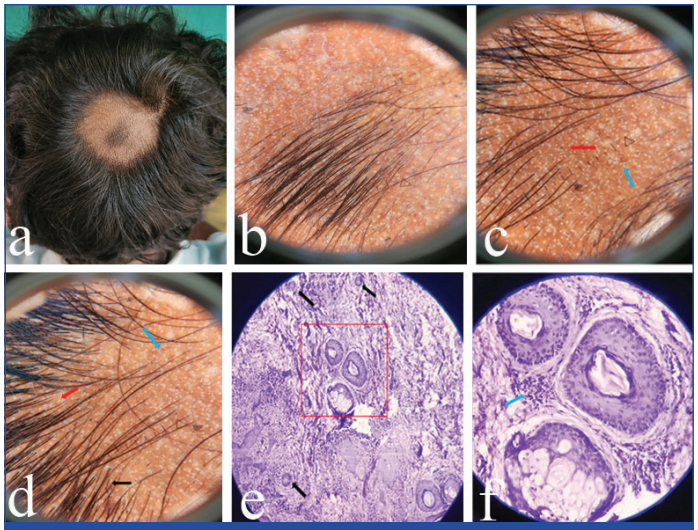
The AA is a common form of non-scarring hair loss that significantly impairs quality of life and has an unpredictable prognosis. It is one of the most common forms of hair loss seen by dermatologists and accounts for 25% of all alopecia cases. It was first described by Cornelius Celsus, and the term AA was coined by Sauvages in 1760. Multiple factors, including environmental influences, autoimmune processes and genetic susceptibility, play an important role in the development of the disease [1]. Recent research has further emphasised the correlation between vitamin D levels and the occurrence of this disease [2]. It has been associated with various systemic conditions such as atopic dermatitis, thyroid disorders, diabetes mellitus, hypertension, systemic lupus erythematosus, anaemia and even malignancies such as acute lymphoblastic leukaemia and Hodgkin's lymphoma. Basic pathology in AA is early anagen arrest coupled with an increase in telogen hair follicles. The disorder presents as smooth well-demarcated, round patches of hair loss without atrophy. Even though patchy hair loss over the scalp is the common manifestation of the disease, it can also affect other parts of the body, such as the eyelashes, eyebrows, beard, armpits, pubis, trunk, and extremities. The patches in AA can take on various forms, including reticular, ophiasis, sisaipho, alopecia totalis, alopecia universalis, linear and can be single or multiple areas of patchy hair loss, of which the latter is most frequently encountered [3]. Other cutaneous features include nail changes like fine pitting, trachyonychia, Beau's lines, onychorrhexis, nail thinning or thickening, onychomadesis, punctate or transverse leukonychia, red lunula, and koilonychia. The presence of nail involvement is linked to a poorer prognosis of the disease. Trichoscopic analysis of the scalp is an important tool to aid diagnosis and helps to differentiate AA from other causes of hair loss. Common trichoscopic findings includes black dots, broken hairs, exclamation mark hairs, vellus hairs, yellow dots and differ in various stages of activity and severity of the disease [4]. Characteristic histopathology reveals increased vellus hairs with intense peribulbar lymphocytic infiltrate. This case series presents five cases of three distinct atypical morphological patterns of AA, along with their trichoscopic and histopathologic features previously not documented in the literature.

Case 1

A 32-year-old male presented with a complaint of asymptomatic scalp hair loss persisting for four weeks. The alopecia began as a small patch and gradually enlarged over time. There was no prior treatment history, and family history was unremarkable. Dermatological examination revealed a well-defined, circular patch of hair loss located just below the vertex, measuring approximately 5 cm in diameter, with a central area of preserved normal hair (central sparing pattern) [Table/Fig-1a]. No additional areas of hair loss were noted. Nail examination was normal, and systemic evaluation showed no abnormalities.

Fungal scraping tested negative for hyphae on Potassium Hydroxide (KOH) mount. Haematological investigations showed haemoglobin at 12 g/dL, total leukocyte count of 8500 cells/mm^3, Erythrocyte Sedimentation Rate (ESR) of 20 mm in the first hour, a normal peripheral smear, and serum ferritin level of 150 ng/mL. Random blood glucose was 98 mg/dL. Thyroid function tests were within normal limits. Liver function tests revealed normal enzyme levels and total bilirubin {Serum Glutamate Oxaloacetate Transaminase (SGOT) 13 U/L, Serum Glutamate Pyruvate Transaminase (SGPT) 20 U/L, total bilirubin 1.0 mg/dL}. Renal function tests showed urea at 24 mg/dL and creatinine at 1.2 mg/dL. Vitamin D and Vitamin B12 levels were 45 ng/mL and 500 pg/mL, respectively.

Trichoscopic evaluation of the AA lesion demonstrated black dots, broken hairs, exclamation mark hairs, vellus hairs, and the characteristic coudability sign. Trichoscopy of the central hair island revealed normal terminal hair follicles [Table/Fig-1b-d]. Histopathological analysis from the lesion's border showed catagen hairs surrounded by lymphocytic infiltration and vellus hairs [Table/Fig-1e,f]. Based on trichoscopic and histopathological findings, a clinical diagnosis of AA was established. The patient was initiated on topical 0.1% betamethasone ointment applied once daily but was subsequently lost to follow-up.



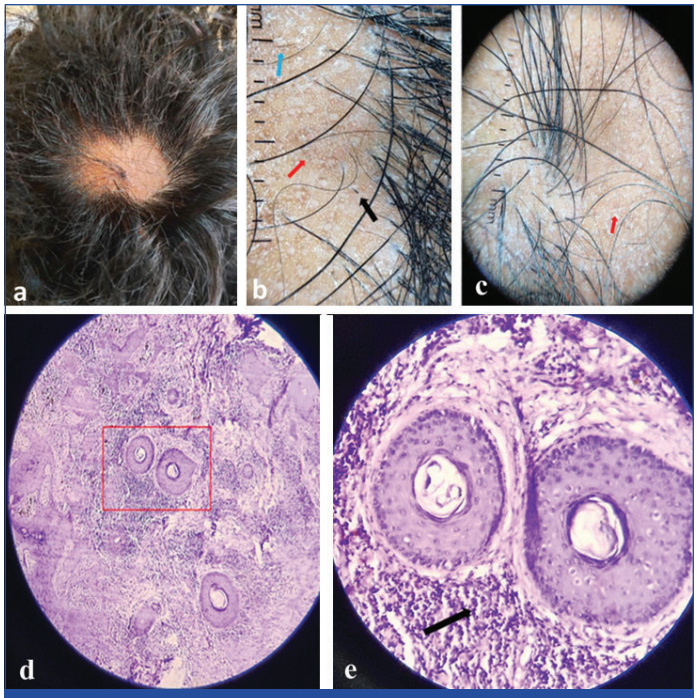
[Table/Fig-1]: a) Patchy hair loss with central island of hair; b) Trichoscopy of central island of hair showing normal terminal hair follicles Heine delta 30: 10x magnification: Polarised mode; c) Trichoscopy: Black dots (Red arrow), Broken hairs (Blue arrow), Heine delta 30: 10x magnification: Polarised mode; d) Trichoscopy: Coudability sign (Blue arrow), Exclamation hair (Black arrow), Vellus hair (Red arrow), Heine delta 30: 10x magnification: Polarised mode; e) Histopathology: Horizontal section under low power showing catagen hairs (Red box), increased vellus hairs (Black arrow) surrounded by lymphocytic infiltrate; e) Histopathology: High power showing lymphocytic infiltrate around catagen hairs (Blue arrow).

Case 2

A 17-year-old boy presented with chief complaints of a localised area of hair loss over scalp for three weeks. Hair loss started as a small patch over the vertex and gradually increased in size with sparing of small island of hair at centre. There were no additional areas of patchy hair loss. The patient had no significant past medical history or family history of similar lesions. Dermatological examination revealed a well-circumscribed area of patchy hair loss over the vertex of size 5×4 cm with an island of normal hair preserved at centre, similar to the previous case (Central sparing pattern) [Table/Fig-2a]. Examination of skin and nails was normal. Systemic examinations were found to be normal. Scraping from the patch did not show any fungus on KOH mount. Complete haematological profile revealed Haemoglobin (Hb) 12.7 mg/dL, Total Cholesterol (TC) was 5600 cells/mm^3 and ESR was 19 mm in first hour. Serum ferritin was 200 ng/mL. Peripheral smear depicted normocytic normochromic erythrocytes. Random blood sugar was 108 mg/dL. Thyroid function test showed patient was euthyroid. Liver enzymes showed SGOT 20 U/L and SGPT 36 U/L and total bilirubin was normal (0.9 mg/dL). Renal parameters revealed urea 25 mg/dL and creatinine 1.1 mg/dL. Vitamin D and Vitamin B12 levels were normal (38 ng/mL and 600 pg/mL). On trichoscopic examination black dots, broken hair, and exclamation hair were noted [Table/Fig-2b]. Trichoscopy of central island of hair showed normal terminal hair follicles and vellus hairs [Table/Fig-2c]. Histopathology from the patch showed catagen and anagen hairs with perifollicular lymphocytic infiltrate [Table/Fig-2d,e]. Diagnosis of AA was confirmed by trichoscopic and histopathologic features. Patient was started on 0.1% Tacrolimus ointment once daily application and is currently under follow-up. Regrowth of vellus hairs was noted over the patches three week after initiation of treatment.

Case 3

A 15-year-old girl presented with chief complaints of multiple patchy hair loss over the scalp for the past five weeks. The hair loss initially started as a single patch and gradually expanded. Past medical history was unremarkable. Family history was also not contributory. On dermatological examination, there was well defined area of patchy hair loss over vertex of size 8×5 cm surrounded by multiple daughter patches of size ranging between 2×2 cm to 3×2 cm extending peripherally, which closely resembled a paw pattern [Table/Fig-3a]. There was sparing of white hairs within the patch [Table/Fig-3a]. There



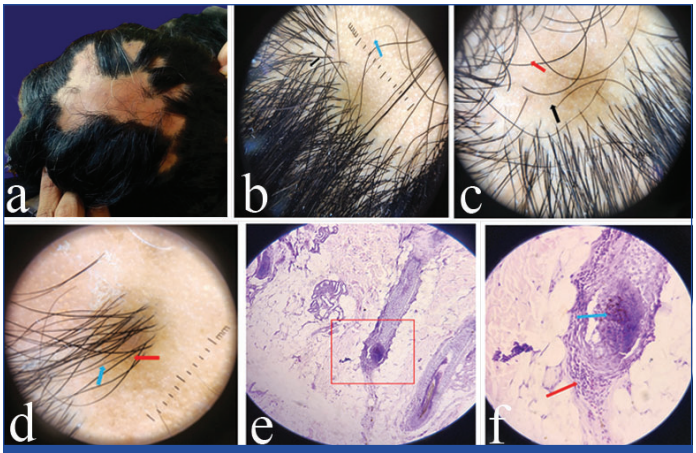
[Table/Fig-2]: Patchy hair loss with central island of hair; b) Trichoscopy: Black dots (Red arrow), broken hair (Blue arrow), exclamation hair (Black arrow), Heine delta 30: 10x magnification: Polarised mode; c) Trichoscopy of the central island of hair showed normal terminal hair follicle and Vellus hair (Red arrows), Heine delta 30: 10x magnification: Polarised mode; e) Histopathology: Horizontal section under low power showing Catagen and Anagen hairs (Red box) surrounded by lymphocytic infiltrate; e) Histopathology: High power showing dense lymphocytic infiltrate (Black arrow).

was no sign of erythema or atrophy within the patches. Premature canities were noted in the regions of normal hair. Examination of skin and nails were normal. No abnormality was noted in systemic examination. Complete haemogram revealed Hb 13.1 g/dL, TC 7800 cells/mm^3 and ESR was 7 mm in the first hour. Peripheral smear showed normocytic normochromic Red Blood Cells (RBCs). Serum ferritin was 250 ng/mL and random blood sugar was 278 mg/dL. Thyroid function test revealed patient was euthyroid. Liver function test showed normal liver enzymes and total bilirubin (SGOT 23 U/L, SGPT 30 U/L, total bilirubin 0.8 mg/dL). Renal parameters revealed urea 23 mg/dL and creatinine 0.9 mg/dL. Vitamin D and Vitamin B12 levels were 33 ng/mL and 450 pg/mL, respectively. Trichoscopic examination of patches showed black dots, broken hairs and vellus hairs [Table/Fig-3b,c]. Characteristic coudability sign was elicited [Table/Fig-3d]. Histopathology showed characteristic peribulbar and intrabulbar lymphocytic infiltrate [Table/Fig-3e,f]. The diagnosis of AA was confirmed based on trichoscopic and histopathologic features. The patient was prescribed topical 0.1% Betamethasone ointment once daily, along with Tab Dexamethasone 5 mg twice weekly, and was eventually lost to follow-up.

Case 4

A 15-year-old female presented with complaints of multiple areas of patchy hair loss over the scalp for the past eight weeks. Upon detailed history evaluation, the patient reported that the hair loss initially began as a single patch over the anterior aspect of the scalp, followed by the development of additional patches. She had not received any treatment prior to presentation, and her past medical history was non-contributory. There was a family history of allergic rhinitis and diabetes mellitus.

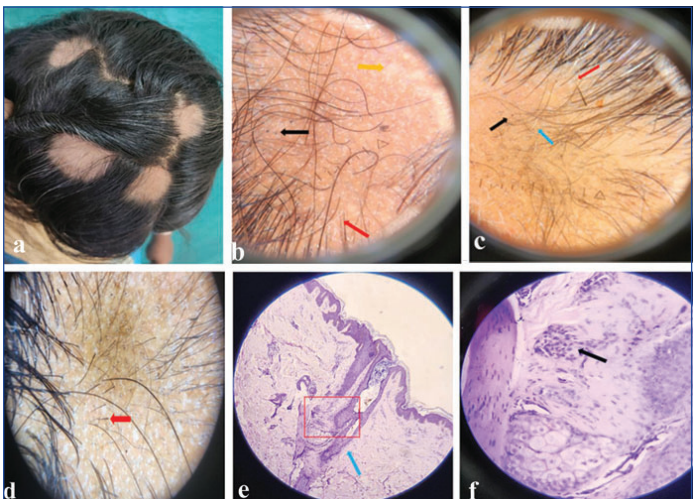
Dermatological examination revealed multiple closely arranged circular patches of hair loss, each approximately 3.5 cm in diameter, distributed over the bilateral parietal, posterior frontal, and high occipital regions of the scalp, resembling the previously described “paw pattern” [Table/Fig-4a]. Sparing of white hairs was observed within the patches, and premature canities were noted in unaffected areas of the scalp. General and systemic examinations were unremarkable.



[Table/Fig-3]: a) Central area of patchy hair loss surrounded by daughter patches (Paw pattern); b) Trichoscopy: Broken hair (Blue arrow), black dots (Red arrow), Heine delta 30: 10x magnification: Polarised mode; c) Trichoscopy: vellus hairs (red arrow), broken hairs (black arrow), Heine delta 30: 10x magnification: polarised mode; d) Trichoscopy: Coudability sign (Red arrow), Broken hair (Blue arrow), Heine delta 30: 10x magnification: Polarised mode; e) Histopathology: Vertical section under low power showing peribulbar lymphocytic infiltrate (Red box); f) Histopathology: High power showing peribulbar and intrabulbar lymphocytic infiltrate (Red arrow) and pigment cast (Blue arrow).

Complete blood count indicated mild anaemia with haemoglobin at 10.6 g/dL. Serum ferritin was 102 ng/mL, total leukocyte count was 8500 cells/mm^3, and ESR was 12 mm in the first hour. Peripheral smear revealed microcytic normochromic red blood cells. Thyroid function was within normal limits. Liver function tests showed SGOT at 15 IU/L, SGPT at 25 IU/L, and total bilirubin at 0.8 mg/dL. Random blood glucose was 112 mg/dL. Renal function parameters included urea at 22 mg/dL and creatinine at 0.9 mg/dL. Vitamin D and Vitamin B12 levels were 39 ng/mL and 350 pg/mL, respectively.

Trichoscopic examination of the patches revealed black dots, broken hairs, yellow dots, vellus hairs, exclamation mark hairs, and a positive coudability sign [Table/Fig-4b-d]. Histopathological analysis from the affected area showed catagen hairs, fibrous streamers, and lymphocytic infiltration [Table/Fig-4e,f]. The clinical diagnosis of AA was confirmed based on trichoscopic and histopathological findings.

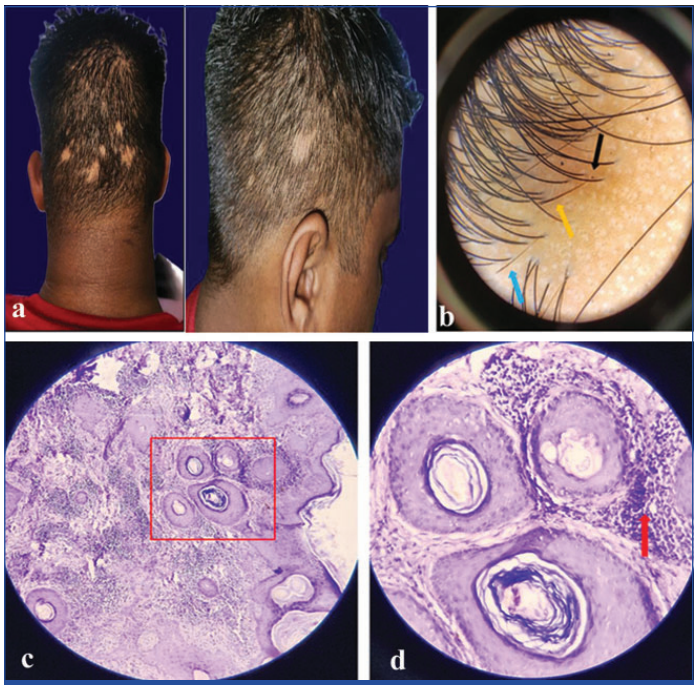


[Table/Fig-4]: a) Paw pattern of Alopecia Areata (AA); b) Trichoscopy: Yellow dots (Yellow arrow), Black dots (Black arrow), Vellus hairs (Red arrow), Heine delta 30: 10x magnification: Polarised mode; c) Trichoscopy: Black dots (Black arrow), Broken hairs (Blue arrow), Coudability sign (Red arrow), Heine delta 30: 10x magnification: Polarised mode; d) Trichoscopy: Exclamation hair (Red arrow); e) Histopathology: Vertical section under low power showing Catagen hair (Blue arrow) and fibrous streamer (Red box); f) Histopathology: High power showing lymphocytic infiltrate within the fibrous streamer (Black arrow).

The patient was initiated on topical 0.1% betamethasone ointment applied once daily, along with oral dexamethasone 5 mg twice weekly and ferrous sulfate with folic acid supplements. She is currently under follow-up. Regrowth of vellus hairs over the affected patches was observed four weeks after the commencement of treatment.

Case 5

A 34-year-old male presented to dermatology outpatient clinic with complaints of multiple areas of patchy hair loss over scalp for past four weeks. No history of past treatment for the same. Past history was not significant and there was no history of similar hair loss among family members. On dermatological examination, there were multiple punctate areas of hair loss over the occipital and temporal region ranging in size between 0.5×1 cm to 1×1.5 cm [Table/Fig-5a]. The patches had no evidence of scaling or inflammation. There was no specific nail or skin changes. Since the morphology was similar to moth eaten pattern of alopecia in secondary syphilis a Venereal Disease Research Laboratory (VDRL) test was done and was found to be non-reactive. Complete haemogram revealed Hb 12.1 g/dL, TC 8700 cells/mm^3 and ESR was 17 mm in the first hour. Peripheral smear showed normocytic normochromic RBCs. Random blood sugar was 78 mg/dL. Liver function tests showed normal liver enzymes and total bilirubin (SGOT 33 U/L,SGPT 25 U/L, total bilirubin 0.7 mg/dL). Renal parameters revealed urea 9 mg/dL and creatinine 0.7 mg/dL. Vitamin D levels and Vitamin B12 were normal (35 ng/mL and 450 pg/mL). Trichoscopy revealed multiple black dots, exclamation hairs and broken hairs [Table/Fig-5b]. Histopathology confirmed the diagnosis of AA which showed follicular unit with anagen and catagen hair surrounded by lymphocytic infiltrate [Table/Fig-5c,d]. The patient was prescribed topical 0.1% Betamethasone ointment for once daily application; however, was subsequently lost to follow-up.



[Table/Fig-5]: a) Multiple punctate areas of patchy hair loss; b) Trichoscopy: Exclamation hair (Yellow arrow), Black dots (Black arrow), Broken hairs (Blue arrow), Heine delta 30: 10x magnification: Polarised mode; c) Histopathology: Horizontal section in low power showing follicular unit with anagen hairs, catagen hairs surrounded by lymphocytic infiltrate (Red box); d) Histopathology: High power showing dense lymphocytic infiltrate (Red arrow).

Clinical, trichoscopic and histopathologic features of all five cases have been described in [Table/Fig-6].

	Case 1	Case 2	Case 3	Case 4	Case 5
Clinical Features					
Age	32	17	15	15	34
Sex	M	M	F	F	M
Duration	4 weeks	3 weeks	5 weeks	8 weeks	4 weeks
Pattern of AA	Central sparing	Central sparing	Paw	Paw	Punctate
Trichoscopy					

Black dots	+	+	+	+	+
Broken hair	+	+	+	+	+
Vellus hair	+	+	+	+	-
Exclamation hair	+	+	-	+	+
Coudability sign	+	-	+	+	-
Yellow dots	-	-	-	+	-
Histopathology					
Perifollicular lymphocytic infiltrate	+	+	+	+	+
Catagen hairs	+	+	-	+	+
Vellus hairs	+	+	-	-	+

[Table/Fig-6]: Clinical, trichoscopic and histopathologic features of cases.

DISCUSSION

The AA is an organ specific autoimmune inflammatory disease targeting the hair follicle mediated by either CD4+ or CD8+ T cells [5]. A multicentre retrospective study reports that it represents 18.2% of all cases of non-scarring alopecia [6]. A systematic review of the epidemiology of AA reported a comparable global lifetime incidence of approximately 2% [7]. The disorder can affect any age group but median age is around 33 years, while the condition affects males and females equally, certain studies have reported a predominance among males.

The key pathogenic mechanism involved in this inflammation is loss of hair follicle immune privilege. There is a complex interplay of various genetic, environmental and immunological factors in the development of disease which ultimately results in loss of hair follicle immune privilege. Genome-wide association studies have identified several single-nucleotide polymorphisms associated with AA, with Human Leukocyte Antigen (HLA-DR) region on chromosome 6 emerging as the strongest genetic risk factor. Additional genetic contributors include genes related to natural killer cell receptor ligands, the Janus Kinase (JAK) signalling pathway, regulatory T cells, autophagy, and apoptosis [8].

Environmental factors commonly related to disease include stress, infections, vaccinations, and diet.

Literature suggests that increased levels of adrenocorticotropic hormone, corticosterone, and estradiol were associated with higher concentrations of pro-inflammatory cytokines in the skin, indicating that psychological and physiological stressors may contribute to the development of the disease. Under normal conditions, hair follicles possess a zone of immune privilege characterised by reduced expression of Major Histocomapatibility Class (MHC) class I and β2-microglobulin, secretion of immunosuppressive molecules such as α-melanocyte-stimulating hormone and transforming growth factor-β, and diminished activity of antigen-presenting cells. It is proposed that in AA this immune privilege collapses, potentially due to an unidentified autoantigen [8]. These autoantigens can be the consequence of molecular mimicry induced by infections and vaccinations leading to activation of immune system. Subsequently, Interferon-γ (IFN-γ) and Interleukin-2 (IL-2) can trigger the infiltration of CD8+, CD4+, and other inflammatory cells into the previously protected area, leading to inflammation of the hair follicle and possible hair loss. Nutritional deficiencies of iron and vitamin D have also been linked to disruption of immune system and autoimmune attack on hair follicles.

The AA is clinically manifested by sudden onset of patchy hair loss over hair bearing areas most commonly scalp. Commonly described morphological patterns documented in literature includes patchy type which can be single or multiple, AA totalis, AA universalis, linear and perinevoid type. Rare morphological variants, which are associated with poor prognosis and progression to alopecia universalis includes reticular, ophiasis and sisaipho pattern.

The present case series includes three morphologically distinct patterns of AA namely central sparing pattern, paw pattern and punctate pattern. The central sparing pattern is characterised by preserved islands of terminal hair within regions of patchy hair loss. In the paw pattern, several patches of hair loss cluster together, resembling the shape of a paw. The punctate pattern is defined by the presence of numerous small, punctate areas of hair loss. Atypical morphological patterns like rectangular and annular variants were previously documented by Shin J et al., and Bansal M et al., [9,10]. The emergence of these atypical forms of AA is likely due to a complex interplay of several underlying factors, such as genetic susceptibility, immune system abnormalities, environmental influences, hormonal changes, and psychological stress. This multifaceted interaction may explain the diverse clinical manifestations observed in the disease.

Differential diagnosis for similar pattern of AA includes cicatricial alopecia like Pseudopelade of Brocq, Central Centrifugal Cicatricial Alopecia (CCCA), tufted folliculitis, Lichen Planopilaris (LPP) and syphilitic alopecia. Central sparing pattern of AA has to be differentiated from scarring alopecias with similar morphology like CCCA and tufted folliculitis. The latter is characterised by patchy hair loss with tufts of hair, perifollicular erythema and follicular pustules which was absent in the first two cases [11]. CCCA starts as patchy hair loss most commonly at vertex and spreads centrifugally similar to (case 1 and 2). Clinically, it can be differentiated from central sparing pattern of AA by the presence of additional characteristics such as perifollicular hyperpigmentation and erythema within the areas of hair loss, which were not observed in case 1 and 2. Absence of follicular ostia on trichoscopic examination serves as a specific marker for confirming CCCA as suggested by Gabros S et al., which was also not seen in our cases of central sparing pattern of AA [12]. Paw pattern of AA morphologically resembled the foot prints in snow appearance seen in pseudopelade of brocq, a type of cicatricial alopecia characterised by smooth irregular atrophic patches of hairloss [13]. The characteristic trichoscopic and histopathologic findings noted in our cases 3 & 4 help differentiate the paw pattern of AA from pseudopelade of Brocq. Moth-eaten alopecia observed in secondary syphilis closely resembled the punctate pattern of AA, but negative serology, trichoscopic and histopathologic features seen in our case 5 further supplement the diagnosis of AA [14]. Multiple irregular patches of hair loss morphologically similar to a punctate pattern can also be seen in early stages of LPP, but associated perifollicular erythema and scaling in LPP was not noted in our case hence differentiating it [15].

The primary objective in managing AA is to mitigate inflammation around the hair follicles and facilitate hair regrowth. First-line treatments typically involve topical and intralesional corticosteroids, with systemic corticosteroids reserved for more severe or progressive cases. Topical corticosteroids inhibit activity of T lymphocytes and suppress release of inflammatory mediators thereby reducing local inflammation around hair follicle. Adjunctive topical therapies include calcineurin inhibitors, vitamin D analogues, and minoxidil. For patients unresponsive to these conventional therapies, alternative options encompass oral minoxidil, immunosuppressants such as methotrexate, azathioprine, and cyclosporine, as well as procedural treatments like platelet-rich plasma, topical anthralin, excimer laser therapy, and PUVA photochemotherapy and immunotherapy. Oral JAK inhibitors such as tofacitinib are emerging as promising therapeutic options. The choice of treatment is guided by the severity of the disease, individual patient characteristics, and previous therapeutic responses [16].

CONCLUSION(S)

This case series highlights atypical morphological variants of AA previously not documented in the literature so far, hence it is mandatory to consider these atypical variants of AA, which can

simulate hair loss patterns seen in cicatricial alopecia. It further emphasises the role of trichoscopy as a crucial tool for confirming clinical diagnosis of AA and differentiating it from cicatricial alopecias, thereby decreasing the requirement for biopsy.

REFERENCES

[1] Seetharam KA. Alopecia areata: An update. Indian J Dermatol Venereol Leprol. 2013;79:563-75.
[2] Tran PT, Chen A, Yi L, Goh C. Vitamin D levels in alopecia areata and other alopecias: A retrospective case- control study at a single institution. Int J Trichology. 2022;14(5):175-77.
[3] Madani S, Shapiro J. Alopecia areata update. J Am Acad Dermatol. 2000;42(4):549-66.
[4] Jain N, Doshi B, Khopkar U. Trichoscopy in alopecias: Diagnosis simplified. Int J Trichology. 2013;5:170-78.
[5] Alkhalifah A, Alsantali A, Wang E, McElwee KJ, Shapiro J. Alopecia areata update: Part I. Clinical picture, histopathology, and pathogenesis. J Am Acad Dermatol. 2010;62:177-88, quiz 189-90.
[6] Vañó-Galván S, Saceda-Corralo D, Blume-Peytavi U, Cucchía J, Dlova NC, Gavazzoni Dias MFR, et al. Frequency of the types of alopecia at twenty-two specialist hair clinics: A multicenter study. Skin Appendage Disord. 2019;5(5):309-15.
[7] Villasante Fricke AC, Miteva M. Epidemiology and burden of alopecia areata: A systematic review Clin Cosmet Investig Dermatol. 2015;8:397-403.
[8] Paus R, Bertolini M. The role of hair follicle immune privilege collapse in alopecia areata: Status and perspectives. J Investig Dermatol Symp Proc. 2013;16:S25-S27.
[9] Shin J, Jang HS, Cho SB. Rectangular-patterned occipital alopecia areata: A report of three cases. Int J Trichology. 2012;4(3):164-66
[10] Bansal M, Manchanda K, Pandey S. Annular alopecia areata: Report of two cases. Int J Trichology. 2013;5(2):91-93.
[11] Annessi G. Tufted folliculitis of the scalp: A distinctive clinicohistological variant of folliculitis decalvans. Br J Dermatol. 1998;138(5):799-805.
[12] Gabros S, Sathe NC, Masood S. Central centrifugal cicatricial alopecia. [Updated 2024 Mar 13]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 Jan-.
[13] Diwan N, Gohil S, Nair PA. Primary idiopathic pseudopelade of brocq: Five case reports. Int J Trichology. 2014;6(1):27-30.
[14] Shrestha A, Kusha KC, Baral A. Moth eaten alopecia as the only manifestation of syphilis: A case from Nepal. Oxford Med Case Rep. 2024;2024(3):omae011.
[15] Lepe K, Nassereddin A, Syed HA, et al. Lichen Planopilaris. [Updated 2024 May 1]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 Jan-.
[16] Darwin E, Hirt PA, Fertig R, Doliner B, Delcanto G, Jimenez JJ. Alopecia areata: Review of epidemiology, clinical features, pathogenesis, and new treatment options. Int J Trichology. 2018;10(2):51-60.

PARTICULARS OF CONTRIBUTORS:
1. Professor and Head, Department of Dermatology, Thanjavur Medical College, Thanjavur, Tamil Nadu, India.
2. Assistant Professor, Department of Dermatology, Thanjavur Medical College, Thanjavur, Tamil Nadu, India.
3. Assistant Professor, Department of Dermatology, Thanjavur Medical College, Thanjavur, Tamil Nadu, India.
4. Junior Resident, Department of Dermatology, Thanjavur Medical College, Thanjavur, Tamil Nadu, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:
Dr. Rosemin Jose Meleth,
Ponni Hostel, Thanjavur Medical College Road, Thanjavur-613004,
Tamil Nadu, India.
E-mail: roseminjose7@gmail.com

PLAGIARISM CHECKING METHODS: [Jain H et al.]
- Plagiarism X-checker: Jan 28, 2025
- Manual Googling: Sep 25, 2025
- iThenticate Software: Sep 27, 2025 (2%)

ETYMOLOGY: Author Origin

EMENDATIONS: 7

AUTHOR DECLARATION:
- Financial or Other Competing Interests: None
- Was informed consent obtained from the subjects involved in the study? Yes
- For any images presented appropriate consent has been obtained from the subjects. Yes

Date of Submission: **Jan 25, 2025**
Date of Peer Review: **May 15, 2025**
Date of Acceptance: **Sep 29, 2025**
Date of Publishing: **Apr 01, 2026**