

Sustained Remission of Alopecia Areata Universalis following Red Cell Transfusion

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Dear Editor,

Alopecia areata (AA) is an immune mediated, non-scarring hair loss disorder. Despite recent advances in therapy with Janus kinase (JAK) inhibitors, an unmet need for additional therapies exists, particularly for more extensive disease such as alopecia areata totalis (AAT) and universalis (AAU)¹. We present a case of a patient with long-standing AAU achieving near-complete, sustained hair regrowth following leukodepleted red cell concentrate transfusion.

An 85-year-old attended our service in 2020 with a twenty-nine-year history of AA, having previously been treated with topical corticosteroids, intralesional corticosteroids, topical minoxidil, and psoralen-ultraviolet A (PUVA) phototherapy. She defaulted follow up in 2012, and was re-referred in 2020 with longstanding AAU, to see if any new treatment options were available to her. At the time of review, comorbidities included hypertension, hypercholesterolemia, rheumatic heart disease, and mitral regurgitation, with no personal history of autoimmune disease. In view of her age and high cardiovascular risk profile, she was deemed unsuitable for treatment with a JAK inhibitor¹. In 2023, she was admitted following two episodes of bleeding per rectum. CT-angiography revealed extensive sigmoid diverticulosis. She was treated for diverticular bleeding with intravenous tranexamic acid, esomeprazole and three units of leukodepleted, packed, matched O+ red cell concentrate. She was not prescribed systemic glucocorticoids during admission. Following discharge, she noted hair regrowth on her scalp within six weeks, for the first time in over 15 years. Four months post transfusion, she had complete scalp regrowth with evidence of eyebrow regrowth and patchy eyelash regrowth. One-year post-transfusion, she has maintained hair growth, with no new reported areas of alopecia. Evidence from the literature suggests a relationship between low serum ferritin levels and non-scarring alopecia's, including AA². However, she was not previously noted to be iron deficient and had ferritin stores within the normal range for her age.

A theory in AA pathogenesis postulates that due to loss of follicular immune privilege, hair follicle autoantigens are presented to autoreactive CD8+ T cells, resulting in immune attack of the follicle and shedding of the hair shaft. A clinical improvement in certain autoimmune conditions such as rheumatoid arthritis has been reported following blood transfusion³. It is hypothesised that autoreactive host T-cells are downregulated by induction of regulatory T-

cells or by the deletion of cytotoxic T-cells directed against the autologous antigens on the donor leucocytes. Her blood transfusion was leucodepleted, therefore a separate mechanism may be at play.

Transfusion-related immune modulation (TRIM) describes a transient immunomodulation observed following transfusion of allogenic blood. There are no cases in the literature linking improvement of autoimmunity and TRIM. However, many RBC mechanisms have been proposed which ultimately promote immunomodulation, including burden of the mononuclear phagocyte system increasing macrophage intracellular heme triggering proinflammatory cytokine release and bias macrophage phenotype from inflammatory (M1) toward immunosuppressive (M2) phenotype⁴. Therefore, we hypothesise, whilst not being a clinically approved therapeutic modality in AA, mechanistic inquiry into links between transfusion-derived mediators and remission of disease may assist in further elucidation of the pathogenesis of AA.

Declarations of Conflicts of Interest:

None declared.

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