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Identical alopecia areata in identical twin sisters

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Full Text

Sir,

Alopecia areata (AA) is an autoimmune dermatosis, characterized by the development of sudden onset of nonscarring patches of hair loss. As per expert consensus, lifetime incidence of AA is approximately 2%. We hereby report a unique case of AA in young monozygotic girl twins developing identical lesions, at the same site and at the same time.

Two 10-year-old monozygotic twin girls (R and L) presented with patchy hair loss, present for 2–3 months. They were born to nonconsanguineous parents. Both parents and twins were healthy. There was no personal or family history of AA, autoimmunity, or atopy. Financial constraints prevented genetic testing to confirm monozygosity. The hair loss was discovered by their mother while oiling their hair, a regular cultural practice in India. The interval of discovery between the two girls was 2 weeks. Both had almost identical alopecia with a single, asymptomatic, noninflammatory circular bald patch on the vertex [Figure 1]. The mother had applied onion juice on the patch of R that showed some regrowth. Treatment was stopped after R developed redness and irritation. L was taking an oral homeopathic medicine, without benefit. Dermoscopy showed yellow dots, black dots, broken hairs, tapering hair (exclamation marks), and short vellus hairs. Neither girl had any other hair disorder nor a history of preceding infection. Mometasone 0.1% lotion was prescribed and both twins receive periodic follow-up.{Figure 1}

The pathogenesis of AA is multifactorial, involving an inherited susceptibility and a cryptogenic environmental trigger. Inter-current viral infection has been suggested as a possible environmental trigger; however, there is no convincing evidence to support this. Cytomegalovirus (CMV) DNA has been identified in patches of AA, but this was not confirmed by *in situ* hybridization.[1]

A genome wide association study identified several candidate genes controlling activation and proliferation of regulatory T-cells, cytotoxic T lymphocyte associated antigen 4, interleukin (IL)-2 and IL-21, the Ikaros family zinc finger 4, and the human leukocyte antigen (HLA) region. Within the HLA region on chromosome 6p, a strong association was found with 2 genes located within the ULBP (CMV UL16-binding protein) gene cluster on chromosome 6q25.1. The latter is particularly interesting as the ULBP3 is expressed in lesional scalp from patients with AA in the hair follicle dermal sheath and dermal papilla during active disease. Both ULBP genes act as NKG2D-activating ligands that bind to the NKG2D receptor and activate the specific interferon–γ producing cytotoxic T lymphocytes (NKG2D+, CD8+, $CD3^+$ T cells) found in the peri-bulbar, swarm of bees infiltrate that destroy the anagen hair bulb in AA.[2]

The frequency of positive family history for AA is approximately 10%–20%.[3] Coexistence of AA in siblings including twins is reported.[4],[5],[6],[7],[8],[9] The concordance rate in monozygotic twins is estimated to be 42%–55%, compared to 10% in dizygotic twins. However, simultaneous appearance of AA in twins is unusual. Even more unusual is to have twin siblings showing the same morphology and location of AA.[10]

Our case is rare because of the development of identical lesions over the same location on the scalp (vertex), at the same point of time as a first episode. It is interesting to speculate that CMV or another viral infection might upregulate expression of the CMV binding protein genes and in turn lead to NKG2D cell activation. If true, it is perhaps surprising that concurrent development of AA in genetically identical twins is not more common. In any case, this hypothesis does not address the identical location of the lesions. AA remains an enigmatic condition, however, this case does provide an interesting and thought provoking observation.

SV was the point of primary contact for the patient and for initiating the concept for the manuscript. AD, SV and RS wrote the manuscript which was submitted by AD after editing by SV and RS.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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