Sir,

Alopecia areata (AA) is a common nonscarring alopecia whose etiology has been linked to genetic predisposition, environmental triggers, and autoimmunity. Diffuse presentation of AA is rare and seems to affect predominantly females, with a short course of acute diffuse hair loss of the scalp. We describe the case of a patient that presented diffuse AA after an infectious pneumonia, despite long-term use of oral cyclosporine, mycophenolate, and prednisone after kidney transplantation.

A 46-year-old Brazilian woman presented with acute scalp hair loss 1 month after a hospitalization due to severe sepsis for pneumonia. She denied personal or familial history of AA. Fourteen years before, she underwent kidney transplantation for hypertensive nephropathy, with the use of systemic immunosuppression including oral mycophenolate sodium, cyclosporine, and prednisone. At the time, dosages were 720, 100, and 5 mg daily, respectively.

Clinical examination showed severe and diffuse nonscarring alopecia of the scalp, some sparse thin hairs, and no involvement of eyebrows, eyelashes, and corporal hairs [Figure 1]. Trichoscopy revealed black dots, few exclamation mark hairs, and short regrowing hairs with no epidermal changes [Figure 1]. Biopsy of the parietal area revealed hair follicle miniaturization with increased vellus hairs and increased telogen hair count. Melanin deposition was present into follicular canal, follicular epithelium, and telogen terminal units [Figure 2].

Treatment with topical minoxidil 5% and clobetasol was instituted, and progressive hair regrowth was observed [Figure 1].

The diffuse presentation of AA is rare and few articles were published so far. Diffuse AA has a noticeable female predominance and is clinically characterized by severe, rapidly progressive, and diffuse scalp hair shedding with remarkably favorable prognosis. Dystrophic and exclamation mark hairs and miniaturization are present. After dramatic presentation, almost all cases are followed by clinical remission and spontaneous hair growth regardless specific treatments. Some authors have termed this particular subtype of AA as “acute diffuse and total alopecia” that we believe to be the same entity. Acute symptoms onset and good improvement after few weeks of topical treatment in our patient reaffirm the classically described clinical course.

Histology of diffuse AA is indistinguishable from the other presentations, but some authors observed higher incidence of perifollicular eosinophilic infiltration or even

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**Figure 1:** Initial presentation with diffuse nonscarring alopecia of the scalp (a). Improvement after 1 (b) and 5 months (c). Initial trichoscopy (×20) showing black dots, localized exclamation mark hair, and short regrowing hairs (d). Normal trichoscopy (×20) after 5 months of treatment (e).

**Figure 2:** Hair follicle miniaturization, increased vellus hairs, and melanin deposits into telogen terminal units (H and E, ×100)
prominent dermal and follicular pigment incontinence in this subtype. In our case, the typical feature of peribulbitis was absent, but the increase of vellus hairs and telogens and melanin deposits in follicular epithelium was consistent with AA as described by Peckman et al.[5]

Few cases of diffuse AA have been described after infections[4] and immunomodulatory drugs.[7] Most of the published cases had no triggers detected.[2] Lew reported thirty diffuse AA cases, and only five had possible triggering factors including psychological trauma, pregnancy, weight loss, and systemic illness.[3]

There is no clear explanation for the occurrence of AA despite long-term administration of immunosuppressive drugs. Few cases of AA in patients using cyclosporine[8,9] and mycophenolate[10] were reported. Some authors suggested that dosage could be insufficient to prevent T-cell activation in those patients.[8] More studies are necessary to understand the pathogenesis of AA and its correlation with autoimmunity and immunosuppressive drugs.

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Conflicts of interest

There are no conflicts of interest.

Karina Lopes Morais, Alessandra Anzai, Neusa Yuriko Sakai Valente, Ricardo Romiti

Department of Dermatology, Univesity of São Paulo Medical School, São Paulo, Brazil

Address for correspondence:
Dr. Karina Lopes Morais,
Av. Dr. Eneas de Carvalho Aguiar, 255, 3rd Floor,
Cerqueira César, São Paulo, SP 05403-900, Brazil.
E-mail: karina_llmorais@yahoo.com.br

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