

Two cases of fibrosing alopecia in a patterned distribution after coronavirus disease 2019

Sir,

The types of alopecia that have been reported to occur in patients with coronavirus disease 2019 (Covid 19) include telogen effluvium, alopecia areata and dystrophic anagen effluvium.¹⁻³ Both preexisting or underlying telogen effluvium and alopecia areata have been noted to flare in relation to the fear of infection or stress of lockdown in patients either with or without Covid 19.^{1,2} However, abrupt onset of hair shedding has also been observed 2–3 months after recovery of Covid 19 as telogen effluvium and dystrophic anagen effluvium.^{1,3} Although no evidence exists regarding a pathogenic inflammatory reaction at the level of hair follicle or direct viral infection of the hair follicle, hair loss was attributed to the multi-systemic inflammatory febrile disease.^{1,3} We were unable to find any previous reports of cicatricial alopecia including fibrosing alopecia in a pattern distribution associated with Covid 19.

A 41-year-old woman presented to our dermatology department with a ten-day-history of severe asymptomatic hair shedding from all areas of the scalp. She had Covid 19 with symptoms of mild fever, fatigue and myalgia three months prior and was treated with favipiravir (day 1: 1600 mg twice daily and days 2–5: 600 mg twice daily). The second patient was a 33-year-old man who presented with a two-week-history of severe hair loss that was more pronounced on the crown, accompanied by mild pruritus and scaling. He had recovered from Covid 19 with mild symptoms including fever, fatigue, myalgia, arthralgia, sore throat and diarrhea three months ago and was treated with oral hydroxychloroquine (400 mg/day for five days). Covid 19 was confirmed by detection of severe acute respiratory syndrome coronavirus 2 RNA by reverse transcription-polymerase chain reaction for nasopharyngeal swabs in both patients. The medical histories of the patients were otherwise unremarkable and they did not report a family history of a similar type of alopecia. Laboratory test results, including total blood cell count, serum ferritin and thyroid-stimulating hormone levels were all within normal limits. Dermatologic examination revealed diffuse hair thinning along with scaling predominantly on the vertex and frontoparietal area [Figures 1a and 1b] and the hair-pull test was strikingly

positive in both patients. Trichoscopically, hair shaft diameter variability, predominance of single hair follicles and loss of follicular ostia [Figures 2a and 2b] were noted in the two patients. Additional findings were peri- and interfollicular scales, tubular silver-white hair casts [Figure 2a], thin arborizing vessels and wavy hair in the first patient and interfollicular scales, multiple dotted, comma and elongated vessels [Figure 2b] in the second patient. A 4-mm punch biopsy specimen was obtained from the frontoparietal scalp. Histopathological examination of the transverse sections demonstrated a reduction in the number of follicles in both patients along with miniaturization of terminal hair follicles and lichenoid interface dermatitis affecting the upper portion of the terminal follicle in the first patient [Figure 3a]. In addition to the miniaturization of terminal hair follicles, basal vacuolar degeneration affecting the isthmus and infundibulum of both miniaturized and non-miniaturized follicles, perifollicular lymphocytic inflammation and fibrosis were noted in the second patient [Figure 3b]. Both patients were diagnosed with fibrosing alopecia in a pattern distribution and treated with oral hydroxychloroquine (400 mg/day), topical 5% minoxidil lotion (twice daily) and intralesional injection of triamcinolone acetonide (5 mg/ml).

Fibrosing alopecia in a pattern distribution is a form of lymphocytic primary cicatricial alopecia, first described in 2000 by Zinkernagel and Trüeb.⁴ Diffuse hair loss in a centroparietal distribution resembling androgenetic alopecia and combined trichoscopic and histopathological signs of lichen planopilaris and androgenetic alopecia are hallmarks of this alopecia type.^{4,5} The exact pathogenesis of fibrosing alopecia in a pattern distribution is unclear, but it is considered to be a T-cell-mediated autoimmune reaction associated with apoptosis of follicular epithelial cells.⁵ However, what triggers the follicular lichenoid reaction is unknown and a viral etiology has not been defined before. Fibrosing alopecia in a pattern distribution generally runs a chronic and slowly progressive course.⁵ However, sometimes, the gradual thinning of hair over several years may be followed by an accelerated period of hair loss and scalp inflammation^{4,5} which may also be the case in the present cases. It is also likely that our patients might

How to cite this article: Özcan D, Vural AT, Özen O. Two cases of fibrosing alopecia in a patterned distribution after coronavirus disease 2019. *Indian J Dermatol Venereol Leprol* 2021;87:848-50.

Received: February, 2021 **Accepted:** May, 2021 **EPub Ahead of Print:** August, 2021 **Published:** October 2021

DOI:10.25259/IJDVL_204_2021 **PMID:** 34491674

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Figure 1a: First patient. Diffuse hair thinning predominantly on the vertex and frontoparietal area



Figure 1b: Second patient. Diffuse hair thinning predominantly on the vertex and frontoparietal area

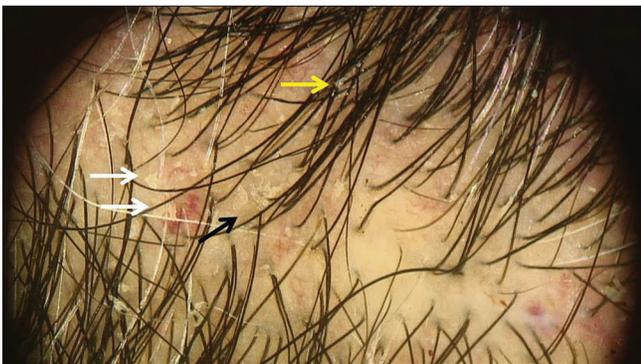


Figure 2a: First patient. Hair shaft diameter variability, predominance of single hair follicles, loss of follicular ostia, perfollicular scales (white arrows), interfollicular scales (black arrow) and tubular silver-white hair casts (yellow arrow). [Polarized light, x20, FotoFinder 2007, MediCam 800 (HD)]



Figure 2b: Second patient. Hair shaft diameter variability, predominance of single hair follicles, loss of follicular ostia, interfollicular scales (white arrows), multiple dotted, comma and elongated vessels (yellow arrows). [Polarized light, x20, FotoFinder 2007, MediCam 800 (HD)]

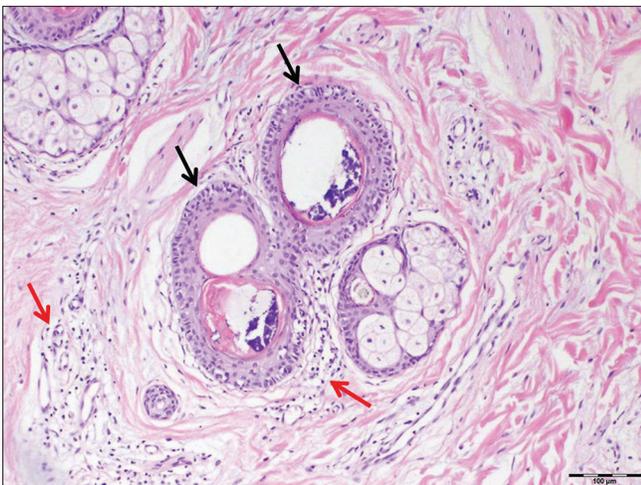


Figure 3a: First patient. Basal vacuolar degeneration (black arrows) and perifollicular lymphocytic inflammation (red arrows) affecting the upper portion of the terminal follicle in the transverse section (hematoxylin and eosin, x100)

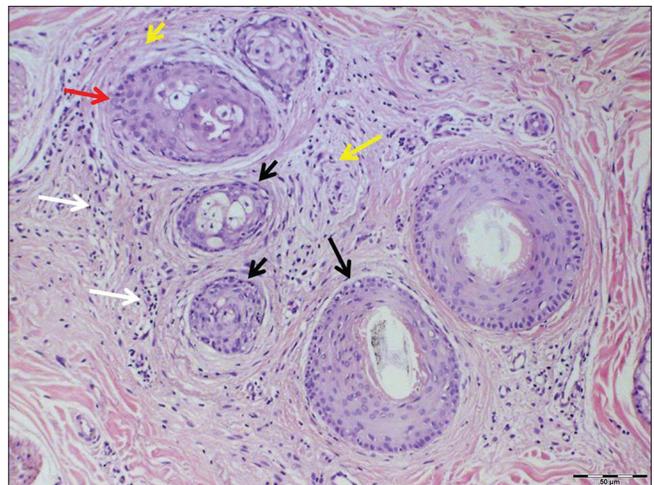


Figure 3b: Second patient. Miniaturization of terminal hair follicles, basal vacuolar degeneration affecting both miniaturized (short black arrows) and non-miniaturized (long black arrow) follicles, fibrous scar (long yellow arrow), mucinous fibroplasia (short yellow arrow), effacement of the basal layer of a miniaturized follicle due to basal vacuolar degeneration (red arrow) and minimal perifollicular lymphocytic inflammation (white arrows) in the transverse section (hematoxylin and eosin, x100)

have developed fibrosing alopecia in a pattern distribution acutely for the first time. Although it is hard to correlate the onset of fibrosing alopecia in a pattern distribution with Covid 19, we propose that in our patients, cytokine storm observed in Covid 19 might have promoted fibrosing alopecia in a pattern distribution by damaging hair follicles which in turn may have led to the expression of cytokines that initiate an inflammatory process. Furthermore, the virus itself might have acted as an antigenic stimulus on hair follicles in immunogenetically susceptible individuals that resulted in a lichenoid tissue reaction. However, it is difficult to estimate whether fibrosing alopecia in a pattern distribution developed as a new condition or existed, albeit unrecognized before and accelerated after Covid 19. Still, fibrosing alopecia in a pattern distribution might have occurred just coincidentally due to a genetic, autoimmune or environmental factor which led to the inflammation of both terminal and vellus hairs.

Our experience in the present two cases suggests that fibrosing alopecia in a pattern distribution may be a new alopecia type which occurs in association with Covid 19. In patients with abrupt onset of severe hair loss months after Covid 19, before making the diagnosis of post-infectious effluvium, we recommend to evaluate the patient regarding the clinical and trichoscopic features of fibrosing alopecia in a pattern distribution and, if necessary, to perform histopathological examination. Thus, with timely diagnosis, appropriate treatment can be instituted to control fibrosing alopecia in a pattern distribution and to prevent permanent alopecia.

Nail involvement in Langerhans cell histiocytosis and its association with multisystem presentation and lung involvement

Sir,
Langerhans cell histiocytosis is a rare proliferation of cells of the mononuclear-phagocyte system mainly affecting children.¹

The great heterogeneity of clinical presentation requires histopathological confirmation and a multidisciplinary approach. According to the number of involved sites, Langerhans cell histiocytosis is clinically classified

Declaration of patient consent

The patient's consent is not required as the patient's identity is not disclosed or compromised.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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How to cite this article: Bonometti A, Passoni E, Finotto S, Berti E. Nail involvement in Langerhans cell histiocytosis and its association with multisystem presentation and lung involvement: Case study and a comprehensive review of the literature. *Indian J Dermatol Venereol Leprol* 2021;87:850-4.

Received: January, 2021 **Accepted:** May, 2021 **EPub Ahead of Print:** August, 2021 **Published:** October 2021

DOI: 10.25259/IJDVL_50_2021 **PMID:** 34491685

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