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REVIEW ARTICLE

Differentiation of frontal fibrosing alopecia and Lichen planopilaris on trichoscopy: A comprehensive review

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Abstract

Trichoscopy in dermatology has opened up the new concept in overcoming the optical challenge faced clinically. It reveals the diagnostic details to the depth of superficial dermis, and thereby increasing the clinician's diagnostic accuracy and the level of confidence. The importance of trichoscopy in cicatricial alopecia is very evident in the current scenario for its precise diagnosis. The clinical picture in delineating lichen planopilaris (LPP) and frontal fibrosing alopecia (FFA) from each other and also from other close mimickers such as androgenetic alopecia, alopecia areata, female patterned hair loss, and other cicatricial alopecia is bewildered due to its varied presentation and co-existence with the above disease. The indecisive comparative findings on histopathology make the situation even more perplexed. In this article, we have reviewed the characteristic trichoscopy patterns that can differentiate LPP and FFA and from their close mimickers.

KEYWORDS
dermoscopy, diagnosis, frontal fibrosing alopecia, lichen planopilaris, trichoscopy

1 | INTRODUCTION

Lichen planopilaris (LPP) and frontal fibrosing alopecia (FFA) are grouped under primary lymphocytic cicatricial alopecia. The term "cicatricial" originates from the Latin word "cicatrix," meaning "scar" in which inflammatory infiltrate cause complete destruction of the follicular stem cells with permanent scaring.[1]

After the first description of LPP by Pringle in 1985,[2] several subtypes have been depicted. The classical form of LPP usually affects middle-aged women and men, involves the vertex that can be associated with violaceous keratotic follicular papules of lichen planus on trunk and extremities.[3] Other variants are Graham-Little-Piccardi-Lasseur Syndrome (GLPLS),[4] fibrosing alopecia in a patterned distribution[5] and Cicatricial Pattern Hair Loss (CPHL).[6]

In 1994, Kossard first described Frontal Fibrosing Alopecia (FFA) as an individual disease considering the unique clinical presentation.[7] It typically presents as an androgenetic patterned fronto-temporal band of alopecia with receding frontal hairline, mostly seen in postmenopausal women.[8] Though considered as relatively rare, it is possible that FFA is often underdiagnosed and is, in fact, not a rare disorder.

LPP and FFA can closely mimic discoid lupus erythematosus, alopecia areata, androgenetic alopecia, female pattern alopecia and

even trichotillomania and folliculits decalvans. The recognition of LPP and FFA might be clinically challenging due to multiple reasons such as the heterogeneous clinical manifestations, close resemblance between them clinically and histopathologically and possibly, their co-existence in the same patient. Trichoscopy is a very useful tool not only for an appropriate diagnosis but also for planning the treatment protocol, assessing the response following treatment and its prognosis.

2 | PREDISPOSING FACTORS

The etiology of LPP is not fully elucidated but is known to involve an autoimmune process, probably triggered by stress, contacts sensitization, infection, metal exposure, or pollution.[9,10] Sunlight is also considered to be an important predisposing factor as well. Other possible factors are hepatitis C, amla oil, mustard oil, hair dyes, fragrances, etc.

FFA most commonly occurs in postmenopausal women, but few cases of premenopausal women and men have been reported. Hormonal and autoimmune factors are also involved in the evolution of the disease. The hormonal etiology is supported by the fact the predilection for postmenopausal women, early onset of disease in premature menopause and better treatment response with finasteride and dutasteride.[11] Thirty percent of FFA seems to be coexistent with different autoimmune diseases namely vitiligo, thyroid dysfunction, lupus, Sjogren's syndrome depicting the autoimmune etiology as well.[12]

The follicular bulge area is an immunologically privileged site due to downregulated MHC class I and β2 microglobulin and it is guarded by expression of TGFβ2 and CD200. In LPP, defective genetic expression exposes the follicular stem cells triggering an autoimmune response. Various familial cases of FFP have supported the evidence of FFA as an autosomal dominant inheritance with inadequate penetrance. A recent genomic study on FFA observed the genetic variation of four different susceptibility foci such as "2p22.2, 6p21.1, 8q24.22 and 15q2." The study also has reported FFA as "genetically predisposed immuno-inflammatory disorder" with a strong association with Class I allele HLA-B*07:02 on chromosome 6p21.1. HLA-B*07:02 may promote the expression of the autoantigen resulting in the initiation of lymphocytic destruction of the follicular bulge.[13]

Several other predisposing factors such as environmental pollution, stress, infection do play a role. Daily facial sunscreen usage is also proposed as a possible contributor to the pathophysiology of FFA.[14]

3 | CLINICAL FEATURES

The course of LPP can be extensive and highly active. The lesions frequently involve the vertex, even though any of the body area can be affected. The early lesions present with itching, pain and burning sensation. Initially, LPP manifests with a violet-red-colored erythema around the follicle, better seen in fair skin individuals. The erythema is mainly seen at the periphery of the affected area, indicating an active inflammatory border. The inflammation tends to cease spontaneously at the center as the hair follicles are replaced by the fibrous tissue, resulting in cicatricial alopecia, whereas the periphery may still show the active inflammatory process and tend to expand peripherally.[15] The scar tissue replaces the pilosebaceous structures and destroys the stem cells of hair follicle. LPP also can involve rarely other body hair such as eyebrow, eyelash, axillary, and genital hair. Other findings are pustules at the edge mimicking folliculitis decalvans with tufted hairs. Positive pull test under microscopy show predominant anagen hairs.[16]

FFA has a chronic phase and has a relatively slow progression (0.9 mm/month or 0.6–1.1 cm recession per year).[9] It manifests with the band of a receding frontal and temporo-parietal hairline resulting in cicatricial alopecia.[17] FFA can be classified into linear, diffuse and double-line or pseudofinge pattern. Other rare patterns are ophiasis-like, cockade-like, androgenic alopecia-like pattern.[18] The band may progress to involve the pre-auricular, post-auricular, and also to the occipital areas. Eyebrows involvement in the form of complete or partial loss may be an initial sign of FFA in around 75% of cases.[19] It can also affect eyelash and body hair.[20] The vellus hair involvement on the face may present as multiple facial papules.[21] Though most of the time it is asymptomatic, one-third of the patient can present with pruritus, burning sensation, or trichodynia. After the complete hair loss, FFA leaves a smooth shiny skin in contrast to the normal skin of the forehead. It is also called as clown alopecia.

The co-existence of both LPP and FFA though rare but have also been reported in literature. Corralo et al analyzed 103 female patients with FFA of which 16.5% were associated with concomitant LPP. The involvement of non-scalp areas, facial papules and intense lichenoid reactions with histopathology have been reported in such cases.[22]

4 | HISTOPATHOLOGY

Previous studies reported that differentiating LPP from FFA is very difficult.[23] Histopathology of LPP show hyperkeratosis with or without follicular plugging, absent or decreased or miniaturized hair, infundibular wedge-shaped hyperkeratosis, and vacuolar degeneration of basal cell layer, epidermal and follicular cystoid bodies. The dense perifollicular and perivascular lymphocytic infiltrate with infiltration at the infundibular or isthmic level is seen with relative sparing of the lower parts. The perifollicular fibrosis can be appreciated as concentric lamellar pattern in which mucinous perifollicular fibroblasts is also seen. At later stage, there is a complete destruction and loss of sebaceous glands and hair follicles.

Histopathology of FFA is also typified by a perifollicular lymphoid cell infiltrates and perifollicular fibrosis with late-stage showing hair follicle destruction, similar to the findings in LPP. As compared to LPP, FFA has less inflammation and a greater degree of apoptosis.[24] Presence of terminal hair with more of catagen-telogen type favors

the diagnosis of FFA, whereas intense inflammatory infiltrate in the peripilar zone with concentric lamellar fibroplasia more suggestive of LPP. Similarly, interfollicular epidermis is almost never involved in FFA, while it might be affected in LPP.[25] But these observational findings were not conclusive of either LPP or FFA.

5 | DIFFERENTIAL DIAGNOSIS IN DERMOSCOPY

The characteristic feature of LPP is the perifollicular collar-like scaling better seen on dry trichoscopy. They are shiny silvery-white in color encircling 2–3 hair follicles. This tubular cast surrounds 2–3 mm of the proximal hair shaft (Figure 1). It is observed that in the early stages, the yellow hue on an erythematous background can be seen in fair-skinned individual. It corresponds to the dilated follicular ostia filled with sebaceous material or dense perifollicular lichenoid infiltrate with basal cell degeneration on histology. (Figure 2) The melanocytic incontinence with melanophages and free melanin in the papillary and reticular dermis around the follicle, on trichoscopy, shows bluish-gray circle encircling perifollicular area giving a "target" appearance with relatively sparing the interfollicular region. When the infiltration extends across the follicular area and with simultaneous extensive fibrosis, diffuse milky white areas on a homogenous pink-red background can be seen. Milky red areas always correspond to early onset fibrosis. (Figure 3) In the center of the bald area, there is loss of vellus hair with loss of follicular orifices and hence predominant terminal hair can be appreciated in the early stage (Table 1).

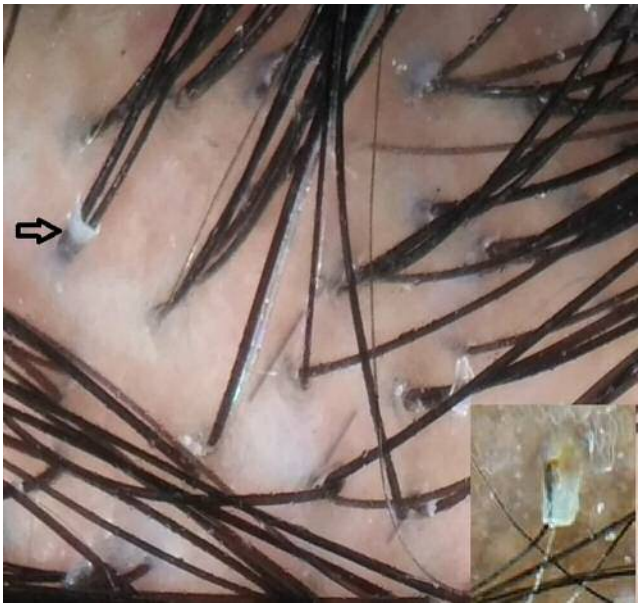

FIGURE 1 Dermoscopic image of lichen planopilaris showing Perifollicular tubular cast encircling 2–3 follicles, 2–3 mm of the proximal hair shaft (black arrow) (inlet- closer view of tubular cast)

The white dots are of two types, pinpoint white dots (ductal opening) and larger white dots (underlying fibrosis). At a later stage, pinpoint white dots disappear due to destruction of glands and the multiple fibrotic white dots coalesce to form diffuse white structure less areas corresponds to underlying dense fibrosis. The external manipulation of stalk can be visualized as a black dots, but it may also be correlated with high activity of the disease. (Figure 4) Margin may show elongated dilated vessels, keratotic plugging, broken hairs, tufted hairs, or pili torti.[26] But significant miniature of hair with predominant vellus hair is typical of androgenetic alopecia, which is not seen in LPP. In the skin of color, the erythema is less appreciated, whereas the tubular cast is well appreciated, and the pigmentation is more intense with multiple white dots in a background of honeycomb pigmentation forming a "starry sky pattern." The active disease is indicated with the presence of perifollicular cast, targetoid blue-gray pigmentation, and milky red areas.[27,28]

Anagen pull test can also predict the disease activity. It correlates with the active interface dermatitis on histopathology.[29] Anagen hair has pigmented bulb and honey stick or golf-stick appearance while telogen hair show the club-shaped cornified bulb with the remnant of epithelial sac-the earbud appearance.

The characteristic feature of FFA is the intense perifollicular erythema in the early stages. This perifollicular erythema with mild scaling can also be appreciated even in the prodromal phase, before receding frontal hairline. Though all other trichoscopy features are same as LPP, the findings are more evident on the margin of the frontal hairline. Some features differ from LPP such as relatively milder perifollicular scaling, associated facial papules showing mild perifollicular scaling and erythema and predominant single hair units at the margin of hair-bearing area. (Figure 5) As the vellus hairs are predominantly affected, some of the terminal hair stands out as single lone hair along the margin of the receding frontal and pre-auricular area termed as "lonely hair sign."[30] (Figure 6) Unlike the milky red background in LPP, FFA has an ivory-beige background.

Eyebrow may show dystrophic hair, black dots, red dots in early stage and blue-gray dots in the later stage.[31]

Nirmal et al. described the concept of "invisible LPP" in which lesions were not visible to the naked eye but the trichoscopy findings gave a clue for the diagnosis before even the clinical evidence of hair loss.[32] Especially, when LPP is more commonly confused with AGA. It is similar to the concept of "invisible LP" published by Gougerot.[33]

LPP in a patterned distribution mimics AGA. Though both FFA and AGA have an indolent course individually, when FFA occurs on a long-standing AGA it can progress rapidly.[34] This can be precipitated either spontaneously or after an interventional procedure such as follicular hair transplantation. The catastrophic event give can rise to permanent scarring alopecia on the donor, as well as the recipient site.[35] When AGA coexist with FFA trichoscopic features shows the feature of both the condition. The mixture of trichoscopic findings such as hair thinning more than 20%, group of hair (2–4 in number) arising from the same ostium in the area of thinning, perifollicular erythema and scaling of the affected scalp hair, a normal-appearing eyebrow and without predominant loss of vellus hair should arise

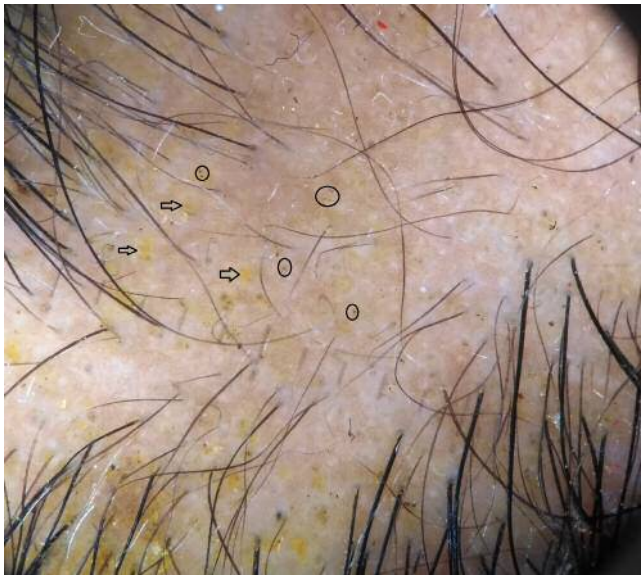

FIGURE 2 Dermoscopic image of lichen planopilaris showing yellow areas corresponds to the dilated follicular ostia filled with sebaceous material (black arrow) and multiple black dots (black arrow)

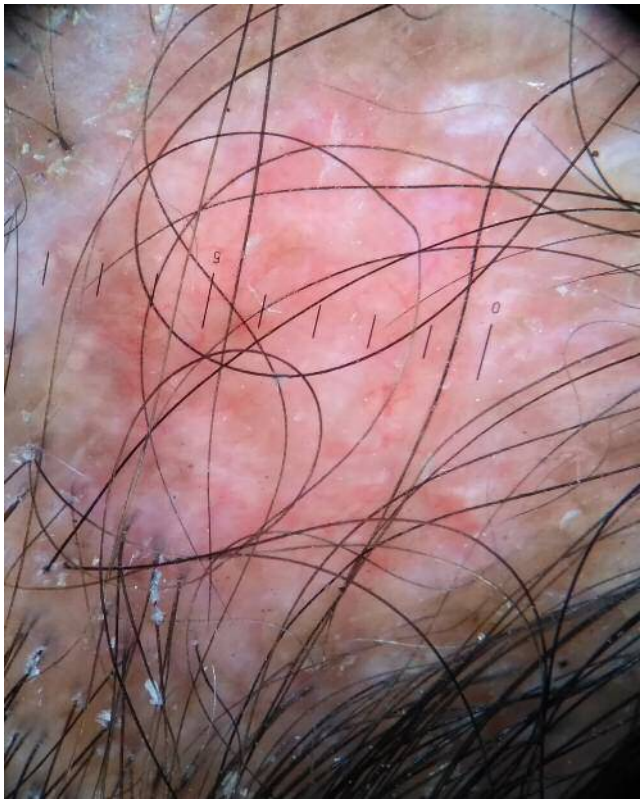

FIGURE 3 Dermoscopic image of lichen planopilaris showing milky red areas with perifollicular scaling

the suspicion of co-existence of both condition.[36] The aim of treatment should be stopping the progression of cicatrization through an early intervention and in such scenario surgical treatment is to be deferred for a minimum period of 2 years.[37]

In Female Patterned Hair Loss (FPHL), presence of vellus hair at the frontoparietal region is the consistent diagnostic feature and on analysis, the terminal hair to vellus hair ratio is drastically reduced from 8:1 to 2.2:1. Any ratio less than 4:1 can be diagnostic of FPHL.[38] Absence of perifollicular erythema and tubular cast and presence of variable hair diameter more than 20%, peripilar sign favors the diagnosis of FPHL. The pulled hair of hair pull test can be analyzed under trichoscopy and microscope.

Trichoscopy of discoid lupus erythematosis (DLE) shows large multiple yellow dots along with scattered dark-brown pigmentation

TABLE 1 Correlation between histopathological and trichoscopic findings

No	Trichoscopic findings	Histopathological corelation
1	Intense perifollicular pink-reddish areas	Follicular interface dermatitis
2	Yellowish hue on a erythematous background	Liquefaction degeneration of basal cell layer
3	Comedone-like opening filled with brownish-black keratinous plugging.	Dilated hair follicles and keratinous plugging
4	Decreased pigmentation and hair shaft diameter	Miniaturized hair
5	Perifollicular tubular cast/ silver white collar-like scaling encircling 2–3 hair follicles	Perifollicular hyperkeratosis
6	Loss of pinpoint white dots	Atrophy of eccrine ducts
7	Decreased/ loss of larger white dots	Atrophy of hair follicle
8	Perifollicular streaks of lusterless white area	Concentric lamellar perifollicular fibrosis
9	Interfollicular vessels with single loop and arborizing vessel	(no histopathological correlation)
10	Multiple blue-gray globules and dots	Pigment incontinence with pigment laden macrophages and free melanin
11	Stellate-shaped white areas	Wedge-shaped fibrosis
12	Milky-red white areas	Early onset fibrosis with mucin and active inflammation
13	Dull-White structureless areas	Dense fibrosis (at later stage)

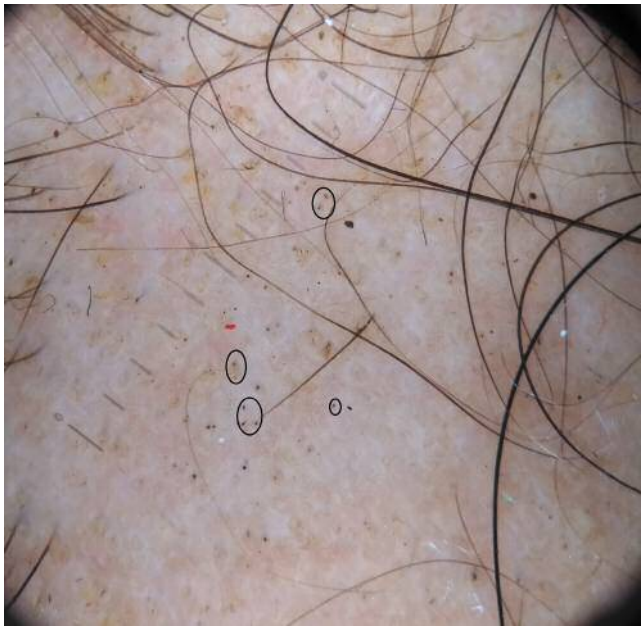

FIGURE 4 Dermoscopic image of lichen planopilaris showing yellow areas, areas of erythema and extensive black dots indicating the high activity of the disease. (black arrow)

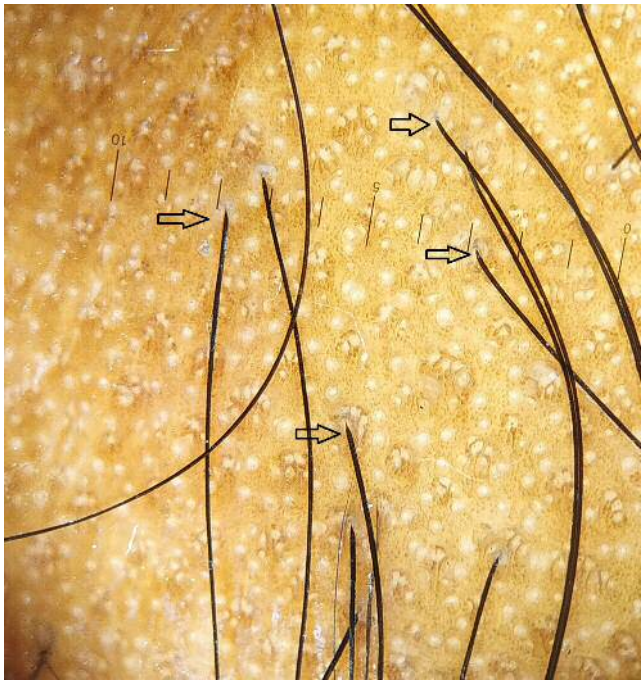

FIGURE 5 Dermoscopic image of frontal fibrosing alopecia predominant single hair units at the margin of hair-bearing area and lonely hair sign

and dense arborizing vessels. It has a characteristic follicular keratotic plugging that can be appreciated well on non-polarized mode.[39] The tissue fibrosis in DLE appear as white structureless area and the speckled patterned arrangement of blue-gray dots signifying

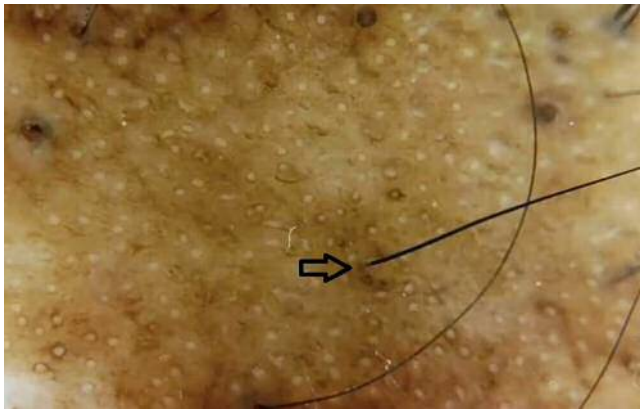

FIGURE 6 Dermoscopic image of frontal fibrosing alopecia showing lonely hair sign. (black arrow)

the interfollicular involvement with the sprinkling of melanin, unlike LPP where the unique targetoid pattern is appreciated. Honeycomb pattern also favor LPP.[40]

Although, multiple patches of classical alopecia areata (AA) and theophiasis form can simulate LPP, the sensitive markers to distinguish alopecia areata can be short vellus hair and yellow dots and the specific markers are exclamatory hair, broken hairs, black dots, and tapering hair. Peripilar tubular cast and erythema with follicular plugging especially at the margin and reduced follicular opening at the center, favor the diagnosis of LPP. The weakening and periodic constriction of the anagen hair shaft is called as Pohl-Pinkus constriction that can be observed in both LPP and AA.[41]

Intense perifollicular erythema in some of the cases of trichotillomania (TTM), as well was observed. However, it is associated with multiple superficial erosions. Other definitive features of TTM such as broken hair of different length, broken hair with coiled tip, trichoptillosis, black dots, flame hair, mace hair, tulip hair, hair dust can aid in definitive diagnosis.[42]

Tufted hair is also observed in folliculitis decalvans in which the tufts of six or more hair emerging from the single ostium and has yellowish peripilar cast with multiple perifollicular pustules and scaling.[43]

Yet another significant role of trichoscopy is guidance to decide the site of biopsy and also the assessment of clinical improvement after therapy. Trichoscopic assessment of all case of AGA before planning any interventional procedure will also prevent the catastrophic events of permanent hair loss after transplantation.[44]

6 | CONCLUSION

LPP and FFA are two common cicatricial alopecia that needs early diagnosis and appropriate therapeutic intervention to prevent permanent hair loss. It poses a great challenge for the dermatologist to delineate it from the closest mimickers due to lack of epidemiological data, varying clinical presentation and often leading to misdiagnosis. It does affect strongly the quality of life and the social well-being. Trichoscopy, on

the other hand, helps with sufficient evidence for the adequate diagnosis of LPP and FFA from its differentials such as AGA, AA, DLE, and other cicatricial alopecia. The use of trichoscopy has increased the confidence of decision making among the dermatologist reciprocated with a rise in the incidence of LPP making the "invisible" to "visible."

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CONFLICT OF INTEREST

No conflict of interest to declare.

AUTHOR CONTRIBUTIONS

Aswath Rajan: Writing and revising the manuscript. Lidia Rudnicka: Review and revising the manuscript. Jacek C Szepietowski: Review and revising the manuscript. Aimilios Lallas: Review and revising the manuscript. Ghasem Rahmatpour Rokni: Review and revising the manuscript. Stephan Grabbe: Review and revising the manuscript. Mohamad Goldust: Conception, writing, review and revising the manuscript.

ETHICS STATEMENT

Written consent was obtained from the patients whose pictures were used in the study.

DISCLAIMER

We confirm that the manuscript has been read and approved by all the authors that the requirements for authorship as stated earlier in this document have been met and that each author believes that the manuscript represents honest work.

DATA AVAILABILITY STATEMENT

Data sharing not applicable to this article as no datasets were generated or analysed during the current study.

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