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Case Report

Facial Discoid Dermatosis Mimicking Vitiligo in a South Asian Woman with Fitzpatrick Type IV Skin: Response to Methotrexate and Topical Ruxolitinib

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Abstract: Facial discoid dermatosis (FDD) is a rare, recently described condition characterized by persistent facial lesions that are often refractory to topical and systemic treatments. It typically presents as annular, well-demarcated papules and plaques with fine scale on the face, with a histopathologic pattern resembling pityriasis rubra pilaris. The differential diagnosis is broad, including seborrheic dermatitis, psoriasis, lupus, rosacea, and mycosis fungoides, making diagnosis challenging. In darker skin, FDD may present as hypopigmented macules that are mistaken for vitiligo or post-inflammatory hypopigmentation. Treatment options for FDD remain limited, with few reports of successful interventions. We present a 40-year-old South Asian woman with Fitzpatrick type IV skin and a 10-year history of hypopigmented facial lesions initially diagnosed as vitiligo. We describe the dermoscopic findings, including retained pigmented vellus hairs, and her improvement with oral methotrexate combined with topical ruxolitinib 1.5% cream, with continued repigmentation on topical ruxolitinib alone. FDD should be considered in the differential diagnosis of treatment-resistant facial hypopigmentation in darker skin.

Keywords: facial discoid dermatosis; topical ruxolitinib, Janus kinase inhibitor, methotrexate, skin of color, dermoscopy, vitiligo

1. Introduction

Facial discoid dermatosis (FDD) is a rare and newly established diagnosis, having first been described in 2010 [1]. The most characteristic feature of this condition is the presence of persistent lesions refractory to both topical and systemic treatments, such as corticosteroids, topical calcineurin inhibitors, antibiotics, immunomodulators, and phototherapy. FDD typically presents as annular, well-demarcated, pink-orange papules and plaques with fine scale that are primarily localized to the face. The number and size of lesions vary, and reported patients have ranged in age from 11 to 61 years, most commonly women [2]. Similar to pityriasis rubra pilaris (PRP), histopathology of FDD demonstrates hyperkeratosis, parakeratosis, psoriasiform acanthosis, follicular plugging, and a perivascular lymphocytic infiltrate. The differential diagnoses include seborrheic dermatitis, contact dermatitis, atypical psoriasis, cutaneous lupus, rosacea, mycosis fungoides, post-inflammatory hypopigmentation [1], nummular dermatitis, and tinea faciei [3]. Most patients do not improve with treatment [2,4]. Among the limited literature, improvement has been reported with topical calcipotriol alone or combined with betamethasone [5–8], topical sirolimus [9], oral retinoids [6,10], ustekinumab [11], secukinumab [12], and most recently, topical ruxolitinib [13] (Table 1).

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Herein, we report a case of FDD in a 40-year-old woman with Fitzpatrick type IV skin whose hypopigmented lesions were initially diagnosed as vitiligo, and who improved with oral methotrexate combined with topical ruxolitinib cream 1.5% (Opzelura).

Table 1. Reported treatments for facial discoid dermatosis and their outcomes.

Treatment	Reported Outcome	References
Topical calcipotriol, alone or with betamethasone	Improvement to near-complete clearance in single case reports; in one cohort, moderate improvement in one patient and no improvement in another with a topical vitamin D analogue	[4–8]
Topical sirolimus 0.2%	Complete response after one month in one case	[9]
Topical Janus Kinases (JAK) inhibitors	Ruxolitinib 1.5% cream: complete clearance within three weeks as monotherapy in one case. Unspecified topical JAK inhibitor: no improvement in one cohort patient. Compounded tofacitinib 2%: no improvement in the present case	[4,13]
Oral retinoids	Acitretin: improvement with concurrent topical calcipotriol/betamethasone in one case; near-complete clearance after six months in another case; and moderate improvement in one cohort patient (stopped because of hair loss). Isotretinoin: complete response in one case	[4,6,10]
Methotrexate	Partial response in one patient (10 mg weekly); Moderate improvement in one cohort patient and no response in another (20 mg weekly); no response in a further case report; listed among ineffective treatments in a literature review; improvement in combination with topical ruxolitinib in the present case	[4,12,14]
Ustekinumab	Improvement in individual case reports; near-complete clearance in combination with apremilast in one cohort patient	[2,4,11]
Secukinumab	Marked improvement with near-complete resolution in one case; partial improvement after six months in another	[9,12]
Topical corticosteroids, calcineurin inhibitors, and antifungals; systemic antibiotics, antifungals, and hydroxychloroquine; phototherapy; pulsed dye laser	Generally no improvement or inconsistent response	[2,4]

2. Case Report

A 40-year-old South Asian woman with Fitzpatrick type IV skin presented with a 10-year history, beginning in September 2014, of round, pink to slightly hypopigmented macules and slightly elevated papules, patches and plaques ranging in size from 6 mm to 20 mm, isolated to her face (Figure 1A,B). The patient described the lesions as moderately pruritic and scaly, with a mild burning sensation. Since onset, the lesions had progressively increased in both number and size. They were initially localized to the forehead and subsequently expanded to involve the glabella, the paranasal, and mandibular region, the cutaneous upper and lower lips, the lower cheeks, and the chin.

The lesions had never fully resolved since their onset. Review of systems was negative for constitutional symptoms, and there were no concurrent signs suggestive of autoimmune disease. She was initially diagnosed with vitiligo and later with petaloid seborrheic dermatitis. When she did not respond to therapy, she had three skin biopsies, which varied in their histopathologic diagnoses and were ultimately inconclusive. Skin scraping for fungal culture was negative, and syphilis serologies were negative. Medical history was significant for asthma, seasonal rhinitis, and exercise-induced bronchospasm. Her medications included a levonorgestrel intrauterine device that was started after the onset of the facial lesions. Before October 2024, she tried multiple treatments, none of which improved her lesions (Table 2). These include topical calcineurin inhibitors, corticosteroids, antifungals, compounded tofacitinib 2% cream, and roflumilast 0.3% cream; oral antifungals, antibiotics, and hydroxychloroquine; and narrowband ultraviolet B (UVB) phototherapy administered one to three times weekly from 2022 to 2023.



Figure 1. Clinical images of the face before starting methotrexate and topical ruxolitinib, in summer 2024: (**A**) Upper frontal view of face, (**B**) Lower frontal view of face.

Table 2. Summary of treatment from onset to the time of submission.

Timeline	Topical Treatment	Systemic Treatment
September 2014 (onset) to September 2024	Tacrolimus 0.1% ointment; Pimecrolimus 1% cream; Ciclopirox olamine 1% cream; Ketoconazole 2% cream; Hydrocortisone 1% cream; Betamethasone dipropionate 0.05%/clotrimazole 1% cream; Ketoconazole 2%/hydrocortisone valerate 0.2% (compounded); Tofacitinib 2% in dermabase (compounded); Roflumilast 0.3% cream	Itraconazole 200 mg/week × 4 weeks; Fluconazole 150 mg weekly × 6 weeks; Amoxicillin 500 mg TID × 7–10 days, two courses; Nitrofurantoin 100 mg BID × 5 days; Sulfamethoxazole/trimethoprim 800 mg/160 mg BID × 30 days; Hydroxychloroquine 200 mg × 3 months; Narrowband UVB phototherapy one to three times weekly, 2022–2023
October 2024–December 2024	None	Methotrexate 20 mg/week; Folic acid 5 mg/day
December 2024–February 2025	Ruxolitinib 1.5% cream BID	Methotrexate 20 mg/week; Folic acid 5 mg/day
February 2025 to present	Ruxolitinib 1.5% cream BID	None

TID, three times daily; BID, twice daily.

The three biopsies were taken in 2016, 2018, and 2024. The first, taken from the left temple in December 2016, showed mild epidermal hyperplasia and spongiosis with overlying hyperkeratosis, focal parakeratosis, and a superficial perivascular lymphocytic infiltrate with pigment incontinence. Melanoma Antigen Recognized by T cells-1 (MART-1) staining showed a normal melanocyte population, making vitiligo unlikely. Fungal stains with Periodic Acid-Schiff (PAS) were negative. A second biopsy from the right cheek in February 2018 again showed normal basal melanocytes, mild pigment incontinence, and no active dermatitis. PAS and alcian blue pH 2.5 (AB 2.5) stains were negative. Findings remained non-specific, consistent with post-inflammatory pigmentary alteration.

The third and final biopsy (Figure 2A,B), obtained in May 2024 from the left temple, revealed patchy parakeratosis with hyperkeratosis with follicular plugging, as well as mild spongiosis, irregular acanthosis, and a

superficial perivascular lymphohistiocytic infiltrate with occasional plasma cells and rare eosinophils. No vacuolar interface change, dermal mucin, or granulomatous inflammation was seen. These findings were most consistent with lichenified spongiotic dermatitis, such as seborrheic dermatitis. Given the clinical distribution and morphology, a petaloid variant of seborrheic dermatitis was also considered, though the histologic differential diagnosis included other eczematous processes. Based on the histopathologic findings, she was treated for seborrheic dermatitis and other eczematous processes with the agents listed above, without success.

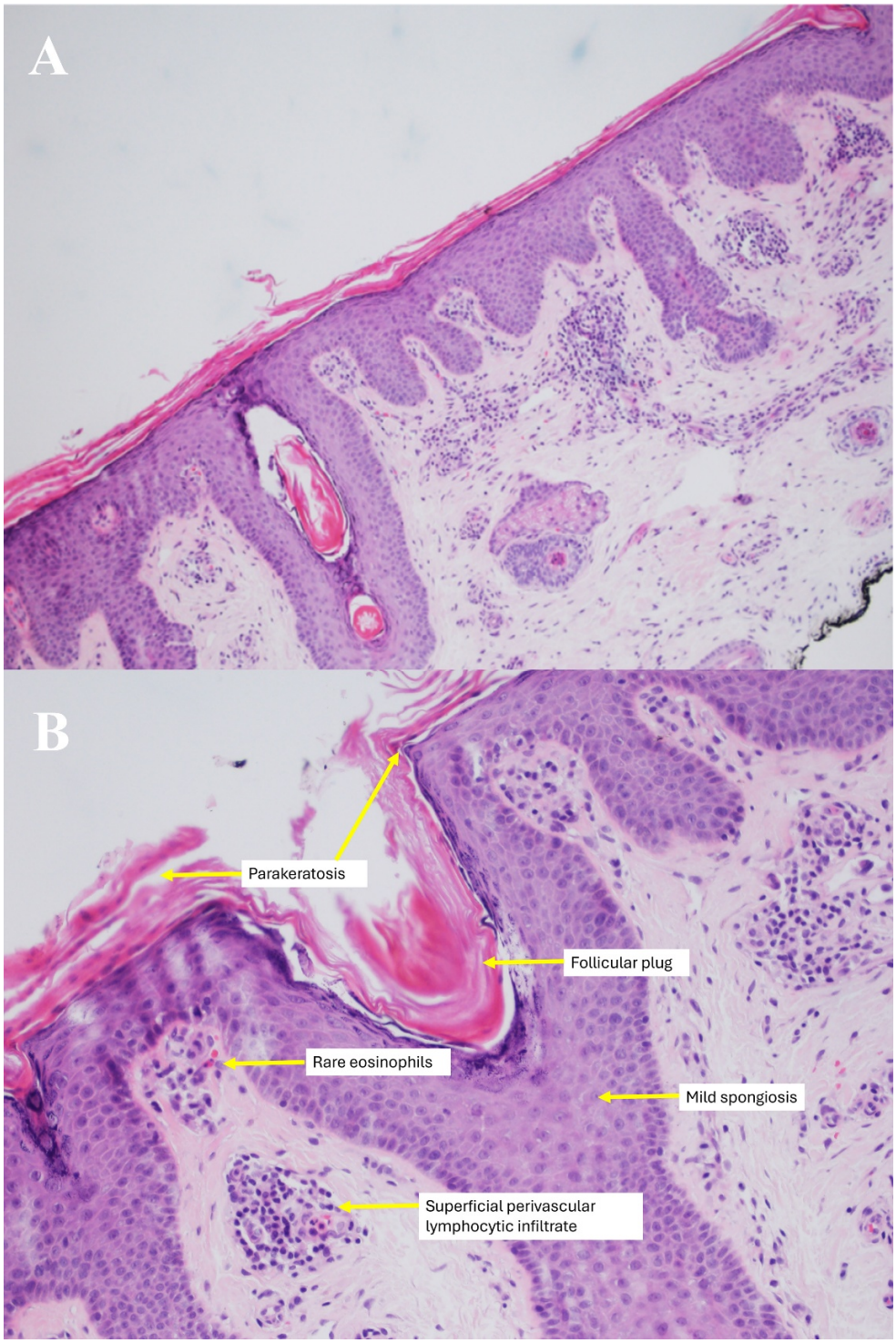


Figure 2. Histopathologic Images of the facial biopsy (Hematoxylin and eosin, H&E), from May 2024. (**A**) Parakeratosis with hyperkeratosis with follicular plugging. The granular layer is retained to mildly thickened. The epidermis shows mild irregular spongiosis and acanthosis. Within the dermis, there is mild fibrosis and a superficial perivascular lymphocytic inflammatory infiltrate with lymphocytes, occasional plasma cells and rare eosinophils (H&E, ×10). (**B**) There is perifollicular parakeratosis, mild spongiosis extending into the hair follicle. A rare eosinophil is apparent. Follicular plugging is also appreciated here (H&E, ×20).

Because her eruption had not responded to any of these treatments, and the May 2024 biopsy suggested a spongiotic dermatitis, oral methotrexate 20 mg weekly with folic acid 5 mg daily was started in October 2024. In December 2024, twice daily topical ruxolitinib 1.5% cream (Opzelura), which is approved in Canada for nonsegmental vitiligo and atopic dermatitis [15], was added, before the diagnosis of FDD was established. In January 2025, she was patch tested to rule out allergic contact dermatitis contributing to her facial eruption. She was tested to the North American Comprehensive series (Dormer) and the cosmetic and fragrance series (Dormer), and had positive reactions to amerchol L101/lanolin, diphenylguanidine, and carba mix. However, upon review of her history, personal care products, and exposures, none of these allergens were relevant to her facial eruption. Based on her clinical presentation, her history, review of past biopsy results, patch test results that were not relevant to her eruption, and her long history of treatment failures, she was diagnosed with FDD.

At the time of the patch testing visits in January 2025, she had been on 4 weeks of topical ruxolitinib 1.5% cream and 3 months of methotrexate 20 mg/week (and folic acid 5 mg/day), and had started to notice improvement of her lesions, with less scaling and mild repigmentation (Figure 3A–C). Dermoscopy revealed hypopigmented macules and patches with ill-defined central erythema and mild patchy fine scale, and retained pigmented vellus hairs (Figure 4A,B).

By eight weeks of use of the topical ruxolitinib cream (and approximately four months of oral methotrexate), in February 2025, she noted significant improvement of her facial eruption. At that time, she discontinued methotrexate but continued using topical ruxolitinib with continued good effect until approximately two months later, in April 2025, when she noticed a mild flare of her lesions, although the overall severity was not marked. At her follow-up visit in late April 2025 (Figure 5A,B), despite the earlier flare, there was visible repigmentation of previously affected areas, as well as a marked reduction in both the number and size of residual macules. The most significant improvement occurred during combined therapy with methotrexate and the topical ruxolitinib. At her most recent clinic visit in July 2025, she reported continued repigmentation of her lesions on topical ruxolitinib alone. At the time of manuscript submission (August 2026), she reported progressive repigmentation of her lesions and that her skin was better than before she started on the topical ruxolitinib. Table 2 summarizes her treatment.

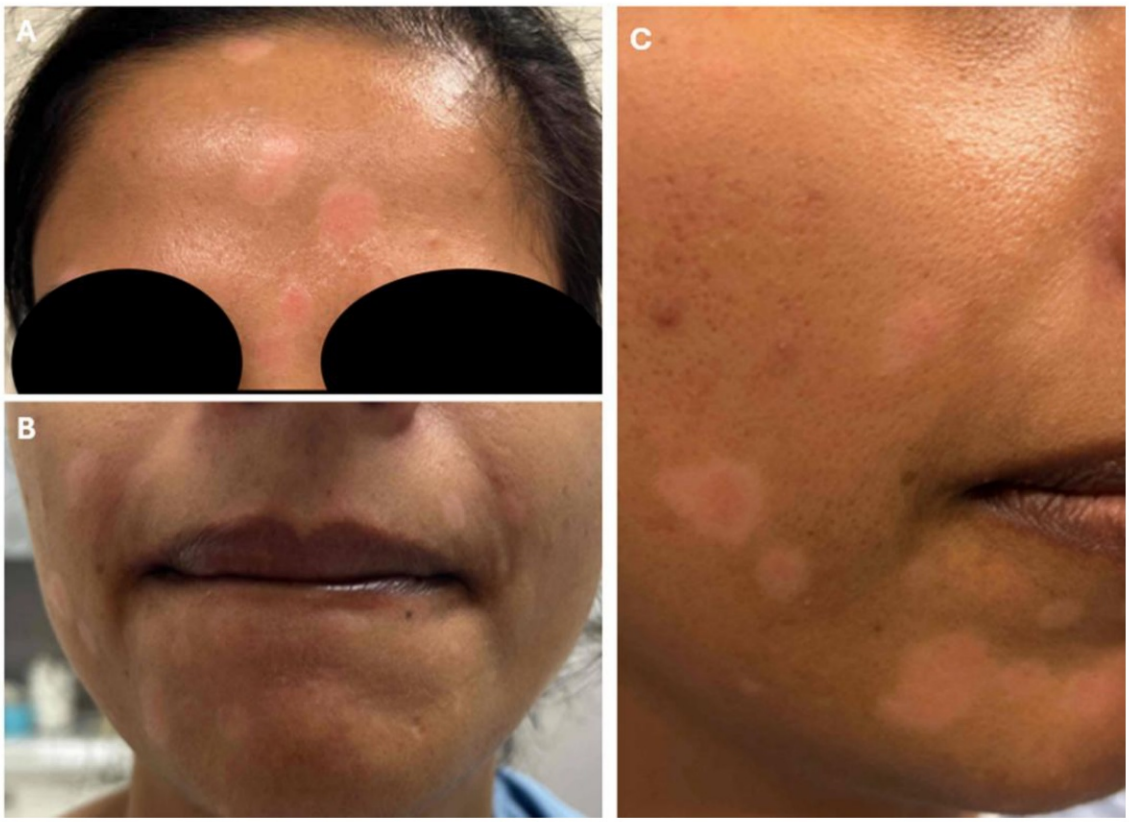


Figure 3. Clinical images of the face after initiation of topical ruxolitinib (4 weeks) and oral methotrexate and folic acid (approximately 3 months), in January 2025. (**A**) Upper frontal view of the face. (**B**) Lower frontal view of the face. (**C**) Right lateral view of the lower face.

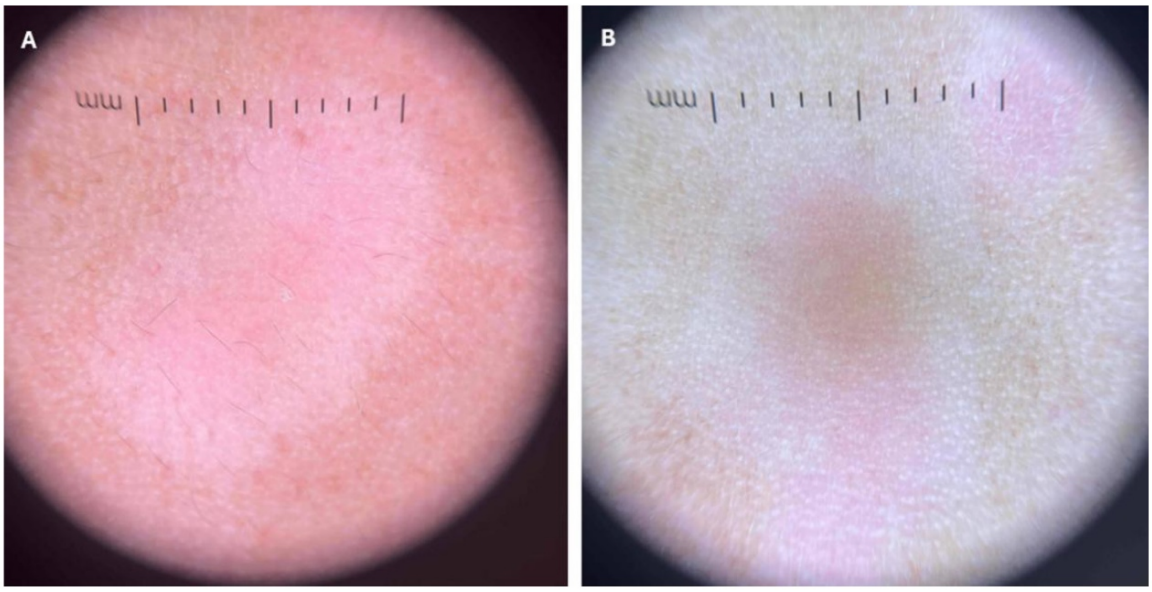


Figure 4. Dermoscopic images of the facial lesions, in January 2025. (**A**) More inflamed lesions on the forehead showing hypopigmentation, faint central erythema, fine white patchy scales, and pigmented vellus hairs. (**B**) Less inflamed lesions along the jawline showing hypopigmentation, faint central erythema without any signs of scales. Note the slight central hyperpigmentation is due to the shadow cast by the dermatoscope.

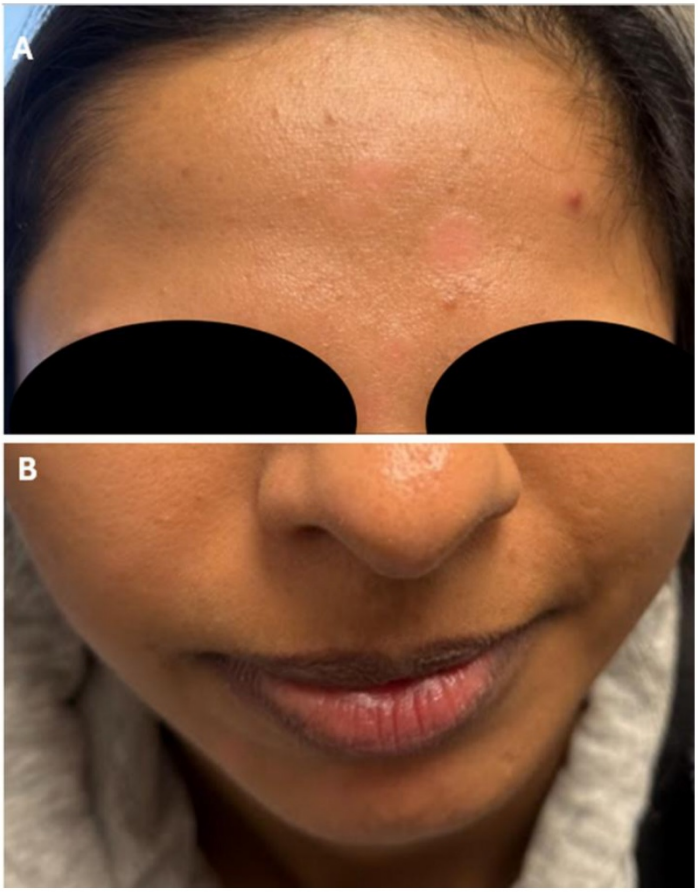


Figure 5. Clinical images of the face in late April 2025, after approximately 4.5 months of topical ruxolitinib cream and approximately 2 months after methotrexate (taken for approximately 4 months) was stopped: (**A**) Upper frontal view of the face showing repigmentation of lesions on forehead. (**B**) Lower frontal view of the face showing repigmentation of the lesions.

3. Discussion

FDD is a cutaneous condition that remains challenging to diagnose due to its rarity, relatively recent recognition [1], non-specific histopathologic findings, and the broad range of clinical mimics. FDD has rarely been described in darker skin (Fitzpatrick types IV to VI). Most published cases describe pink to pink-orange, erythematous lesions, including a series of eight Chinese patients from Singapore [16] and a recent cohort of six White women [4]. A hypopigmented presentation has been described in only one other patient, a 48-year-old Indian woman with Fitzpatrick type IV skin whose lesions improved with topical calcipotriol [2,5]. Our patient, also a South Asian woman with Fitzpatrick type IV skin, presented similarly, which suggests that in darker skin FDD may present as round hypopigmented macules clinically resembling post-inflammatory hypopigmentation or vitiligo. Only a few treatments have improved or resolved lesions, reflecting the treatment-refractory nature of FDD. Reported treatments and their outcomes are summarized in Table 1.

Some have coined FDD as a "seventh form" of PRP, with some authors hypothesizing it could be a variant of PRP localized to the face. This is because both PRP and FDD may show similar histopathological findings, such as psoriasiform acanthosis, follicular plugging, alternating orthokeratosis, and parakeratosis [7,16]. Due to similarity between FDD and PRP, treatment with ustekinumab has been proposed [11]. Welborn et al. reported a case of FDD in which a biopsy demonstrated atrophy of sebaceous lobules, a feature more typical of psoriasis or seborrheic dermatitis [17]. More recently, Ziebart et al. found negative or weak IL-36γ staining in FDD, in contrast to the strong expression typical of psoriasis and PRP, and both they and Zygadlo et al. have argued that FDD is a distinct entity rather than a variant of PRP [4,18]. A familial case suggests a possible genetic contribution [19].

Although FDD has been reported by several authors, its histopathologic features remain non-specific and variable across the literature. Welborn et al. identified focal parakeratosis, follicular plugging, and psoriasiform hyperplasia as the most common findings, but demonstrated that not all cases necessarily display these features [17]. Ko et al. similarly described parakeratosis, hyperkeratosis, acanthosis, and follicular plugging, with occasional spongiosis, but emphasized that histopathological variability does not necessarily exclude FDD, especially if the clinical picture correlates with the diagnosis [1]. In our case, the biopsies demonstrated several of these features, including parakeratosis, follicular plugging, acanthosis, and spongiosis. There are no pathognomonic features of FDD, so histopathology mainly serves to exclude mimics. In our patient, normal MART-1 staining on two biopsies was the strongest evidence against vitiligo, and the absence of interface change and dermal mucin argued against cutaneous lupus. The combination of clinical judgment with histopathology is therefore essential for diagnosis.

Janus kinases (JAKs) are cytoplasmic protein tyrosine kinases that phosphorylate and activate signal transducer and activator of transcription (STAT) proteins in response to cytokine stimulation [20]. JAK inhibitors therefore block signaling by multiple cytokines and reduce the expression of genes that contribute to inflammation [20]. Topical ruxolitinib 1.5% cream, a JAK1/JAK2 inhibitor, is used to treat vitiligo and atopic dermatitis [21,22]. Garcia et al. recently reported complete clearance of FDD within three weeks of topical ruxolitinib monotherapy in a 60-year-old man, with sustained remission after tapering to once-daily application [13]. In contrast, a topical JAK inhibitor produced no improvement in one patient in the cohort of Ziebart et al. [4], and our patient had not responded to compounded topical tofacitinib 2%.

The response in our patient cannot be attributed to topical ruxolitinib alone. Improvement was first noted after approximately three months of methotrexate and only four weeks of topical ruxolitinib, the greatest improvement occurred during combined therapy, and a mild flare followed the discontinuation of methotrexate. Methotrexate 20 mg weekly produced moderate improvement in one patient and no response in another in the cohort of Ziebart et al. [4]. Spontaneous fluctuation, and repigmentation of post-inflammatory hypopigmentation as inflammation subsided, may also have contributed. Nevertheless, she reported continued repigmentation for 18 months on topical ruxolitinib alone, after 16 previous treatments had failed, which suggests that topical ruxolitinib contributed to disease control.

Dermoscopy descriptions of FDD are limited to two recent reports of erythematous lesions, neither of which states the patient's phototype. Murtas et al. described a brownish-red background with white scale, follicular plugs, red dots, and polygonal vessels [8], and Zygadlo et al. described fine scale over dilated vessels over large yellow dots corresponding to enlarged follicular ostia [18]. In a review of dermoscopic patterns of common inflammatory dermatoses that present as erythematous macules or plaques on the face, none of the described findings resembled those observed in our case. Seborrheic dermatitis is characterized by dotted vessels in a patchy distribution with yellow scales; erythematotelangiectatic rosacea demonstrates linear vessels forming polygonal networks; and discoid lupus shows perifollicular whitish halos, follicular keratotic plugs, white scales, and linear branching vessels [23]. In contrast, stable vitiligo is characterized by white, structureless areas, loss of the pigment network,

perifollicular pigmentation, and leukotrichia (depigmented hairs), whereas active vitiligo shows poorly defined borders, an altered pigment network, and starburst, comet-tail, and polka-dot appearances [24].

In this patient, the dermoscopic features of the FDD lesions showed hypopigmented patches with faint central erythema, fine patchy scales, and preserved pigmented vellus hairs. Pigmented hairs may help distinguish FDD from stable vitiligo, but this sign should be interpreted with caution. In a review of 15 dermoscopy studies, leukotrichia was reported inconsistently in active, stable, and repigmenting vitiligo [24]. The hypopigmented, rather than depigmented appearance, with scale and erythema, is an additional clue. Other reports have described dermoscopic features of FDD, including vascular changes, white scaling, and follicular abnormalities [8,18]. However, to our knowledge, dermoscopic findings in a predominantly hypopigmented presentation of FDD in darker skin have not previously been reported.

Future studies with larger case series will be needed to validate these observations and further delineate the dermoscopic spectrum of FDD.

4. Conclusions

This case highlights FDD in a patient with Fitzpatrick type IV skin, initially suspected of having vitiligo, emphasizing the need for clinicians to consider FDD in the differential diagnosis of treatment-resistant facial hypopigmentation in darker skin. Based on assessing clinical, histopathological, and dermoscopy features, as well as conducting patch testing and observing the lack of response to conventional treatments, we were able to rule out other potential dermatoses, such as seborrheic dermatitis, psoriasis, and vitiligo. Our patient improved with oral methotrexate combined with topical ruxolitinib 1.5% cream and continued to repigment on topical ruxolitinib alone. Together with the recent report by Garcia et al. [13], this case suggests that topical JAK inhibition merits further study in FDD.

Author Contributions

E.Y.C.: conceptualization, methodology, investigation, resources, data curation, writing—original draft preparation, writing—review and editing, Supervision, Project administration. S.A.Q.: methodology, investigation, data curation, writing—original draft preparation, writing—review and editing. A.F.: investigation, resources, writing—original draft preparation, writing—review and editing. K.L.: investigation, resources, writing—original draft preparation, writing—review and editing. All authors have read and agreed to the published version of the manuscript.

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Institutional Review Board Statement

Ethical approval was not sought for the present study because a descriptive report of a single clinical case does not constitute research requiring research ethics board review under the Tri-Council Policy Statement: Ethical Conduct for Research Involving Humans (TCPS 2), as applied by the University of Alberta Research Ethics Office.

Informed Consent Statement

Written informed consent for publication of this case report, including the clinical images, was obtained from the patient.

Data Availability Statement

All data supporting the findings of this publication are available within this article.

Conflicts of Interest

Samples of topical ruxolitinib 1.5% cream were provided to the patient by the manufacturer, Incyte Biosciences Canada; the manufacturer had no role in the preparation of this manuscript. The authors declare that they have no conflicts of interest.

Use of AI and AI-Assisted Technologies

During the preparation of this work, the authors used Claude (Anthropic; Sonnet 5.5) for language editing to improve clarity, readability, and scholarly tone. Following the use of this tool, the authors carefully reviewed and edited the content as necessary and take full responsibility for the accuracy, integrity, and content of the published article. The authors confirm that the use of this AI tool was limited to language editing and did not replace the authors' intellectual contributions, scientific judgment, or responsibility for the research.

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