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CASE REPORT

Successful Treatment of Eosinophilic Pustular Folliculitis with Secondary Follicular Mucin Deposition Using Indomethacin: An Atypical Case and Literature Review

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Abstract: This case report describes a 45-year-old Asian female who presented with pruritic plaques on the face and back for 4 months. Upon examination, the diagnosis of eosinophilic pustular folliculitis with secondary follicular mucinosis was made. The uniqueness of this case lies in the simultaneous occurrence of two relatively uncommon dermatopathological alterations. Oral indomethacin was administered with favorable outcomes, providing valuable clinical insights for diagnosis and treatment.

Keywords: eosinophilic pustular folliculitis, follicular mucin deposition, indomethacin

Introduction

Eosinophilic Pustular Folliculitis (EPF) is a rare inflammatory skin disorder first described by Ofuji in 1970.[1] It is characterized by recurrent erythematous follicular papules and pustules that may coalesce into annular plaques. EPF can be classified into several types, including the classic, HIV-associated, and infantile forms. Commonly affected areas include the face, upper limbs, and upper back. The classic form of EPF typically occurs in Japan, predominantly affecting males. Histologically, EPF is characterized by an infiltration of eosinophils around the folliculopilosebaceous unit, forming microabscesses.[2] Studies have reported that mucin deposition in the hair follicles is primarily observed in HIV-associated EPF, with a relatively lower incidence in the classic form.[3] Current evidence indicates that cases of eosinophilic pustular folliculitis (EPF) with secondary follicular mucin deposition are predominantly documented as case reports and small case series. Since its initial description, fewer than 100 confirmed cases have been reported in the global literature, demonstrating a distinct ethnic predisposition—with a predilection for Asian populations (particularly Japanese and East Asian ethnic groups) and a male-to-female ratio of approximately 3:1.[2,3] Therefore, this report presents an atypical and rare case of classic EPF in an HIV-negative Asian female, accompanied by secondary follicular mucin deposition, offering clinical insights for the diagnosis and management of similar cases. A review of the literature on eosinophilic pustular folliculitis with secondary follicular mucin deposition is also provided.

Clinical Presentation

This case involves a 45-year-old female who presented with pruritic plaques on her face and back for 4 months. Physical examination revealed well-defined, scaly, annular plaques with scattered follicular erosive papules, ranging from millet to soybean size, over erythematous areas (Figure 1A and B). The patient denied any history of relevant medication use but

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Figure 1 (**A** and **B**) Well-defined, scaly, annular plaques are visible on the face and back, with scattered follicular erosive papules ranging from millet to soybean size over the erythematous areas. (**C** and **D**) During the regular administration of indomethacin, the lesions were effectively controlled.

reported having a history of animal exposure. The initial diagnosis included 1. Majocchi's granuloma? 2. Cutaneous lymphocytic infiltration? To further clarify the diagnosis, a skin biopsy was performed. Histopathological examination revealed mild epidermal spongiosis and follicular epidermal spongiosis. There was noticeable mucin within the hair follicles, and eosinophilic granulocytes were aggregated around the hair follicles and follicular openings, as well as in the superficial and mid-dermal vascular regions. No atypical lymphocytes were observed (Figure 2A–D). Fungal examination was negative, HIV testing was negative, and all blood parameters were within normal limits, with eosinophils comprising 15.3%. Clonality evaluation using T-cell receptor rearrangement polymerase chain reaction (PCR) revealed no clonality peaks.

Considering the above findings, the diagnosis of eosinophilic pustular folliculitis with secondary follicular mucin deposition was established. The patient initially received oral traditional Chinese medicine, but the response was inadequate. Subsequently, oral indomethacin was prescribed at a dosage of 50 mg once daily. Within one week, the symptoms completely resolved. The dosage was then reduced to 25 mg once a week, and during the regular administration of indomethacin, the disease was effectively controlled (Figure 2D).

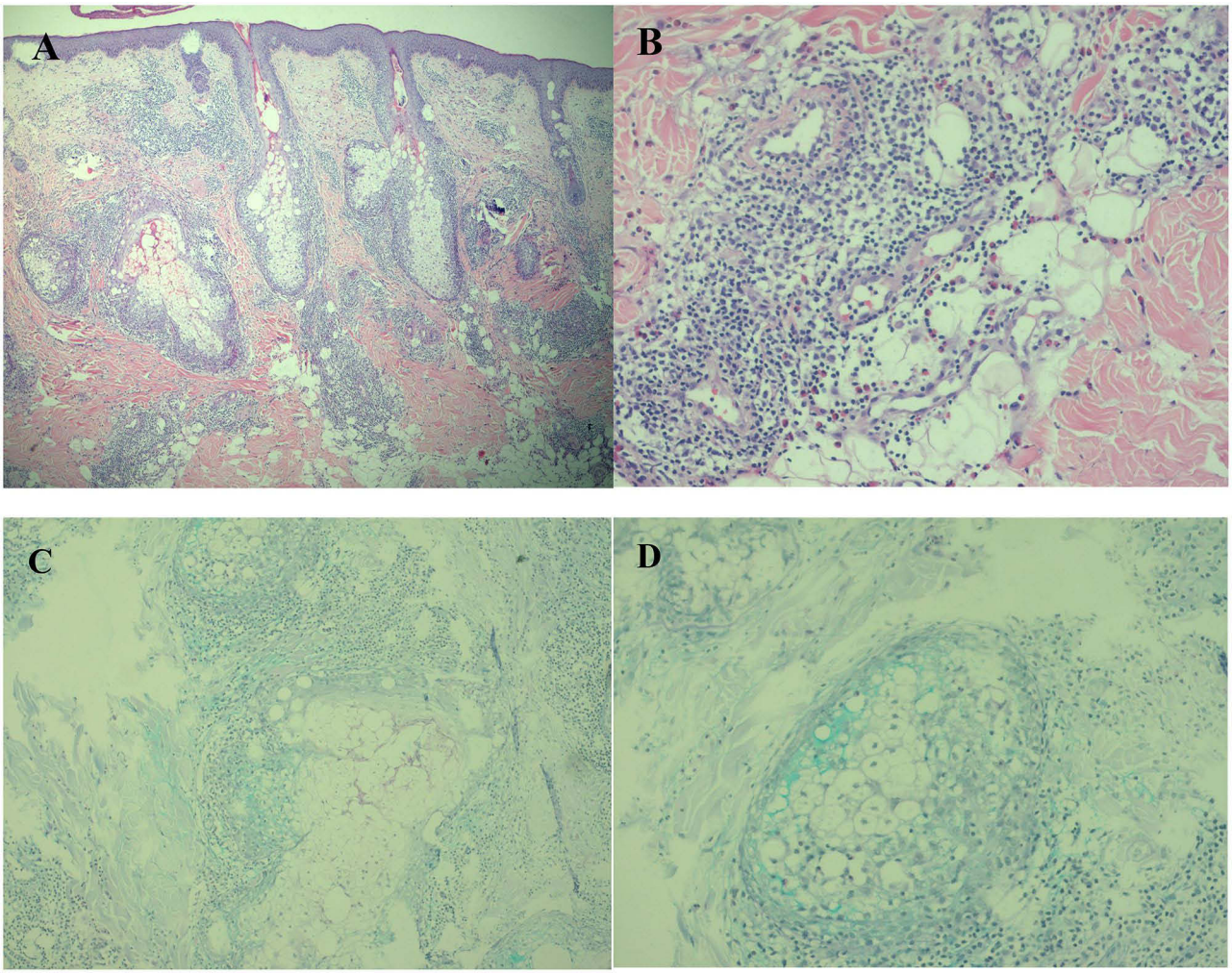

Figure 2 (**A** and **B**) Epidermal spongiosis and follicular epithelial spongiosis were observed, with eosinophilic granulocyte accumulation around the follicular epithelium, follicular openings, and perivascular areas in the superficial and mid-dermal layers. No atypical lymphocytes were found (HE×100x, HE×400x). (**C** and **D**) There was prominent mucin deposition within the hair follicles (HE×100x, HE×200x).

Discussion

Eosinophilic pustular folliculitis (EPF) is a skin disorder characterized by distinctive clinical and pathological features, and the concurrent presence of secondary follicular mucin deposition, in this case, is even rare. From an etiological perspective, the exact pathogenesis of EPF remains unclear, though it is believed to be associated with immune dysregulation.[4] In this case, no obvious triggering factors were identified in the patient's medical history or through related tests. However, the patient's history of animal exposure could be a contributing factor, suggesting that environmental factors play a role in the onset and progression of the disease.

Pathologically, this case displayed the typical features of EPF, such as eosinophilic infiltration within the follicular epithelium, along with prominent mucin deposition within the hair follicles. This combination of pathological changes added complexity to the diagnosis. During the diagnostic process, it was necessary to differentiate EPF from other similar conditions, such as Majocchi's granuloma and cutaneous lymphocytic infiltration. The initial differential diagnosis considered these conditions; however, the final diagnosis was confirmed through pathological examination.

In terms of treatment, this case did not respond to oral traditional Chinese medicine, but oral indomethacin yielded significant results. Indomethacin has been shown to be effective in the treatment of EPF, and its mechanism of action is likely related to the inhibition of inflammatory mediators. Furthermore, the prognosis of this case was favorable, with

effective disease control achieved during regular indomethacin administration. This provides a valuable reference for the treatment of similar cases of EPF with secondary follicular mucinosis.

The association between classic EPF and follicular mucin deposition has only been occasionally observed. Moreover, outside of Japan, reported cases of EPF with concomitant follicular mucin deposition have primarily occurred in HIV-infected individuals (Table 1).[5–29] However, there is also a report of a 19-year-old HIV-negative Caucasian female living

Table 1 Literature Review of Age, Gender, History of AIDS/Tumor, Follicular Mucin Deposition and Treatment in Patients with Eosinophilic Pustular Folliculitis

Age	Gender	HIV/Oncology History	Follicular Mucin Deposition	Treatment
71 years	Male	Chronic Lymphocytic Leukemia	Yes	10 days of gradually reduced prednisone, topical corticosteroids
70 years	Male	Chronic Lymphocytic Leukemia	No	Topical corticosteroids, oral hydroxyzine
34 years	Female	No	No	Topical corticosteroids, after pathological confirmation, use of tacrolimus or oral indomethacin
20–50 years (avg. 31.2 years)	13 cases (4 male, 9 female)	1 hIV positive, HIV patients with follicular mucin deposition	Yes	Varies: corticosteroids, indomethacin
7–47 years (avg. 31 years)	3 female, 4 male	No	No	Varies: corticosteroids, dapsone, naproxen
22 years	Male	No	No	Da Chai Hu Tang combined with Liang Xue Wu Hua Tang
44 years	Male	No	No	Oral doxycycline, topical compound triamcinolone
32 years	Male	No	No	Not mentioned (treated in another hospital, related to light exposure)
30–40 years	Predominantly male (4.8:1)	2 hIV positive, HIV patients with follicular mucin deposition	Yes	Varies: ibuprofen, dapsone, minocycline, cetirizine, tacrolimus, itraconazole, PUVA, acitretin, interferon α-2b, interferon γ, cyclosporine A
35 years (avg.)	8 male, 15 female	3 hIV positive, HIV patients with follicular mucin deposition	Yes	Oral indomethacin (most patients effective), a few use dapsone
39 years	Female	No	Not mentioned	Initially oral tranilast, later switched to oral indomethacin due to stomach pain, continued tranilast
40 years	Male	No	Not mentioned	Oral indomethacin and topical tacrolimus
29 years	Male	Yes	Not mentioned	Resistant to multiple oral and topical medications, later resolved with HIV treatment, topical tacrolimus
56 years	Female	No	Not mentioned	Oral indomethacin
60 years	Female	No	Not mentioned	Oral indomethacin and topical tacrolimus
26 years	Male	No	Not mentioned	Oral indomethacin, topical tacrolimus ointment
29–61 years	6 male, 2 female	1 hIV positive, HIV patients with follicular mucin deposition	Yes	Oral indomethacin, cetirizine, topical tacrolimus ointment
36 years	Male	No	Not mentioned	Minocycline, Tripterygium glycosides

(*Continued*)

Table 1 (Continued).

Age	Gender	HIV/Oncology History	Follicular Mucin Deposition	Treatment
64 years	Male	No	Not mentioned	Hydroxychloroquine, ketotifen, combined with topical triamcinolone and econazole (back), polysaccharide mucopolysaccharide cream (face), later switched to methylprednisolone sodium succinate IV and oral
32 years	Male	No	Not mentioned	Oral sustained-release indomethacin, thymosin, epitisine, combined with heat-clearing, dampness-dispelling, detoxifying traditional Chinese medicine, local topical dinanide cream, San Huang wash etc.
26 years	Male	No	Not mentioned	Oral indomethacin, 0.03% tacrolimus ointment topical
18–74 years	Male-to-female ratio 3:1	2 hIV positive, HIV patients with follicular mucin deposition	Yes	Oral indomethacin, triamcinolone, hydroxychloroquine, topical corticosteroid ointment
65 years	Male	No	Not mentioned	Oral indomethacin, topical 0.1% tacrolimus ointment
24 years	Male	No	Not mentioned	Oral acitretin capsules, Tripterygium glycosides, compound glycyrrhizin tablets, epitisine tablets; narrowband UVB therapy, topical 0.1% tacrolimus (face), Halobetasol ointment (back and limbs)

in the United States presenting with EPF accompanied by secondary follicular mucin deposition.[4] In HIV-negative patients, histological features suggest that the combination of EPF and follicular mucin deposition makes this case atypical. Our case describes a unique presentation of EPF with secondary follicular mucin deposition in a 45-year-old HIV-negative Asian female. EPF with follicular mucin deposition is relatively rare in non-immunosuppressed adults, and its rarity makes the diagnosis of this patient particularly challenging.

The etiology of EPF with secondary follicular mucin deposition is multifactorial. Some cases of EPF with follicular mucin deposition have been associated with HIV and hematologic malignancies. In these disease states, alterations in the immune system and the internal environment of the body lead to immune dysregulation, resulting in an imbalance of the immune modulation mechanisms that typically target hair follicles.[17] Additionally, tumor cells may release various cytokines or other bioactive substances, which can affect the physiological function of follicular epithelial cells and promote mucin deposition.[18]

Dysregulation of immunoglobulins exhibits a bidirectional pathological association with hematologic malignancies. In multiple myeloma and chronic lymphocytic leukemia, the clonal proliferation of monoclonal immunoglobulins (eg, IgG or IgM) is frequently accompanied by suppression of normal polyclonal immunoglobulins (particularly IgA/IgG subtypes), creating an "immune paralysis" microenvironment. This imbalance may increase susceptibility to opportunistic infections and facilitate tumor immune evasion.[19] Notably, in some cases of eosinophilic pustular folliculitis (EPF) occurring in seemingly "immunocompetent" individuals, serum total immunoglobulin levels remain within reference ranges, yet a significant association with selective IgA deficiency (IgA <0.07 g/L) has been identified. As a key defense protein for mucosal barriers, IgA deficiency may permit skin commensal microbiota to penetrate follicular epithelia, triggering a Th2-polarized inflammatory response. This localized immune dysregulation explains why EPF patients exhibit IL-5-driven eosinophilic infiltration despite normal peripheral CD4+/CD8+ T-cell ratios.[20] Recent studies further elucidate unique molecular mechanisms underlying IgA deficiency-associated EPF: Mucocutaneous axis dysregulation: Intestinal IgA deficiency leads to microbial translocation, activating skin-resident group 2 innate lymphoid cells (ILC2s) via the "gut-

skin axis", thereby promoting thymic stromal lymphopoietin (TSLP)-mediated eosinophil chemotaxis.[21] Complement alternative pathway activation: IgA deficiency results in abnormal C3/C5 complement deposition around hair follicles, directly stimulating eosinophil degranulation through C5a-C5aR1 signaling.[22] These findings underscore the necessity for routine IgA subclass screening and mucosal barrier integrity assessment in "immunocompetent" EPF patients.

EPF must be differentiated from diseases such as follicular mucinosis (FM), eosinophilic folliculitis (EF), and cutaneous T-cell lymphoma (CTCL). In FM, the primary feature is the accumulation of mucin within the hair follicles, whereas EPF is characterized by eosinophilic infiltration around the follicular structures.[23] EF typically presents with prominent eosinophilic infiltration, while EPF with secondary follicular mucin deposition generally shows the accumulation of a translucent, gel-like substance within the follicular lumen, with eosinophils or other types of immune cells as the predominant inflammatory cells.[24] CTCL is primarily characterized by T-cell infiltration, whereas the pathology of EPF with secondary follicular mucin deposition is centered on follicular damage and secondary mucin deposition. Immunohistochemical staining can aid in differentiation, as CTCL typically demonstrates CD4-positive T-cell infiltration.[25]

In cases reported in relevant studies, some patients with chronic lymphocytic leukemia (CLL) have developed EPF accompanied by follicular mucin deposition. The pathological features in these cases may exhibit characteristics of both eosinophilic folliculitis (EF) and eosinophilic dermal hyperplasia (EDHM), presenting as inflammation involving superficial and deep lymphocytes and eosinophils, affecting perivascular, perifollicular, and follicular centers, with or without follicular mucin deposition.[26,29] Eosinophilic pustular folliculitis with mucin deposition associated with hematologic malignancies may represent a disease spectrum, ranging from simple EF to EDHM, as well as cases with follicular mucin deposition. This spectrum might reflect a continuous process. Within this spectrum, different disease stages may present distinct clinical and pathological features, yet there are overlapping and interconnected elements.[28]

The co-occurrence of eosinophilic pustular folliculitis (EPF) and follicular mucinosis in HIV-negative patients without malignancy suggests a distinct and complex immunopathological pathway that differs from both classical EPF and mycosis fungoides-associated mucinosis. While HIV-associated EPF is clearly linked to TH2-mediated immune dysregulation and IL-5 overexpression, the present case highlights alternative potential mechanisms: Dysregulated Eosinophil-Chemokine Axis: Non-HIV-associated EPF with mucin deposition may reflect localized overproduction of eotaxin-3 and thymic stromal lymphopoietin (TSLP) by keratinocytes, a mechanism independent of systemic TH2 immune activation.[30] Mucin deposition likely represents a consequence of chronic eosinophilic inflammation, wherein eosinophil-derived major basic protein (MBP) disrupts follicular keratinocyte adhesion, exposing hair follicle stem cells to a TGF-β-mediated mucinogenic microenvironment.[31] Follicular Epithelial Stress Response: Histological overlap with pityriasis rubra pilaris (PRP)-like mucinosis suggests potential HSP70 overexpression in the outer root sheath, which may simultaneously induce eosinophil chemotaxis and mucin synthesis via CD44-hyaluronic acid interactions. Genetic Predisposition: The predominance among Asian populations implies that HLA-DRB1*04 alleles or filaggrin (FLG) loss-of-function mutations may create a permissive microenvironment facilitating concurrent eosinophilic infiltration and mucin deposition.[32] Critically, the HIV-negative/malignancy-free status excludes lymphokine-driven mechanisms (eg, IL-4/IL-13 in cutaneous T-cell lymphoma), instead implicating primary follicular unit dysfunction—where mucin deposition may represent an "exhaustion" phenomenon following recurrent eosinophilic assaults, rather than neoplastic or infectious triggers. This perspective aligns with reports of indomethacin-responsive mucin-positive EPF cases, as prostaglandin D2 inhibition concurrently blocks eosinophil migration and mucin production.[33]

The treatment of eosinophilic pustular folliculitis (EPF) includes pharmacological and physical therapies. Indomethacin is the first-line treatment for EPF, demonstrating a high efficacy rate, followed by corticosteroids and topical tacrolimus ointment. Other medications, such as cyclosporine, dapsone, hydroxychloroquine, sulfasalazine, and minocycline, may also offer benefits in some patients.[27] For instance, oral cyclosporine may be particularly effective in patients who have not responded well to other treatments. Oral dapsone or sulfasalazine might also be beneficial in some cases. Antihistamines such as loratadine and cetirizine can help alleviate pruritus. In terms of physical therapy, narrowband ultraviolet B (NB-UVB) phototherapy has been shown to penetrate the dermis, suppressing inflammatory cells and proliferating endothelial cells, thereby improving symptoms in patients. For patients who do not respond to conventional treatments, physical therapies like ultraviolet light therapy may offer an effective alternative. In cases associated with hematologic malignancies, treatment may also need to address the underlying primary disease.[34] For

example, in patients with chronic lymphocytic leukemia (CLL), chemotherapy or targeted therapies might be required. During the treatment of the hematologic disorder, close monitoring of the skin symptoms for improvement is essential.[35]

The prognosis of simple EPF with follicular mucin deposition is generally good, and symptoms can be alleviated with appropriate treatment. However, when associated with hematologic malignancies, the prognosis is influenced by the underlying hematologic disease. Some studies have found that while skin symptoms may improve with treatment if the hematologic malignancy progresses, the overall prognosis of the patient remains poor.[36]

Conclusion

Eosinophilic pustular folliculitis with secondary follicular mucin deposition is a complex dermatological condition, and its diagnosis requires a comprehensive evaluation of both clinical and pathological features. Treatment and prognosis must be assessed and managed according to the specific circumstances. In this case, the patient was HIV-negative yet developed follicular mucin deposition, which, due to its rarity, made the diagnosis particularly challenging.

Ethic Statement

The patient has granted permission for the images to be published along with the case report, and The Fifth People's Hospital of Hainan Province has given its approval for the case details to be disclosed after obtaining approval from its own Ethics Committee.

Consent Statement

Informed consent was provided by the patient for publication of the case.

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Disclosure

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