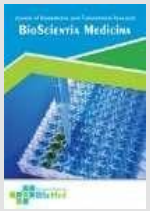
eISSN (Online): 2598-0580



Bioscientia Medicina: Journal of Biomedicine & Translational Research

Journal Homepage: www.bioscmed.com

Eosinophilic Pustular Folliculitis Mimicking Varicella: A Diagnostic Challenge in an Adolescent Patient

Farisa Mutahallia[1], Triasari Oktavriana[2*], Novan Adi Setyawan[3], Aizawanda Rizqi Eiffellia[2], Arista Kautsar Rahman[2]

[1]General Practitioner, Universitas Sebelas Maret Hospital, Sukoharjo, Indonesia

[2]Department of Dermatology and Venereology, Faculty of Medicine, Universitas Sebelas Maret, Surakarta, Indonesia

[3]Department of Anatomical Pathology, Faculty of Medicine, Universitas Sebelas Maret, Surakarta, Indonesia

ARTICLE INFO

Keywords:
Adolescent
Eosinophil
Ofuji disease
Pustular folliculitis
Varicella mimicry

***Corresponding author:**
Triasari Oktavriana

E-mail address:
triasari_oktavriana@staff.uns.ac.id

All authors have reviewed and approved the final version of the manuscript.

https://doi.org/10.37275/bsm.v10i4.1560

A B S T R A C T

Background: Eosinophilic pustular folliculitis (EPF), or Ofuji disease, is a rare non-infectious inflammatory dermatosis typically affecting middle-aged adults. Its clinical resemblance to infectious exanthems often leads to significant diagnostic delays, particularly in atypical age groups. **Case presentation:** A 15-year-old male presented with a one-month history of progressive erythematous papules, pustules, and crusted erosions distributed across the face, trunk, and extremities. The patient was initially misdiagnosed with varicella and treated with acyclovir without clinical response. Physical examination and Gram staining ruled out primary bacterial infections. Laboratory investigations revealed significant peripheral eosinophilia (12.3%). A 5-mm punch biopsy confirmed the diagnosis, showing dense eosinophilic and neutrophilic infiltration of the hair follicles and peri-adnexal structures. Management with systemic and topical corticosteroids led to rapid resolution of lesions and normalization of eosinophil levels. **Conclusion:** This case underscores the necessity of considering EPF in the differential diagnosis of persistent papulopustular eruptions in adolescents. Early histopathological intervention is critical to prevent unnecessary antimicrobial therapy and ensure targeted anti-inflammatory treatment.

1. Introduction

Eosinophilic pustular folliculitis (EPF) represents a rare and clinically distinct chronic inflammatory dermatosis of unknown etiology that primarily targets the pilosebaceous units. Since its inaugural description by Shigeo Ofuji and colleagues in 1970, the condition—frequently referred to as Ofuji disease—has remained a subject of intense dermatological scrutiny due to its unique histopathological signature and its tendency to mimic more common cutaneous infections.[1] The hallmark of the disease is the presence of sterile, pruritic follicular papules and pustules that exhibit a characteristic eosinophil-rich infiltration upon microscopic examination. While the precise pathogenesis of EPF remains elusive, current hypotheses favor a multifactorial origin involving complex immune dysregulation, potentially triggered by various antigenic stimuli that stimulate the recruitment of eosinophils to the hair follicle.[2]

Historically, the clinical landscape of EPF has been divided into three principal variants, each defined by

its demographic predilection and underlying immunological status: classic EPF, immunosuppression-associated EPF (IS-EPF), and infantile EPF (I-EPF).[3] Classic EPF, the original variant described in Japan, typically manifests in the third and fourth decades of life and shows a significant male predilection, with a reported male-to-female ratio as high as 5:1. This variant is characterized by recurrent clusters of follicular papulopustules that often arrange themselves in arcuate or annular plaques with central clearing and centrifugal extension, favoring the face, trunk, and upper extremities. In contrast, the IS-EPF variant is most frequently encountered in the context of HIV infection or hematological malignancies, often presenting as more disseminated, follicular erythematous papules. The infantile variant, I-EPF, typically emerges within the first year of life, is frequently localized to the scalp, and follows a self-limiting but recurrent course.[4]

Despite this established classification system, EPF remains a significant diagnostic chameleon in clinical practice. The morphology of the lesions, which can range from discrete vesicles to confluent pustules and crusted erosions, frequently overlaps with a broad spectrum of dermatological conditions.[5] In many instances, the primary clinical suspicion leans toward infectious etiologies, including acne vulgaris, rosacea, fungal infections such as trichophytosis, and parasitic infestations like scabies. Perhaps the most challenging diagnostic hurdle, however, is the resemblance of EPF to viral exanthems. The acute appearance of papulopustular eruptions on the face and trunk in younger patients often leads clinicians toward a diagnosis of varicella-zoster virus (VZV) infection. This mimicry is particularly deceptive because the vesicular appearance of sterile EPF pustules can mirror the early stages of varicella, leading to the inappropriate administration of antiviral therapies and delayed definitive treatment.[6]

The emergence of the classic variant of EPF in an adolescent population represents an exceedingly rare and under-documented phenomenon. While I-EPF is well-recognized in the pediatric literature, the bridge between infancy and the typical third-decade onset of Ofuji disease is sparsely populated with case reports.[7] Adolescent patients often present a unique diagnostic dilemma; they are in a transitional life stage where common conditions like acne vulgaris or viral exanthems are prevalent, yet they are theoretically susceptible to adult-onset inflammatory disorders. When an adolescent presents with a widespread, crusted, papulopustular eruption, the clinician is frequently pressured to rule out infectious causes first, often leading to a cycle of failed antibiotic and antiviral trials.

The diagnostic gold standard remains the integration of clinical morphology with histopathological evidence. A skin biopsy typically reveals the defining feature of the disease: a dense, eosinophil-rich inflammatory infiltrate localized around the hair follicles and sebaceous glands. This is often accompanied by epidermal spongiosis and follicular mucinosis. Furthermore, laboratory investigations often reveal peripheral eosinophilia and elevated serum immunoglobulin E (IgE) levels, which may serve as dynamic markers of disease activity. The presence of these markers, particularly the peripheral eosinophil count, can be instrumental in pivoting the diagnosis away from infection and toward a sterile inflammatory process.[8]

Understanding the mechanisms of eosinophil recruitment is essential to appreciating the inflammatory nature of EPF. It is proposed that the expression of adhesion molecules, such as intercellular adhesion molecule 1 (ICAM-1) and vascular cell adhesion molecule 1 (VCAM-1), facilitates the migration of inflammatory cells to the follicular site.[9] Furthermore, lipid mediators like Prostaglandin D2 (PGD2) are believed to play a central role by binding to chemoattractant receptors on eosinophils, thereby driving the recruitment and activation of these cells within the pilosebaceous unit. The success of treatments such as indomethacin, which inhibits prostaglandin synthesis, or corticosteroids, which broadly suppress these inflammatory pathways,

reinforces this immunological framework.[10]

The aim of this study is to provide a comprehensive analysis of adolescent-onset EPF, focusing on the profound diagnostic challenges posed by its close clinical resemblance to varicella. By detailing a case that navigated through failed antiviral and antibiotic therapies, we intend to illuminate the mimicry that often delays the diagnosis of this rare condition in younger populations. The novelty of this work lies in the meticulous documentation of the clinical-pathological correlation in a non-classic age group, specifically highlighting the utility of peripheral eosinophilia as a primary diagnostic indicator and the rapid therapeutic response to targeted anti-inflammatory intervention. Through this analysis, we emphasize the necessity of early histopathological investigation in persistent adolescent papulopustular eruptions to prevent unnecessary medication use and ensure timely definitive care.

2. Case Presentation

Written informed consent was obtained from the patient's legal guardian for the publication of this case report and any accompanying clinical images and laboratory data. The identity of the patient has been anonymized to ensure privacy and confidentiality in accordance with the Declaration of Helsinki.

A 15-year-old male of Asian descent was admitted to the dermatology department of Sebelas Maret University Hospital with a complex and progressive skin eruption that had persisted for approximately one month. The patient's clinical course began roughly four weeks prior to his initial consultation, at which time he observed a solitary, discrete erythematous papule on his right arm (Table 1). This primary lesion rapidly evolved into a pustule, and within a matter of days, similar lesions began to manifest across multiple anatomical regions, including the face, trunk, and extremities. Despite the extensive nature of the cutaneous involvement, the patient remained systemically well, specifically denying any history of fever, chills, night sweats, or unintentional weight loss. Furthermore, he reported no symptoms of arthralgia, myalgia, or respiratory distress.

The diagnostic timeline prior to hospitalization was marked by several clinical attempts to manage what was initially suspected to be a common infectious process. During the second week of the illness, the patient sought care at a primary clinic where he was prescribed a three-day course of amoxicillin, 500 mg administered three times daily, under the suspicion of bacterial folliculitis. This antibiotic intervention yielded no observable improvement in the lesions, which continued to proliferate in number and size.

By the third week of the illness, the clinical morphology changed, leading to a secondary diagnosis of varicella-zoster infection. At this juncture, the patient was initiated on a therapeutic regimen consisting of topical acyclovir applied three times daily, oral cetirizine 10 mg twice daily for symptomatic relief, and oral paracetamol 500 mg three times daily as a precautionary measure. Despite these efforts, the patient's condition continued to deteriorate, with the lesions becoming more confluent and crusted, eventually necessitating hospitalization for further diagnostic evaluation and specialized management. The patient's past medical history was unremarkable, with no documented history of atopy, asthma, or allergic rhinitis. He denied any known drug allergies and confirmed that no other family members or household contacts exhibited similar dermatological symptoms.

Upon admission, the patient appeared clinically stable and in no acute distress. A comprehensive systemic evaluation revealed that the cardiovascular, respiratory, and abdominal systems were within normal limits. However, a detailed dermatological examination uncovered an extensive and multi-morphic eruption. The lesions consisted of multiple erythematous papules and sterile-appearing pustules, some of which had coalesced into larger, irregularly shaped, circumscribed plaques. These lesions exhibited both dry and wet characteristics, with dimensions ranging from 2 mm by 2 mm up to larger configurations of 2 cm by 3 cm. A prominent feature of the presentation was the presence of significant

yellowish crusting and erosions, particularly concentrated on the facial region. While the face, back, trunk, arms, and legs were extensively involved, a critical diagnostic finding was the total absence of lesions on the palms of the hands and the soles of the feet. This distribution provided an essential clue in the differential diagnosis, as it helped to narrow the focus toward folliculocentric inflammatory disorders.

The diagnostic workup was structured to investigate the systemic involvement of the inflammatory process and to rule out potential underlying immunosuppression or occult infection. Initial laboratory investigations revealed a striking abnormality in the complete blood count. While the total leukocyte count was within the expected range, a peripheral blood smear demonstrated significant eosinophilia, with the eosinophil count measured at 12.3 percent, substantially exceeding the normal reference range of 0.0 to 4.0 percent. This finding was instrumental in shifting the clinical suspicion toward a diagnosis of eosinophilic pustular folliculitis (EPF). To investigate potential triggers or associated conditions, an HIV screening was performed and returned negative. Furthermore, to definitively address the possibility of a secondary or primary bacterial infection, a Gram stain was conducted on pus aspirated from a fresh lesion. The microscopic analysis revealed fewer than 10 solitary gram-positive cocci per visual field, a finding interpreted as representing normal skin flora or incidental colonization rather than an active infectious process.

Table 1. Summary of Clinical Findings on Admission

PATIENT PROFILE & CLINICAL HISTORY	
Age and Gender	15-year-old male
Duration of Symptoms	Approximately one month prior to admission
Initial Presentation	Solitary papule on the right arm evolving into a pustule
Systemic Symptoms	Absence of fever, arthralgia, or weight loss
Previous Therapy	Failed trials of oral amoxicillin and topical acyclovir
DERMATOLOGICAL EXAMINATION	
Morphology	Erythematous papules, sterile pustules, and confluent crusted erosions
Primary Distribution	Face, back, trunk, and upper/lower extremities
Specific Sparing	Negative for lesions on palms and soles
DIAGNOSTIC & LABORATORY RESULTS	
Peripheral Eosinophils	**12.3%** (Reference: 0.0 – 4.0%)
HIV Serology	NON-REACTIVE
Gram Stain of Pus	<10 solitary Gram-positive cocci per visual field
Histopathological Diagnosis	Epidermal spongiosis with dense peri-adnexal eosinophilic and neutrophilic infiltration

The definitive diagnosis was sought through a 5-mm punch biopsy obtained from a newly developed papule on the patient's right forearm, which included a margin of healthy-appearing perilesional skin. The biopsy specimen was processed for routine hematoxylin and eosin staining. The histopathological examination provided the definitive evidence required for a diagnosis of EPF. The epidermis exhibited areas of spongiosis, accompanied by mild hyperkeratosis and irregular acanthosis. Within the dermis, a dense and focal inflammatory cell infiltrate was observed, primarily localized in a peri-adnexal and perivascular distribution. This infiltrate was characterized by a predominance of eosinophils, along with a secondary population of lymphocytes, neutrophils, and macrophages. Crucially, the presence of numerous eosinophils within the follicular and perivascular spaces confirmed the sterile, inflammatory nature of the dermatosis.

Based on the integrated clinical, laboratory, and histopathological findings, the patient was diagnosed with adolescent-onset classic eosinophilic pustular folliculitis (Table 2). Given the extent of the lesions and the failure of previous outpatient treatments, he was admitted for inpatient care and initiated on a multi-modal anti-inflammatory regimen. The inpatient management protocol included: (1) Systemic Therapy: Intravenous methylprednisolone was administered at a dose of 31.25 mg twice daily to rapidly suppress the systemic inflammatory response. Additionally, intravenous diphenhydramine, 10 mg once daily, was provided as an adjunctive antihistamine therapy; (2) Topical Therapy: Mometasone furoate 0.1 percent was applied twice daily to the facial lesions, while clobetasol propionate 0.05 percent was used for lesions on the trunk and extremities; (3) Supportive Care: Saline compresses were applied twice daily to facilitate the debridement of crusted erosions and promote healing. Topical fusidic acid 2 percent was also utilized twice daily as a prophylactic measure against secondary bacterial infection of the eroded skin. The response to this targeted anti-inflammatory intervention was rapid and substantial. Within four days of initiating therapy, the inflammatory papules and pustules began to subside, and the peripheral eosinophil count demonstrated a dramatic normalization, falling from 12.3 percent to 0.1 percent.

Upon discharge, the patient was transitioned to oral medications to maintain clinical remission. This included oral methylprednisolone 16 mg twice daily, ranitidine 50 mg twice daily for gastric protection, and a five-day course of cefixime 100 mg twice daily. He was instructed to continue the topical corticosteroid regimen and return for regular follow-up. At the three-week follow-up visit, the patient showed remarkable clinical improvement. The previously active lesions had resolved, leaving only residual post-inflammatory hyperpigmentation, and no new papules or pustules were observed. The rapid therapeutic success underscored the accuracy of the diagnosis and the importance of systemic corticosteroids in managing severe cases of adolescent-onset EPF.

3. Discussion

The clinical presentation of eosinophilic pustular folliculitis (EPF), commonly referred to as Ofuji disease, remains a subject of intense dermatological inquiry due to its rarity and the complexity of its underlying biological mechanisms. While the condition is predominantly documented in middle-aged males, the occurrence of the classic variant in a fifteen-year-old patient represents a significant departure from established epidemiological patterns. This case underscores the necessity of a deep understanding of the molecular landscape that governs the disease, as well as the diagnostic rigor required to differentiate it from common infectious mimics.

The pathophysiology of EPF is characterized by a highly specific, sterile inflammatory cascade that targets the pilosebaceous unit.[11] Current scientific evidence suggests that the hair follicle serves as the primary epicenter for an exaggerated immune response, where various antigenic stimuli—ranging from follicular microbes to drug-induced triggers—

may initiate the recruitment of eosinophils. This recruitment is not a random occurrence but is instead mediated by a sophisticated network of adhesion molecules and chemoattractants that facilitate the migration of inflammatory cells from the vascular space into the dermal and follicular compartments.[12]

TABLE 2. DIAGNOSIS, TREATMENT, FOLLOW-UP, AND OUTCOME

DIAGNOSTIC CONFIRMATION	
Definitive Diagnosis	**Classic Eosinophilic Pustular Folliculitis (Ofuji Disease)**
Clinical Rationale	Persistent papulopustular eruption (one month) non-responsive to antibiotics and antivirals; anatomical distribution involving face and trunk.
Pathological Evidence	Dermal inflammatory infiltration predominantly composed of eosinophils and neutrophils in a peri-adnexal distribution.
THERAPEUTIC INTERVENTION (INPATIENT CARE)	
Systemic Medications	Intravenous Methylprednisolone 31.25 mg twice daily Intravenous Diphenhydramine 10 mg once daily
Topical Regimen	Mometasone furoate 0.1 percent (face) and Clobetasol propionate 0.05 percent (trunk and extremities) applied twice daily; saline compresses for crusted lesions.
FOLLOW-UP AND CLINICAL OUTCOME	
Laboratory Response	Normalization of peripheral eosinophil count from **12.3 percent** on admission to **0.1 percent** at discharge.
Clinical Resolution	Marked reduction in inflammatory papules and pustules within four days of corticosteroid initiation.
Longitudinal Result	Complete clearance of active lesions at the three-week follow-up; no reported recurrence or new lesion development.

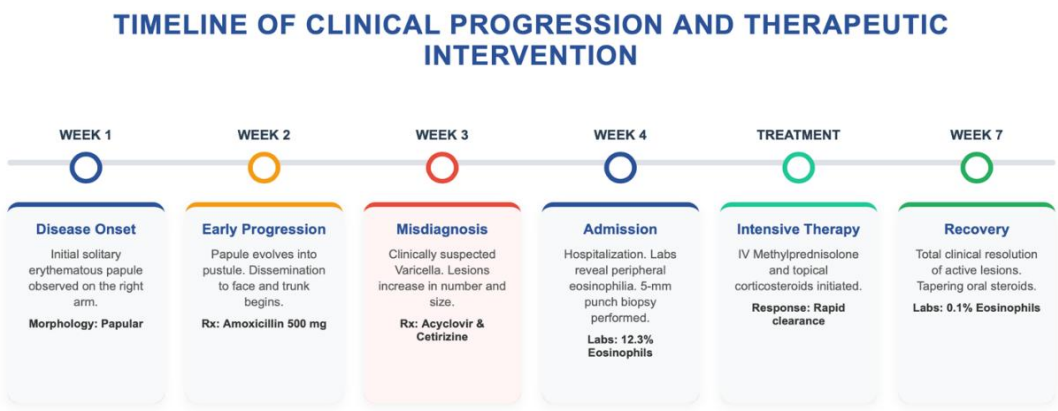


Figure 1. Timeline of clinical progression and therapeutic intervention.

The pathophysiology of eosinophilic pustular folliculitis (EPF) is defined by a highly orchestrated migratory event, where eosinophils are systematically recruited from the systemic circulation into the cutaneous microenvironment (Figure 2).[13] The initial phase of this process involves the targeted upregulation of specific adhesion molecules on the vascular endothelium of the dermal capillaries. Key players in this molecular anchoring include intercellular adhesion molecule-1 (ICAM-1) and vascular cell adhesion molecule-1 (VCAM-1). These proteins function as biological ligating agents that facilitate the tethering and rolling of circulating leukocytes along the vessel wall. Specifically, these adhesion molecules allow eosinophils and T-helper type 2 (Th2) lymphocytes to establish a stable attachment to the endothelium, a prerequisite for their subsequent extravasation into the dermal space. A critical mediator in this trans-endothelial migration is the interaction between lymphocyte function-associated antigen-1 (LFA-1), expressed on the leukocyte surface, and its corresponding endothelial ligands. In patients with EPF, this interaction is speculated to be a vital step in directing the inflammatory infiltrate toward the pilosebaceous unit. This molecular bridge not only permits the entry of cells into the dermis but also ensures that the inflammatory response is localized to the perifollicular structures, setting the stage for the characteristic follicular infiltration seen on histopathology.[14]

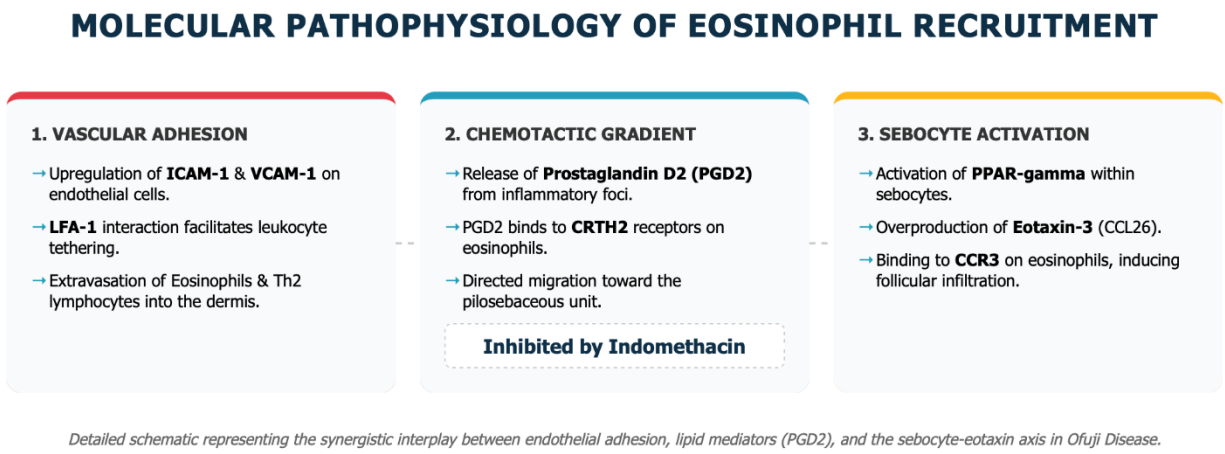


Figure 2. Pathophysiology of eosinophilic pustular folliculitis (EPF).

Central to the sterile inflammatory landscape of Ofuji disease is the lipid mediator Prostaglandin D2 (PGD2). PGD2 is increasingly recognized as the primary chemoattractant responsible for the recruitment and subsequent activation of eosinophils within the pilosebaceous units. This lipid mediator exerts its biological effects by binding with high affinity to the chemoattractant receptor-homologous molecule expressed on Th2 cells (CRTH2), which is prominently displayed on the surface membranes of eosinophils, basophils, and Th2 lymphocytes.[15] The activation of the PGD2-CRTH2 signaling axis promotes the polarized, directed movement of eosinophils toward the concentrated foci of follicular inflammation. This chemotactic gradient effectively guides the eosinophils from the general dermal space to the specific microenvironment of the hair follicle. The fundamental importance of this pathway is underscored by the therapeutic profile of classic EPF; specifically, the high clinical efficacy of indomethacin. Indomethacin functions by inhibiting cyclooxygenase enzymes, thereby suppressing the

synthesis of PGD2. By depleting the source of this potent chemoattractant, the drug effectively reduces CRTH2-mediated eosinophil recruitment, providing a molecular explanation for the rapid clinical resolution often observed with its use.[16]

Beyond the recruitment of cells from the blood, recent research has illuminated the sebaceous gland itself as an active, proinflammatory participant in the pathogenesis of EPF. It is now suggested that the pilosebaceous unit is not merely a passive target of inflammation but a primary source of chemoattractant signals. In this model, PGD2 acts as a paracrine signal that stimulates sebocytes via the activation of peroxisome proliferator-activated receptor gamma (PPAR-gamma).[17] The activation of PPAR-gamma within sebocytes triggers a transcriptional response that leads to the significant overproduction and secretion of eotaxin-3. Eotaxin-3 is arguably the most potent eosinophil-specific chemoattractant within the cutaneous tissue. Once released into the follicular microenvironment, eotaxin-3 binds specifically to the C-C chemokine receptor type 3 (CCR3) receptors located on the surface of the previously recruited eosinophils. This interaction creates a highly localized and specialized recruitment system—often referred to as the sebocyte-eotaxin axis—that directs eosinophils with surgical precision into the follicular epithelium and sebaceous glands. The result of this molecular synergy is the dense, eosinophil-rich follicular infiltration and subsequent follicular destruction that serves as the histopathological hallmark of the disease.[18]

The clinical management of this 15-year-old patient illustrates a profound diagnostic hurdle frequently encountered in rare inflammatory dermatoses: the striking morphological resemblance of eosinophilic pustular folliculitis (EPF) to common viral and bacterial exanthems. This mimicry often leads to a diagnostic odyssey, where patients undergo multiple unsuccessful treatments before a definitive diagnosis is established. In the presented case, the initial misdiagnosis as varicella followed by a failed trial of acyclovir represents a classic clinical pitfall. This delay is particularly pronounced when the patient falls outside the typical age demographic for Ofuji disease, which traditionally affects middle-aged adults between the third and fourth decades of life. The sterile pustules of EPF can easily be mistaken for the vesicles of varicella-zoster or the inflammatory papules of bacterial folliculitis. This diagnostic confusion is not merely academic; it carries significant implications for patient care, often resulting in the unnecessary administration of antimicrobials and the prolongation of patient discomfort.

Differentiating EPF from varicella-zoster virus (VZV) requires a meticulous analysis of the disease kinetics and the presence of systemic markers. Varicella infections are typically accompanied by a clear prodromal phase consisting of fever, malaise, and respiratory symptoms, which are often more severe in adolescents and adults.[19] In contrast, this patient remained consistently afebrile and systemically well throughout the one-month duration of the illness. The course of varicella is typically acute, with lesions progressing from macules to vesicles and crusts within days. The patient's one-month history of persistent and progressive eruptions strongly argued against a self-limiting viral etiology. While VZV can affect almost any cutaneous surface, the specific follicular distribution and the sparing of the palms and soles in this case provided a critical hint toward a follicular-based inflammatory disorder.

Furthermore, the suspicion of bacterial pathogens—such as *Staphylococcus aureus* in non-bullous impetigo or pyogenic folliculitis—was addressed through objective laboratory assessment. Gram staining of pus aspirated from the active lesions served as a vital tool in this differentiation. The microscopic finding of fewer than ten solitary gram-positive cocci per high-power field, rather than the dense clusters or chains associated with active pyogenic infection, supported the classification of the eruption as a sterile inflammatory process. The complete lack of clinical improvement following a course of amoxicillin further reinforced the non-infectious nature of the dermatosis.[20]

A pivotal diagnostic clue that shifted the clinical focus toward Ofuji disease was the presence of marked peripheral eosinophilia. On admission, the patient exhibited an absolute eosinophil count of 12.3 percent, a value significantly exceeding the standard physiological range of 0.0 to 4.0 percent. While peripheral eosinophilia is not a mandatory diagnostic criterion for all EPF variants, it is a highly supportive finding that frequently correlates with the severity and activity of the inflammatory process. In this case, the hematological abnormality served as a signal to pursue histopathological verification. The value of this biomarker was further demonstrated during the treatment phase. The rapid normalization of the eosinophil level to 0.1 percent upon discharge served as an objective laboratory marker of therapeutic success, paralleling the marked clinical improvement observed in the skin lesions. The presence of eosinophilia in the absence of parasitic infection or atopic dermatitis should always prompt the clinician to consider eosinophilic dermatoses, especially when presenting with recalcitrant follicular pustules.

Perhaps the most challenging aspect of this specific case was the presence of atypical clinical features that diverged from the classic description of Ofuji disease. While pruritus is nearly universally cited as a hallmark feature of EPF, its complete absence in this 15-year-old patient represented a significant diagnostic hurdle. Typically, the intense itching is what drives patients to seek urgent care; however, the silent nature of the inflammation in this case favored the misdiagnosis of an infectious exanthem. The presence of crusted erosions and irregular plaques rather than discrete arcuate clusters meant the clinical gestalt was more suggestive of a viral process than the classic Ofuji presentation. These deviations serve as a potent reminder that the clinical spectrum of eosinophilic pustular folliculitis is remarkably broad. The absence of a single pathognomonic symptom, such as pruritus, should not lead to the premature exclusion of EPF from the differential diagnosis. Instead, clinicians must rely on the integration of history, laboratory markers like eosinophilia, and the gold standard of skin biopsy to achieve an accurate diagnosis.

A primary limitation of the current clinical investigation is the inherent brevity of the follow-up period. While the patient exhibited a remarkably favorable clinical and laboratory response at the three-week assessment, classic eosinophilic pustular folliculitis (EPF) is historically recognized for its chronic and relapsing nature. The disease often persists for several years, characterized by periodic flares and intermittent remissions. Consequently, there is a distinct possibility that this adolescent patient may experience recurrence as he transitions into adulthood, a factor that necessitates meticulous long-term dermatological monitoring to manage potential chronicity.[17,18]

The manifestation of EPF in an adolescent patient raises significant scientific questions regarding the influence of hormonal changes on the phenotypic expression of the disease. Future research should aim to clarify whether the rapid physiological and endocrine shifts associated with puberty—specifically the surge in androgens—play a mechanistic role in the hyper-activation of the sebocyte-eotaxin axis. Specifically, utilizing advanced immunohistochemical staining to quantify the expression of chemoattractant receptor-homologous molecule (CRTH2), eotaxin-3, and peroxisome proliferator-activated receptor gamma (PPAR-gamma) in adolescent cohorts could provide invaluable insights into whether these molecular pathways function differently in younger populations compared to the classic middle-aged demographic. Such investigations are warranted to determine if adolescent-onset EPF represents a unique subset of Ofuji disease driven by age-specific inflammatory triggers.

There currently remains a significant lack of standardized, evidence-based treatment algorithms specifically tailored for the management of adolescent EPF. While systemic corticosteroids and antihistamines proved highly effective in achieving rapid remission in this specific case, the potential

long-term adverse effects of systemic steroid therapy—including growth suppression and metabolic disturbances—must be carefully weighed in a developing adolescent. Further research is urgently required to investigate the safety and efficacy of steroid-sparing agents, such as topical tacrolimus or oral indomethacin, in younger populations. Establishing optimal, individualized management strategies that minimize long-term iatrogenic risks while successfully preventing disease recurrence is essential for improving the quality of life for adolescent patients.[19,20]

4. Conclusion

The clinical documentation of this case underscores that adolescent-onset eosinophilic pustular folliculitis (EPF) is a rare and formidable diagnostic entity that frequently mimics common infectious exanthems. The striking morphological resemblance of sterile, follicular papulopustules to viral pathogens like varicella-zoster or bacterial infections such as non-bullous impetigo often leads to a diagnostic odyssey characterized by the inappropriate and ineffective administration of antiviral and antibiotic therapies. In this 15-year-old patient, the chronicity of the lesions and the complete failure of empiric amoxicillin and acyclovir served as critical indicators that the underlying pathology was non-infectious and inflammatory. A primary clinical pearl derived from this case is the utility of peripheral eosinophilia as a vital laboratory biomarker. While serum markers are not universally diagnostic for all variants of Ofuji disease, the presence of an elevated eosinophil count (12.3% in this case) should immediately raise clinical suspicion for an eosinophilic dermatosis when traditional infectious treatments fail. The dynamic nature of this biomarker was further evidenced by its rapid normalization to 0.1% following targeted treatment, providing an objective metric of systemic anti-inflammatory success alongside clinical clearance.

Furthermore, this report reaffirms that a definitive diagnosis via a 5-mm punch skin biopsy remains the gold standard for navigating the diagnostic chameleon that is EPF. The identification of dense, focal inflammatory cell infiltrates composed predominantly of eosinophils and neutrophils in a peri-adnexal distribution is essential for differentiating this idiopathic disease from its infectious mimics. Such histopathological confirmation is the critical prerequisite for initiating potent corticosteroid therapy, which in this case proved highly effective in suppressing the prostaglandin-mediated inflammatory cascade and resolving the extensive crusted erosions within days. Ultimately, early recognition and accurate diagnosis of EPF in the adolescent population are essential to prevent unnecessary medication use, mitigate the risk of disease recurrence, and ensure the delivery of effective, individualized care. This case contributes valuable insights into the expanding demographic spectrum of Ofuji disease and highlights the need for clinicians to maintain a high index of suspicion for eosinophilic disorders when faced with recalcitrant, sterile papulopustular eruptions in younger patients.

5. References

1. Li Y, Chen G, Zhou X, Zheng X, Zhang M, Yao X, et al. Clinical and pathological analysis of 10 cases of eosinophilic pustular folliculitis. Clin Cosmet Investig Dermatol. 2023; 16: 2467–72.
2. Ottavi M, Velin M, Cardot Leccia N, Passeron T. Successful treatment of eosinophilic pustular folliculitis with dupilumab. JAMA Dermatol. 2024; 160(1): 114–5.
3. Li Y, Yang X, Lu J, Chen K, Wu W, Zhang M, et al. A case of eosinophilic pustular folliculitis misdiagnosed as eczema. Clin Cosmet Investig Dermatol. 2024; 17: 199–204.
4. Chersi F, Zelin E, Mazzoletti V, Caro Caposiena DR, Hasa Z, Nan K, et al. Mepolizumab therapy for eosinophilic pustular folliculitis (Ofuji disease). J Eur Acad Dermatol Venereol. 2024; 38(11): e993–6.

5. Cai L, Yan Y, Li Y, Lin J, She X, Wang X. Two cases of eosinophilic pustular folliculitis successfully treated with abrocitinib. J Dermatol. 2024; 51(12): 1694–7.
6. Zheng C, Yu Y, Zhou G, Liu H. A case of generalized eosinophilic pustular folliculitis: treatment with JAK inhibitor. J Dermatolog Treat. 2024; 35(1): 2331785.
7. Jawad E-A, Mohamed EA, Youssef Z, Rachid F, Naoufal H. Eosinophilic Pustular Folliculitis (EPF) or Ofuji Disease: Case Report. Clin Med Image Libr. 2024; 10(2).
8. Chen J, Kim T, Beatty CJ, Patton T, Choudhary S. Eosinophilic pustular folliculitis in the setting of untreated chronic Lymphocytic leukemia. Cutis. 2024; 113(3): E22–4.
9. Nailling BB, Smetanick MT, Kajoian AB. Eosinophilic pustular folliculitis after phlebectomy: a case report. J Dermatol Physician Assist. 2024; 18(3): 36–8.
10. Masuo Y, Yonekura S, Yasumi T, Seto H, Yamamoto A, Hashida Y, et al. Eosinophilic pustular folliculitis in a child with hyper-IgE syndrome: a case report emphasizing the importance of immunodeficiency screening in infancy. J Dermatol. 2025; 52(11): e1010–2.
11. Jiang X, Wu X, Chen S, Cheng H. Successful treatment of severe and refractory eosinophilic pustular folliculitis with dupilumab: a case report with two-year relapse-free follow-up. J Inflamm Res. 2025; 18: 15779–85.
12. Mendes BX, Antunes VL Jr, Barbosa LM, Defante MLR, Morais BAAH, Silva CR, et al. Clinical features and treatment of eosinophilic pustular folliculitis in childhood: a systematic review and single-arm meta-analysis. Pediatr Dermatol. 2025; (pde.70060).
13. Mansaray L, Comoz F, Dompmartin A, Kottler D. Atypical eosinophilic pustular folliculitis successfully treated with mepolizumab. Ann Dermatol Venereol. 2025; 153(1): 103454.
14. Flowers LE, Subhadarshani S, Quinter S, Shaughnessy C. Eosinophilic pustular folliculitis associated with IgA deficiency. JEADV Clin Pract. 2025; 4(1): 244–7.
15. Li W, Hu X, Li Y, Chen G. Successful treatment of eosinophilic pustular folliculitis with secondary follicular mucin deposition using indomethacin: an atypical case and literature review. Clin Cosmet Investig Dermatol. 2025; 18: 1063–70.
16. Mansilla-Polo M, Navarro-Blanco F, López-Davia J, Botella-Estrada R. Successful treatment of recalcitrant eosinophilic pustular folliculitis with dupilumab. Sultan Qaboos Univ Med J. 2025; 25(1): 622–7.
17. Wang Q-X, He H-Y, Niu Y-L, Fang S. Refractory eosinophilic pustular folliculitis treated with tofacitinib: a case series and literature review. Clin Exp Dermatol. 2025; 50(6): 1196–200.
18. Xu J, Wu Y-M, Wang S-S. A case of eosinophilic pustular folliculitis involving palmoplantar successfully treated by apremilast. Skin Health Dis. 2025; 5(3): 223–6.
19. Liu Y, Liu M, Chen S, Liu Y, Zhou G, Liu G. Baricitinib in the treatment of eosinophilic pustular folliculitis resistant to indomethacin. J Dtsch Dermatol Ges. 2025; 23(8): 1006–8.
20. Tan X, Nie S, Chen B, Wu Z. Successful treatment of eosinophilic pustular folliculitis with dupilumab in a 9-year-old boy. Pediatr Dermatol. 2025; 42(5): 1044–6.