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(CASE REPORT)

SYSTEMIC METHYLPREDNISOLONE–ASSOCIATED ACNEIFORM ERUPTION MIMICKING MALASSEZIA FOLLICULITIS IN AN ADOLESCENT: A CASE REPORT

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ABSTRACT

Drug-induced acneiform eruptions have been well-known side effects of systemic corticosteroids, usually presenting as monomorphic inflamed papules and pustules without comedones. Similar to Malassezia folliculitis, their presentation can make their diagnosis difficult especially among teenagers taking systemic corticosteroids for long periods of time.A 14-year-old boy presented with progressive papulopustular lesions that first affected his back after approximately two months of therapy with oral methylprednisolone for traumatic optic nerve edema. In total, there were numerous discrete, monomorphic, and predominantly folliculocentric red papules and pustules involving his face, neck, chest, shoulders, back, and proximal upper limbs. No comedones, nodules, and cysts were present, and he experienced minimal pruritus. In spite of the morphology and distribution being suggestive of Malassezia folliculitis, the direct examination under potassium hydroxide preparation of lesional material showed no yeast or hyphal forms. Based on the temporal relationship with high doses of methylprednisolone, morphology, minimal pruritus, negative direct microscopy, and lack of an alternate well-established etiology, a steroid-induced acneiform eruption was diagnosed. Methylprednisolone was decreased based on an ophthalmologist-directed taper regimen, along with oral erythromycin, cetirizine, and topical fusidic acid. After one week of treatment, pain, purulent drainage, and inflammation were improved without development of new lesions. With a Naranjo score of 6, it was probable. Systemic corticosteroid-induced acneiform eruption needs to be taken into account in cases where the patient is suffering from a monomorphic

papulopustular eruption while on treatment. Since there is an extensive similarity in both the diseases with respect to the clinical presentation, diagnosis can only be done by considering all aspects together.

Keywords: Acneiform eruption; Methylprednisolone; Steroid acne; Malassezia folliculitis; Adolescent; Adverse drug reaction

1 INTRODUCTION

Acneiform drug eruptions is a condition where there is a sudden or gradual onset of inflammatory papules and pustules caused by a drug which leads to the skin eruptions. Acneiform drug eruptions differ from acne vulgaris in that they occur in a monomorphic pattern, have less or no comedones, and also occur beyond the common areas where acne vulgaris occurs. The systemic use of glucocorticoids is one of the most well-known causes, with the eruptions occurring a few weeks to months after initiating therapy[1].

Data regarding the epidemiology of acneiform drug reactions are scarce because such reactions are often classified under acne vulgaris or nonspecific folliculitis in various clinical and pharmacovigilance studies. The prevalence of acneiform reactions among all drug eruptions is thought to be around 1%, with the male sex being the most affected group; yet, the frequency of acneiform eruptions that are associated with systemic corticosteroids is not known exactly. In contrast, the prevalence rate of Malassezia folliculitis is unknown either, but in-hospital studies have found frequencies between 1%–2% in China and 3%–4% in Saudi Arabia and Türkiye and up to 16% in the Philippines. Malassezia folliculitis is mainly found in adolescents and young people, especially men, which is due to the higher secretion of sebum and increased colonization of the skin with fungi during puberty. Moreover, the condition is favored by heat, humidity, heavy sweating, occlusion, antibiotics, and immunosuppressive treatments including corticosteroids [2–4]. These epidemiological characteristics are particularly relevant in tropical countries such as Indonesia, where climatic conditions and high humidity may increase clinical suspicion for Malassezia folliculitis when evaluating monomorphic truncal eruptions.

Discrimination between corticosteroid-induced acneiform eruptions and Malassezia folliculitis may sometimes be difficult since both disorders have monomorphic follicular papules and pustules on the trunk and face. Malassezia folliculitis is an inflammatory disorder resulting from an excessive growth of lipophilic Malassezia yeasts in the hair follicle and primarily occurs among adolescents and young adults. Itching, follicular papules or pustules, distribution of lesions to seborrheic areas and lack of comedones are some clues pointing to Malassezia folliculitis, but none of these criteria is distinctive enough for every individual case[4,5] .

Exposure to systemic corticosteroids adds yet another dimension of complexity in making this differential diagnosis. Along with being capable of causing a drug reaction with acne-like eruption, the immunomodulatory effects of corticosteroids could help Malassezia proliferate, resulting in marked similarity between the two conditions. Direct visualization of properly obtained follicular samples under potassium hydroxide preparations, however, would prove to be an invaluable aid in this regard, although it should be read along with other factors like morphology of lesions, medications, symptoms, and treatment response. A negative potassium hydroxide finding decreases the possibility of Malassezia folliculitis but is not definitive for excluding it [3,6] .

The current report outlines the case of a young person with a diffuse monomorphic papulopustular eruption affecting the face and trunk, which occurred about three months after the administration of high doses of oral methylprednisolone. Initially, the clinical morphology suggested Malassezia folliculitis, but a direct potassium hydroxide examination revealed no signs of yeasts or hyphae. Based on the temporal relationship between methylprednisolone administration, compatible morphologic and distributional features, the lack of yeast or hyphae in KOH preparation, and successful treatment without antifungal medications, a diagnosis of systemic methylprednisolone-related acneiform eruption was made. This report underscores the diagnostic challenge in differentiating between corticosteroid-related acneiform eruptions and Malassezia folliculitis.

2 METHODS

A systematic review design was employed to synthesize evidence from retrospective and prospective cohort studies. Literature searches were conducted in PubMed, Google Scholar, Wiley Online Library, and ScienceDirect. The primary outcome, defined according to the PICO framework, was the evaluation of mortality in malignant melanoma. The included articles were further assessed for quality and methodological rigor using the Joanna Briggs Institute (JBI) risk of bias tools.

3 CASE PRESENTATION

A 14-year-old boy was referred from the ophthalmology clinic to the dermatology clinic on September 18, 2026, with a two-month history of small bumps on his back. The lesions initially appeared on his back in August 2026 and progressively increased in number, subsequently spreading to the anterior chest and face. Several lesions discharged purulent material and were painful, whereas pruritus was minimal. He denied previous similar eruptions, the application of topical oils or ointments, and any history of drug, food, or environmental allergies.

Approximately three months before his initial dermatologic presentation, the patient had commenced regular oral methylprednisolone therapy for traumatic optic nerve swelling. An ophthalmologic evaluation performed on September 10, 2026, documented right optic neuritis. Complete information regarding the initial three-month corticosteroid regimen was unavailable. The documented tapering schedule consisted of oral methylprednisolone 56 mg once daily, followed by 48 mg once daily for one week and then 40 mg once daily for one week until the subsequent ophthalmologic review. Concomitant medications included oral omeprazole and a vitamin B-complex preparation.

Figure 1: Clinical presentation at the initial dermatologic evaluation. Numerous discrete, monomorphic, predominantly folliculocentric erythematous papules and pustules involving the face, anterior chest, shoulders, upper back, posterior trunk, and proximal upper extremities. The lesions were most densely distributed over the upper trunk and shoulders, without evident comedones, nodules, or cysts.

Dermatologic examination as shown on figure 1. revealed numerous discrete, predominantly folliculocentric, monomorphic erythematous papules and pustules distributed symmetrically over the anterior chest, shoulders, upper back, posterior trunk, face, neck, and proximal upper extremities. The eruption was most prominent over the upper chest, shoulders, and back. Several pustules showed central purulent contents or superficial crusting, accompanied by scattered postinflammatory hyperpigmented macules. No evident comedones, nodules, or cysts were identified.

Based on the clinical presentation and physical examination findings, the patient was diagnosed with a systemic methylprednisolone-associated acneiform eruption, clinically presenting as steroid folliculitis. The diagnosis was supported by the characteristic monomorphic and predominantly follicular lesions, minimal pruritus, absence of comedones, and the temporal relationship with systemic corticosteroid exposure. Malassezia folliculitis was subsequently considered as an important differential diagnosis because of the monomorphic follicular morphology. However, direct potassium hydroxide examination of lesional material demonstrated neither yeast forms nor hyphal elements. The negative mycological examination, together with the clinical findings, supported the exclusion of Malassezia folliculitis. Nevertheless, the negative potassium hydroxide result was interpreted in conjunction with the clinical findings rather than as an independent means of excluding Malassezia folliculitis.

Since the lesions were monomorphic and predominantly follicular, Malassezia folliculitis was initially considered an important differential diagnosis. However, direct potassium hydroxide examination of lesional material demonstrated neither yeast forms nor hyphal elements. The negative mycological examination, minimal pruritus, absence of comedones, characteristic morphology, and temporal relationship with systemic corticosteroid exposure supported a diagnosis of systemic methylprednisolone–associated acneiform eruption, clinically presenting as steroid folliculitis. Nevertheless, the negative potassium hydroxide result was interpreted alongside the clinical findings rather than as independently excluding Malassezia folliculitis

Methylprednisolone was identified as the suspected precipitating medication, and dose reduction was undertaken according to the ophthalmology-directed tapering schedule. The patient was treated with oral erythromycin 500 mg three times daily for seven days, oral cetirizine 10 mg once daily, and topical fusidic acid cream applied twice daily to

pustular lesions. The patient and his caregiver were counselled regarding the suspected medication-related etiology, appropriate skin hygiene, avoidance of prolonged moisture and occlusion, frequent changing of perspiration-soaked clothing, and regular bathing using a moisturizing cleanser.

Figure 2: Clinical findings at the one-week follow-up. Persistent papulopustular lesions and postinflammatory hyperpigmentation with reduced inflammatory activity and no development of new lesions after methylprednisolone dose reduction and seven days of dermatologic treatment..

At the first follow-up visit on September 25, 2026, one week after treatment initiation, the patient reported a reduction in lesion-associated pain and a substantial decrease in purulent discharge. No new lesions had developed during the follow-up period. Examination and serial clinical photographs as shown on figure 2 showed persistent papulopustular lesions and postinflammatory changes, but with reduced inflammatory activity compared with the initial presentation. No treatment-related adverse events were reported during this short follow-up period.

A conservative application of the Naranjo adverse drug reaction probability scale yielded a score of 6, classifying the association between methylprednisolone and the acneiform eruption as probable. Points were assigned for previous reports of this recognized reaction (+1), development of the eruption after drug exposure (+2), the absence of a sufficiently supported alternative cause (+2), and objective confirmation through dermatologic examination and serial photographs (+1). No points were assigned for complete dechallenge, rechallenge, placebo exposure, toxic drug concentrations, previous similar reactions, or a definite dose-response relationship. The latter was not credited because corticosteroid tapering and dermatologic treatment occurred concurrently, preventing the improvement from being attributed exclusively to dose reduction. Scores of 5–8 indicate a probable adverse drug reaction under the Naranjo algorithm.

Table 1: Naranjo adverse drug reaction probability scale applied to the present case. The selected responses and corresponding scores are shown in bold. A total score of 6 indicates a probable association between systemic methylprednisolone and the acneiform eruption. Points were added for previous reports of corticosteroid-associated acneiform eruptions, onset after methylprednisolone exposure, the absence of a sufficiently supported alternative cause, and objective confirmation by dermatologic examination and serial photographs. Dechallenge and dose–response points were not assigned because methylprednisolone tapering and dermatologic treatment occurred concurrently, precluding attribution of the clinical improvement exclusively to corticosteroid dose reduction. Scores of 5–8 indicate a probable adverse drug reaction according to the Naranjo algorithm.

No.	Naranjo question	Yes	No	Unknown/not done	Assessment in this case	Score
1	Are there previous conclusive reports of this reaction?	+1	0	0	Yes	**+1**
2	Did the adverse event appear after the suspected drug was administered?	+2	−1	0	Yes	**+2**
3	Did the adverse reaction improve when the drug was discontinued or a specific antagonist was administered?	+1	0	0	Unknown	**0**

4	Did the adverse reaction reappear when the drug was readministered?	+2	−1	0	Not done	**0**
5	Are there alternative causes that could independently have caused the reaction?	−1	+2	0	No	**+2**
6	Did the reaction reappear when a placebo was administered?	−1	+1	0	Not done	**0**
7	Was the drug detected in blood or other body fluids at a concentration known to be toxic?	+1	0	0	Not done	**0**
8	Was the reaction more severe when the dose was increased or less severe when the dose was decreased?	+1	0	0	Unknown	**0**
9	Did the patient have a similar reaction to the same or a related drug during a previous exposure?	+1	0	0	No/unknown	**0**
10	Was the adverse event confirmed by objective evidence?	+1	0	0	Yes	**+1**
	Total Naranjo score				**Probable adverse drug reaction**	**6**

Table 2: Timeline of events

Date	Event
Approximately June 2026	Oral methylprednisolone was initiated for traumatic optic nerve swelling, approximately three months before the first dermatologic evaluation.
August 2026	Small acne-like lesions first appeared on the back. The lesions progressively increased in number and spread to the anterior chest and face; some discharged purulent material, while pruritus remained minimal.
September 10, 2026	Ophthalmologic assessment documented right optic neuritis, with retrobulbar neuritis as a differential diagnosis. The available methylprednisolone regimen was 56 mg orally once daily, followed by 48 mg once daily for one week and then 40 mg once daily for one week until reassessment. Concomitant medications included omeprazole and a vitamin B-complex preparation.
September 18, 2026	The patient was referred to dermatology. Examination showed numerous discrete, predominantly folliculocentric, monomorphic erythematous papules and pustules involving the face, neck, anterior chest, shoulders, back, and proximal upper extremities, without evident comedones, nodules, or cysts.
September 18, 2026	Direct KOH examination of lesional material revealed no yeast or hyphal elements.
September 18, 2026	The suspected corticosteroid trigger was addressed through the ophthalmology-directed taper. Dermatologic treatment comprised oral erythromycin 500 mg three times daily for seven days, oral cetirizine 10 mg once daily, and topical fusidic acid cream twice daily on pustular lesions, together with skin-care counselling.
September 25, 2026	At the first follow-up, the patient reported reduced pain and substantially decreased purulent discharge. No new lesions had developed, and the clinical photographs showed reduced inflammatory activity with persistent papulopustular and postinflammatory lesions.

4 DISCUSSION

Acneiform eruptions are follicular eruptions which typically appear within a few weeks to months of exposure to a drug responsible for their occurrence. Unlike the common type of acne vulgaris, they consist of monomorphic inflammatory papules and pustules with few or no comedones and they may involve areas other than those usually affected by acne

7 . Glucocorticoids have been well established as causative drugs and steroid eruptions typically arise from two weeks up to several months of treatment, mostly involving the face, neck, chest, upper back, and extremities. In the case being considered, there was an onset of eruption two months after oral methylprednisolone administration with lesions involving the back, chest, face, neck, shoulder, and upper limbs [1].

The pathogenesis of corticosteroid-associated acneiform eruption is incompletely defined and is probably multifactorial [8] . The current scientific literature indicates that corticosteroids may affect sebum production and lipid content, disrupt the follicular microbiological milieu, affect keratinocyte growth and differentiation, and even affect inflammation mechanisms and neutrophil migration. This results in a lipid-abundant and pro-inflammatory pilosebaceous unit, facilitating the formation of fairly homogenous follicular inflammatory lesions. The significant systemic exposure of the current case makes a biologically rational cause for the condition, despite the unknown initial dosing information and lack of a dose-response effect[1,9].

The major problem with making a diagnosis was that of distinguishing between the eruption and Malassezia folliculitis. Malassezia folliculitis is due to symptomatic growth of lipophilic yeast organisms in the pilosebaceous follicles and is characterized by monomorphic follicular papules and pustules on the upper trunk, shoulders, neck, hairline, and face. The eruption is usually pruritic but without comedones [10]. Adolescence, seborrhoea, heat and humidity, sweating, occlusion, antibiotics, and immunosuppression can increase the risk; systemic corticosteroids are especially important since they may be responsible for causing both a non-infective form of acne as well as an overgrowth of Malassezia. Thus, neither the morphology nor the use of corticosteroids can differentiate these conditions[11,12].

A number of observations supported the corticosteroid-induced acneiform reaction hypothesis. First, pruritus was mild, the lesions were developed following the start of methylprednisolone treatment, and no yeast cells were found on direct potassium hydroxide examination. Furthermore, the patient improved with no antifungal therapy but only the tapering off the corticosteroid treatment. However, the negative result of direct potassium hydroxide examination cannot be considered as exclusion of Malassezia folliculitis. According to the 2023 position statement of the European Academy of Dermatology and Venereology, clinical examination and direct microscopic examination should be regarded as main diagnostic tools, while histology, culture of fungi, wood lamp examination, and molecular or optical diagnosis are recommended as auxiliary methods in case of diagnostic uncertainties. One study cited in the position statement found 40 cases out of 49 by potassium hydroxide microscopy, which is 81.6% sensitivity; therefore, sampling method significantly influences the result of the test [10] .

A multicenter Indonesian study of 353 patients found that *Malassezia* folliculitis predominantly affected males and younger individuals; pruritus occurred in 83.9%, lesions involved the chest, back, or shoulders in 68.3%, and follicular papulopustules were the most frequent morphology. That study also found positive potassium hydroxide examinations in 87.4% of diagnosed cases, indicating good practical yield while confirming that a minority of clinically diagnosed cases may have negative microscopy. The tropical Indonesian climate, the patient's adolescent age, truncal involvement, and corticosteroid-associated immunomodulation therefore remained relevant risk factors despite the negative examination [13] .

Correct specimen selection becomes especially crucial in this case because Malassezia is an integral part of the skin mycobiome. Follicular material from pustules or comedo extraction should provide more information compared to surface scale, and microscopic examination will show budding yeast cells, hyphae are rare in folliculitis. Thus, absence of yeast in this patient made Malassezia folliculitis less probable but one negative result might mean insufficient amount of the fungus or improper sampling technique. Recurrence of lesions, resistance to treatment or appearance of severe itching in the future will warrant further investigation including repeated microscopic examination of follicular material, fungal stain, biopsy with periodic acid-Schiff or Grocott methenamine silver stains, or an antifungal trial [14] .

Other pertinent differential diagnoses were bacterial folliculitis and acne vulgaris. Painful nature, pus production, and early response to antibiotic treatment suggested the possibility of bacterial infection; nevertheless, the broad symmetrical appearance and strong association with the usage of methylprednisolone made it more likely a reaction to drugs rather than bacterial infection. Since bacterial cultures were not taken, the diagnosis of bacterial folliculitis or bacterial infection could not be ruled out. Diagnosis of acne vulgaris was less probable due to the absence of comedones, nodules, or cysts, as well as the acute onset and homogenous nature of the eruption that was predominantly truncal in location.[15]

This is clinically relevant, as the Malassezia folliculitis can actually be exacerbated during antibacterial treatment for acne. As noted by Prindaville et al. in their review of cases in the pediatric/young adult population, more than 75% of affected individuals had recent antibiotic use for acne.[12] Assessment of causality tended to support, but not demonstrate, the presumed relationship. The Naranjo score is 6, which falls under the "probable" classification of 5–8. This score was based on an established reaction, plausible timing, no other more strongly proven alternative, and objective skin findings. No re-challenge was performed, there was no full dechallenge seen, and no dose-response item could be

assigned due to the concomitant use of methylprednisolone tapering and dermatology treatment. Causality scales serve as tools to support, but do not serve as diagnostic tests, particularly in pediatrics where many Naranjo criteria are not available.[16]

Management of a drug-induced acneiform eruption begins with identifying the precipitating agent and balancing cutaneous morbidity against the indication for continued therapy. When the implicated medication remains clinically necessary, symptomatic dermatologic treatment may permit continuation or gradual dose reduction [7,9]; coordination with the prescribing specialist is essential. In this patient, ophthalmology supervised methylprednisolone tapering rather than abrupt withdrawal. A seven-day course of oral erythromycin and topical fusidic acid was used for inflammatory pustular lesions, and reduced pain, purulent discharge, and inflammatory activity were documented at one week. Because antimicrobial treatment and corticosteroid reduction occurred simultaneously, improvement cannot be attributed to either intervention alone. The absence of antifungal therapy with no new lesions during early follow-up is supportive but not conclusive evidence against *Malassezia* folliculitis.

There are certain limitations in this case report. The first regimen with methylprednisolone is not described fully; direct microscopy was done only once; the exact method of sampling and the type of fungal staining are unknown; bacterial culture and histopathology tests could not be done; and the follow-up lasted only one week. Thus, the complete recovery from the corticosteroid withdrawal was not proven, and the rechallenge would have been inappropriate in this case. In spite of all the limitations, the presented case has significant educational value since it clearly demonstrates the great similarity between acneiform eruption in connection with the steroid intake and Malassezia folliculitis in a patient with many risk factors for both disorders.

5 CONCLUSION

Systemic methylprednisolone administration may trigger a monomorphic papulopustular acneiform rash resembling Malassezia folliculitis. This case, where the time course of onset in an adolescent patient correlated with high-dose corticosteroid use, truncal monomorphic eruptions without comedones, mild pruritus, negative mycological analysis, and prompt response to supervised corticosteroid tapering suggests corticosteroid-induced acneiform rash. A negative potassium hydroxide test makes the possibility of Malassezia folliculitis less likely but not impossible and should warrant repeat sampling or histopathological confirmation in cases where the course is otherwise suspicious or refractory. A coordinated effort with the responsible specialist will help manage the cutaneous side effect without compromising the treatment of the underlying disease.

STATEMENTS ON COMPLIANCE WITH ETHICAL STANDARDS

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Disclosure of conflict of interest

The authors declare that they have no conflict of interest.

Statement of informed consent

Informed consent was obtained from all individual participants included in the study.

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