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A rare case of symmetrical drug-related intertriginous and flexural exanthema (SDRIFE)

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ABSTRACT

Symmetrical drug-related intertriginous and flexural exanthema (SDRIFE) is a rare maculopapular drug eruption mediated by type IV hypersensitivity reaction and characterized by symmetrical erythematous involvement of flexural and intertriginous areas without systemic symptoms. This case report describes a rare presentation of SDRIFE and emphasizes the importance of early recognition and prompt withdrawal of the offending drugs. A 27-year-old woman presented with well-demarcated erythematous patches involving the axillae, inframammary region, antecubital fossae, inguinal, popliteal, and gluteal areas following exposure to multiple systemic medications, including cefadroxil, mefenamic acid, amoxicillin, oral dexamethasone, paracetamol, and loratadine. No mucosal involvement or systemic manifestations were observed. Histopathological examination revealed non-specific findings of cutaneous drug eruption. The clinical presentation met the established diagnostic criteria for SDRIFE, and drug causality assessment using the Naranjo Scale identified several medications as probable triggers. Management consisted of discontinuation of the suspected drugs, patient education to avoid re-exposure, and treatment with systemic and topical corticosteroids, resulting in marked clinical improvement within approximately two weeks. Differential diagnoses, including drug reaction with eosinophilia and systemic symptoms (DRESS), acute generalized exanthematous pustulosis (AGEP), and fixed drug eruption (FDE), were considered and excluded based on clinical features and laboratory findings. Histopathological findings in SDRIFE are known to be variable and non-specific. Although drug patch testing is recommended to identify the causative agent, it has not yet been performed in this case. In conclusion, this report highlights the diagnostic value of clinical criteria and the Naranjo Scale in SDRIFE. It also highlights the importance of early diagnosis and prompt drug withdrawal, particularly in patients exposed to multiple medications.

ABSTRACT

Symmetrical drug-related intertriginous and flexural exanthema (SDRIFE) merupakan erupsi obat makulopapular yang jarang terjadi, dimediasi oleh reaksi hipersensitivitas tipe IV, dan ditandai dengan *patch* eritematosa pada area fleksural dan intertriginosa yang simetris tanpa disertai gejala sistemik. Laporan kasus ini bertujuan untuk mendeskripsikan manifestasi SDRIFE yang jarang serta menekankan pentingnya deteksi dini dan penghentian segera obat penyebab. Seorang perempuan berusia 27 tahun datang dengan keluhan bercak kemerahan batas tegas yang melibatkan aksila, regio inframammae, fossa antekubiti, inguinal, poplitea, dan glutea setelah terpapar beberapa obat sistemik, yang meliputi sefadroksil, asam mefenamat, amoksisilin, deksametason oral, parasetamol, dan loratadin. Tidak ditemukan keterlibatan mukosa maupun manifestasi sistemik. Pemeriksaan histopatologi menunjukkan gambaran tidak spesifik untuk erupsi obat pada kulit. Gambaran klinis memenuhi kriteria diagnostik SDRIFE, dan penilaian kausalitas obat menggunakan Skala Naranjo mengidentifikasi beberapa obat pemicu yang bersifat *probable*. Tata laksana meliputi penghentian obat-obatan yang dicurigai, edukasi pasien untuk menghindari pajanan ulang, serta pemberian kortikosteroid sistemik dan topikal, yang menghasilkan perbaikan klinis bermakna dalam waktu sekitar dua minggu. Diagnosis banding pada kasus meliputi *drug reaction with eosinophilia and systemic symptoms* (DRESS), *acute generalized exanthematous pustulosis* (AGEP), dan *fixed drug eruption* (FDE) dipertimbangkan dan disingkirkan berdasarkan gambaran klinis dan temuan laboratorium. Temuan histopatologis pada SDRIFE diketahui bervariasi dan nonspesifik. Meskipun uji tempel obat direkomendasikan untuk mengidentifikasi agen penyebab, pemeriksaan tersebut belum dilakukan pada kasus ini. Laporan ini menegaskan nilai diagnostik kriteria klinis dan Skala Naranjo dalam menegakkan diagnosis SDRIFE serta pentingnya diagnosis dini dan penghentian segera obat penyebab, terutama pada pasien yang terpapar banyak obat.

Keywords:
Cutaneous adverse drug eruption;
Naranjo scale;
Symmetrical drug-related intertriginous and flexural exanthema

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INTRODUCTION

Cutaneous adverse drug reactions (CADRs) are skin changes that develop following the systemic use of medications.[1] These reactions are immune-mediated, unpredictable, and affect more than 7% of the global population.[2] According to the International Consensus on Drug Allergies (ICON), cutaneous drug hypersensitivity reactions are classified into immediate and delayed types. Immediate-type hypersensitivity reactions typically appear within 1-6 hours after drug exposure, presenting as angioedema, urticaria, or anaphylaxis. In contrast, delayed-type hypersensitivity reactions arise several days to weeks after drug administration and range from mild eruptions such as maculopapular exanthema and fixed drug eruption (FDE), to severe conditions including drug reaction with eosinophilia and systemic symptoms (DRESS), Stevens-Johnson syndrome/ toxic epidermal necrolysis (SJS/TEN), and acute generalized exanthematous pustulosis (AGEP).[2]

Symmetrical drug-related intertriginous and flexural exanthema (SDRIFE) is a rare variant of maculopapular drug eruption characterizes with symmetrical erythema affecting at least one flexural area, without systemic manifestations, mediated by type IV hypersensitivity reactions or delayed-type reactions.[3-5] Over the 25 years since its initial recognition, approximately 100 cases have been documented in the literature, with amoxicillin and other β-lactamantibiotics remaining the most frequently implicated agents. However, SDRIFE has also been associated with immunoglobulin therapy, chemotherapeutic drugs, and biologic agents.[3] The present case is important because the rash appeared after the patient was exposed to several systemic medications, making it difficult to identify the causative agent. This case report aims to describe a rare case of SDRIFE following multi-drug exposure and highlights practical clinical lessons, including the importance of constructing a clear chronological drug timeline, applying diagnostic criteria systematically, and the importance of timely identification of CADR and withdrawal of the offending drugs.

CASE

A 27-year-old woman presented with progressively worsening erythematous patches predominantly involving the flexural areas, including the axillae, antecubital fossae, inguinal folds, popliteal fossae, and bilateral gluteal regions. Five weeks prior to admission, the patient received a 14-day course of cefadroxil and mefenamic acid without adverse reactions. One week before admission, she was re-exposed to the oral cefadroxil and mefenamic acid. On five days before admission, she developed erythematous patches over both antecubital fossae, which gradually extended to the inframammary region and back, accompanied by mild pruritus. She had no other complaints of fever, odynophagia, or dyspnea. Four days before admission, the medications were subsequently replaced with oral amoxicillin and oral dexamethasone; however, the eruption continued to worsen. Two days before admission, she received parenteral dexamethasone and diphenhydramine, followed by oral paracetamol and loratadine. As the rash continued to spread, she was referred to Sardjito General Hospital for further evaluation. There was no mucosal involvement, systemic symptoms, or evidence of vesiculation or desquamation. She had no significant past medical history, including no previous drug eruptions or food allergies. Family history was notable for a paternal seafood allergy, with no known drug allergies.

Dermatological examination revealed multiple erythematous macules, patches, and plaques of varying sizes, some coalescing, symmetrically distributed over the inframammary region, axillae, antecubital fossae, abdomen, posterior trunk, inguinal folds, gluteal region, and bilateral popliteal fossae (FIGURE 1).

The main differential diagnoses considered were SDRIFE, DRESS, AGEP, and FDE. Laboratory investigations demonstrated leukocytosis (16.8 × 10^3/μL) with neutrophilia (10.3 × 10^3/μL), mild eosinophilia (0.61 × 10^3/μL), and elevated liver transaminases (AST 38 U/L, ALT 118 U/L), with normal renal function. Skin biopsy was performed on the second day of hospitalization, histopathological examination demonstrated orthohyperkeratosis with a basket-weave pattern, moderate spongiosis, basal layer hyperpigmentation, and mild patchy perivascular lymphocytic infiltration in the dermis, findings that were non-specific for cutaneous drug eruption (FIGURE 2). The non-specific histopathological findings may be related to the biopsy being performed on the second day of hospitalization, after systemic corticosteroid therapy had already been initiated, which may have influenced the observed histological features. The characteristic symmetrical flexural distribution, absence of mucosal and systemic involvement, and supportive laboratory and histopathological findings supported the diagnosis of SDRIFE. Given the exposure to multiple medications within a short period of time, structured causality assessment was performed using the Naranjo Scale for each suspected drug separately. Cefadroxil, mefenamic acid, amoxicillin, paracetamol, and dexamethasone were classified as probable offending drugs, while loratadine was considered as possible trigger. Management included discontinuation of the suspected drugs, patient education to avoid these drugs in the future, and treatment with systemic and topical corticosteroids, resulting in clinical improvement within approximately two weeks. A drug patch test was planned; however, the patient was lost to follow-up.

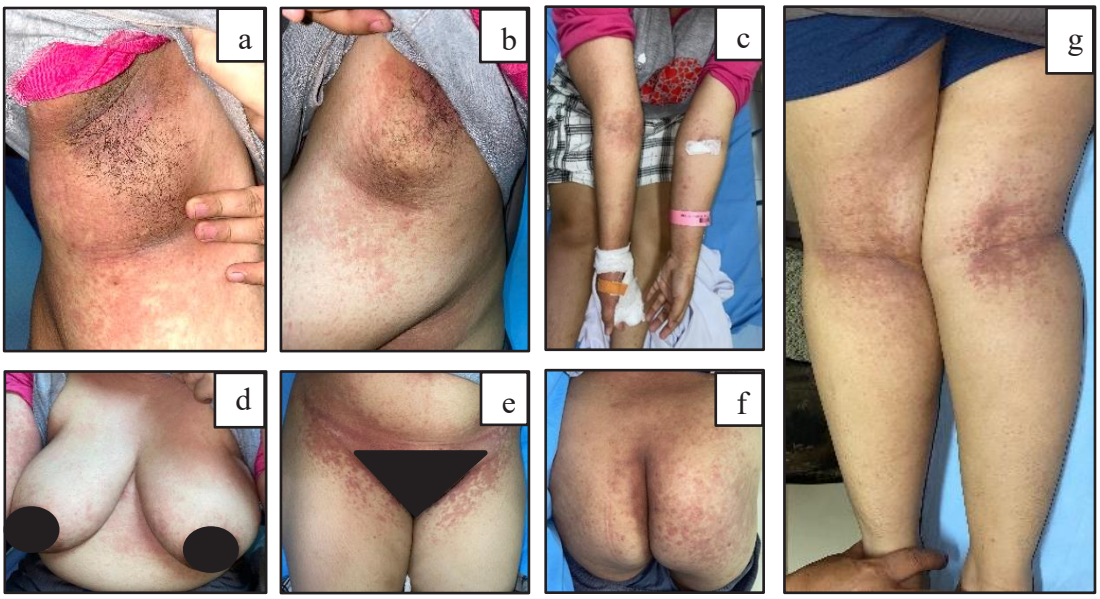

FIGURE 1. Symmetrical erythematous macules, patches, and plaques involving the axillae (a&b), antecubital fossae (c), inframammary region (d), inguinal folds (e), gluteal region (f), and bilateral popliteal fossae (g).

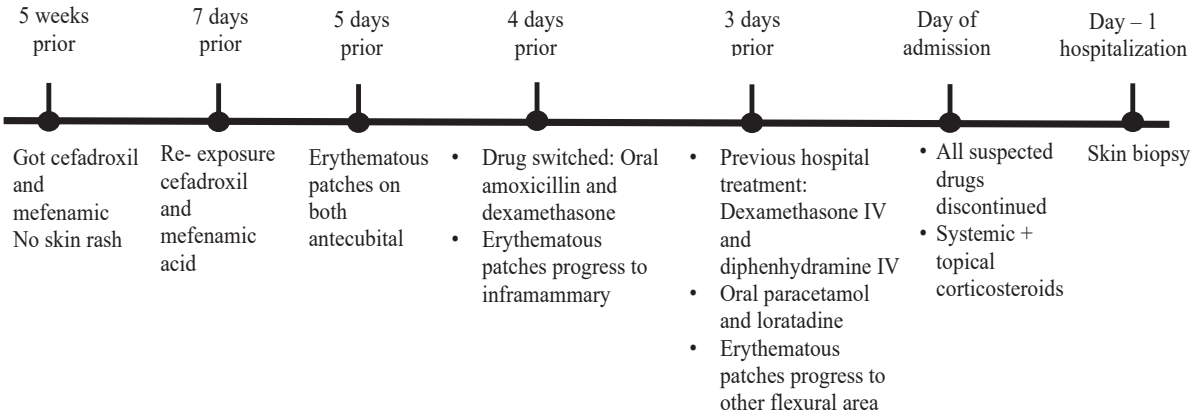

FIGURE 2. Timeline of drug exposure, skin lesion, and biopsy timing

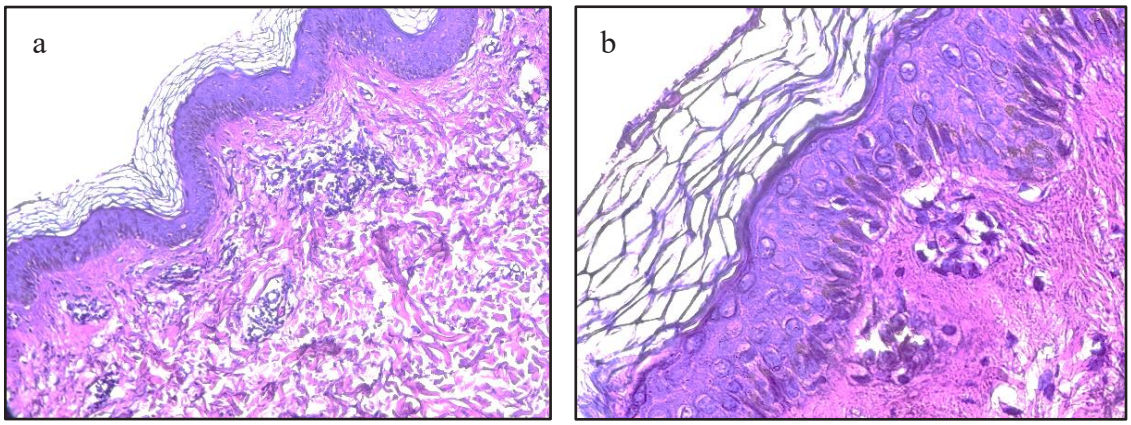

FIGURE 3. (A) Low-magnification view (×100) showing patchy perivascular lymphocytic infiltration within the dermis. (B) High-magnification view (x400) showing moderate spongiosis and basal layer hyperpigmentation.

TABLE 1. Naranjo Adverse Drug Reaction Probability Assessment for Each Suspected Drug

Naranjo Item	Cefadroxil	Amoxicillin	Mefenamic acid	Paracetamol	Dexamethasone	Loratadine
Previous reports of this reaction	+2	+1	+2	+2	+2	0
Event appeared after drug was given	+2	+2	+2	+2	0	0
Reaction improved after drug withdrawal	0	0	0	0	0	0
Reaction on re-administration	0	0	0	0	0	0
Alternative causes present	+2	+2	+2	+2	+2	+2
Reaction with placebo	0	0	0	0	0	0
Toxic drug level detected	0	0	0	0	0	0
Dose–response relationship	+1	+1	0	+1	0	0
Similar reaction previously	0	0	0	0	0	0
Objective evidence	+1	+1	+1	+1	+1	+1
Total score	8	7	7	8	5	+3
Interpretation	Probable	Probable	Probable	Probable	Probable	Possible

DISCUSSION

The diagnosis of SDRIFE in this case was established based on the fulfillment of all five diagnostic criteria proposed by Hausermann *et al.* (TABLE 3).[6] Cutaneous eruptions associated with SDRIFE typically occur within 2–3 days after drug exposure in previously sensitized patients and within 9–10 days to more than two weeks in non-sensitized individuals.[7] Previous reports indicate that the inguinal folds and axillae are the most commonly affected sites, with characteristic lesions presenting as erythematous plaques and/or papules, occasionally accompanied by scaling.[5] This observation supports the presence of prior sensitization in our case, where the eruption happened on the second day following re-exposure to cefadroxil and mefenamic acid.

Although several systemic drugs were administered in this case, causality was assessed using the Naranjo Scale as a pragmatic pharmacovigilance tool. However, its performance may be limited in routine clinical practice due to confounding factors, and it is primarily designed as a screening tool rather than for definitive causality determination. In comparative studies, both the Naranjo and Liverpool tools demonstrate low specificity, supporting the need for confirmatory testing when feasible.[8] Although the Naranjo Scale is not a diagnostic tool, it remains one of the most commonly used screening instruments for evaluating the causal relationship between a suspected drug and an adverse drug reaction when interpreted alongside clinical findings, with a reported sensitivity of approximately 81.2%.[8] Based on the available literature, β-lactam antibiotics are most often implicated in SDRIFE and are therefore the most likely culprits in this case. Other medications, including mefenamic acid, should be considered possible contributors pending confirmation through tests such as patch testing. Caution is warranted when labeling multiple drugs as definite allergens without confirmation, as this may result in unnecessary restriction of future treatment options.

SDRIFE is typically associated with a single identifiable trigger, with β-lactam antibiotics—particularly amoxicillin—accounting for approximately 45–50% of reported cases. Other recognized causes include iodinated contrast media and antihypertensive agents.[9] In contrast, this case involved exposure to multiple systemic drugs, creating a more complex causality scenario. Reports of SDRIFE in the setting of polypharmacy remain limited, and attributing the reaction to a single agent becomes challenging when multiple potential triggers are present. This highlights the importance of careful reconstruction of the drug exposure timeline, a structured approach to causality assessment, and underscores the need for systematic pharmacovigilance in clinical practice.

Histopathological examination in the present case revealed moderate spongiosis with basal layer hyperpigmentation and mild perivascular lymphocytic infiltration. Although these findings are non-specific for SDRIFE, they may have been influenced by treatment administered prior to biopsy. According to the literature, the histopathological features of SDRIFE are variable, with the most commonly reported findings including superficial perivascular lymphocytic infiltration, dermal eosinophils, and epidermal spongiosis.[5] Therefore, the histopathological features observed in this case remain supportive of the diagnosis of SDRIFE when correlated with the characteristic clinical presentation. Histopathological evaluation is particularly recommended in atypical presentations of SDRIFE, such as the presence of pustular or bullous lesions without systemic symptoms, to rule out alternative diagnoses.[10]

The pathomechanism underlying the predilection for flexural involvement in SDRIFE remains unclear.[10] One proposed mechanism suggests accumulation of the causative drug or its metabolites in apocrine glands located in flexural areas, leading to local toxicity and subsequent keratinocyte injury. This process may be further exacerbated by occlusion, friction, and sweating, which can increase drug concentration on the skin surface and further amplify the local inflammatory response. In addition, the pharmacological interaction with immune receptors concept (p-i concept) has been proposed, that certain drugs can bind to T-cell receptors directly and non-covalently without requiring major histocompatibility complex (MHC) molecules to process or present antigens.[11] These mechanisms may explain the characteristic distribution of lesions observed in SDRIFE. The proposed pathophysiological pathway of SDRIFE is summarized in FIGURE 4.

TABLE 2. Diagnostic Checklist for SDRIFE Based on Hausermann Criteria

Diagnostic criteria (Hausermann *et al.*,)[6]	Finding in this case	Fulfillment
Exposure to a systemically administered drug (initial or repeat dosing)	History of exposure to multiple systemic drugs	√
Sharply demarcated erythema in gluteal and/or inguinal regions	Well-demarcated erythematous lesions in gluteal and inguinal regions	√
Involvement of at least one additional flexural area	Axillae, antecubital fossae, popliteal fossae	√
Symmetrical distribution of lesions	Bilateral and symmetrical involvement of flexural areas	√
Absence of systemic symptoms	No fever, lymphadenopathy, or organ involvement	√

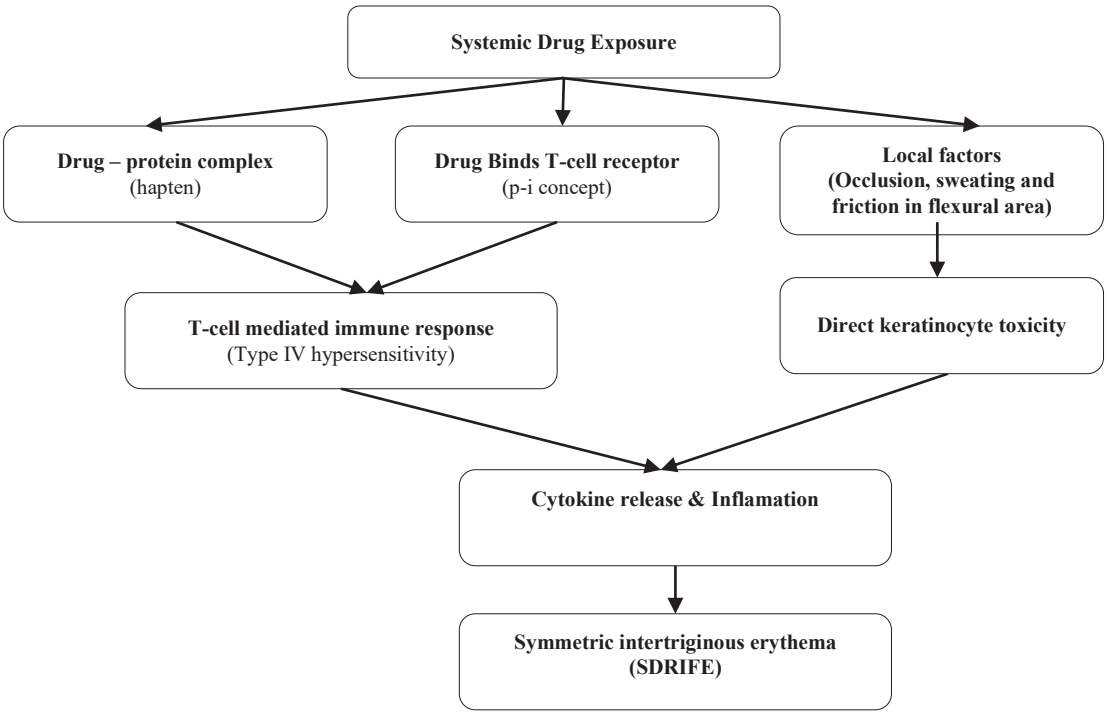

FIGURE 4. Proposed pathophysiological mechanism of symmetrical drug-related intertriginous and flexural exanthema (SDRIFE).

TABLE 3. Comparison of SDRIFE with other drug-induced cutaneous adverse reactions

Case	Drug exposure/ Onset	Clinical manifestations	Systemic Symptoms	Mucosal Involvement	Histopathology
SDRIFE[5,6]	First or repeated doses of systemic drug exposure	Well-defined gluteal/ perianal erythema and/ or V-shaped inguinal erythema; involvement of ≥1 additional flexural site; symmetrical	Absent	Absent	Non-specific findings; superficial perivascular lymphocytic infiltrate ± mild spongiosis
DRESS[12]	Delayed onset after drug exposure (2–8 weeks)	Polymorphic eruption (morbilliform, urticarial, pustular, bullous); trunk and limbs	Present (fever, lymphadenopathy, organ involvement)	Possible	Variable; interface dermatitis and dermal infiltrates; no pathognomonic features
AGEP[13]	Acute onset, usually 24–48 hours after drug intake	Edematous, erythematous skin with multiple small, non-follicular sterile pustules; frequently begins in intertriginous areas	Present (fever, malaise)	Mild	Subcorneal or intraepidermal pustules with neutrophilic infiltrate
FDE[14]	Re-exposure to offending drug	Well-demarcated erythematous to violaceous patch or plaque with dusky centre; often lips or extremities	Absent	Common	Vacuolar interface dermatitis; necrotic keratinocytes; superficial and deep perivascular lymphocytes and eosinophils; pigment incontinence

SDRIFE: Symmetrical drug-related intertriginous and flexural exanthema; DRESS: drug reaction with eosinophilia and systemic symptoms; AGEP: acute generalized exanthematous pustulosis; FDE: Fixed drug eruption.

The differential diagnoses of SDRIFE include DREESS, AGEP, and FDE. In the present case, DRESS was considered because of eosinophilia and elevation of transaminase, but the absence of fever, lymphadenopathy, mucosal involvement, and systemic organ dysfunction made DRESS unlikely according to RegiSCAR criteria. AGEP was ruled out due to the absence of sterile pustular lesions, fever, and characteristic histopathological findings such as subcorneal or intraepidermal pustules. Fixed drug eruption was considered unlikely because no recurrent lesions occurred at the same anatomical sites following re-exposure to the suspected medications. Collectively, these findings support the diagnosis of SDRIFE in this patient.

The suspected causative drugs were discontinued in this present case, and initiation of systemic and topical corticosteroid therapy leading to clinical improvement within approximately two weeks. In line with previous reports, the principal management of SDRIFE is the identification and discontinuation of the offending drugs. Skin lesions are typically self-limiting. Supportive treatment with topical corticosteroids and emollients can reduce erythema and promote resolution, which generally occurs within approximately three weeks.[3,10,15] Administration of intravenous glucocorticoids may shorten the disease duration. Previous reports have described rapid clinical improvement following treatment with intravenous hydrocortisone, intramuscular and oral antihistamines, and topical corticosteroids, with near-complete resolution of skin lesions within eight days.[15]

Prognosis is generally excellent, and lesions resolve after drug withdrawal, but recurrence on re-exposure has been

reported; therefore, clear documentation and patient counseling are essential to prevent avoidable re-challenge.[5,6] For suspected β-lactam-associated SDRIFE, consideration of class avoidance and structured allergy evaluation (including delayed skin testing) can help address potential cross-reactivity and preserve future treatment options.[11] Management of SDRIFE is centered on prompt identification and discontinuation of the offending drug(s), as the eruption is typically benign and self-limited once exposure stops.[9] In our patient, short-course systemic corticosteroids were used to reduce inflammation, while topical corticosteroids and emollients supported barrier recovery. Although many SDRIFE cases resolve with supportive care alone, systemic corticosteroids may be considered for extensive involvement or significant symptoms, recognizing the limited evidence base and the need to balance benefits against adverse effects.[3,5] Resolution generally occurs within days to a few weeks after withdrawal, consistent with the approximately two-week improvement observed here.[9]

In this case, a drug patch test was planned to identify the suspected causative drugs; however, the patient had not returned for further evaluation at the time of manuscript preparation, which limits causality certainty. The patient was also counseled to avoid medications classified as probable or possible triggers. Drug patch testing is a safe and well-tolerated diagnostic tool used in the evaluation of delayed drug hypersensitivity reactions. Although its overall sensitivity is relatively low, patch testing remains particularly useful in SDRIFE, with reported positivity rates of approximately 52–82%.[11,16] A positive result on patch test can help identify the cause, especially in cases of exposure to multiple drugs.[6,17]

CONCLUSION

We reported a rare case of symmetrical drug-related intertriginous and flexural exanthema (SDRIFE), the diagnosis was established based on clinical history, physical examination, and supportive histopathological findings. This case demonstrates that eruptions confined to flexural and intertriginous areas should be recognized as a distinct manifestation of an adverse drug reaction, particularly SDRIFE. In patients with multiple drug exposure, clinicians should systematically review the medication history and reconstruct a clear chronological drug timeline. Causality assessment tools such as the Naranjo scale can assist in causality assessment, but should be interpreted cautiously to avoid misattribution of multiple agents as the cause. Early recognition and withdrawal of the most likely offending drug remain essential to optimize patient outcomes. Patch testing should be considered, when feasible, to help identify the definite causative agent.

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