Section: Dermatology, Venereology and Leprosy

Case Series

CLINICAL SPECTRUM OF CUTANEOUS ADVERSE DRUG REACTIONS: A CASE SERIES FROM A TERTIARY CARE CENTRE

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ABSTRACT

Background: Cutaneous adverse drug reactions (CADRs) represent a clinically important group of drug-induced mucocutaneous disorders, ranging from self-limiting eruptions to severe cutaneous adverse reactions such as Stevens-Johnson syndrome, toxic epidermal necrolysis, acute generalized exanthematous pustulosis, and drug hypersensitivity syndromes. The objective is to describe the clinical patterns, suspected causative drugs, and immediate outcomes of CADRs observed in a tertiary care dermatology setting.

Materials and Methods: This descriptive case series included 10 patients with clinically diagnosed CADRs. Clinical history, recent medication exposure, latency between drug intake and onset of lesions, morphology, distribution, mucosal involvement, and severity indicators were reviewed. The suspected offending drug was withdrawn, and patients received supportive and symptomatic treatment according to clinical severity.

Results: The series included patients aged 20-65 years. The observed patterns were acute generalized exanthematous pustulosis, symmetrical drug-related intertriginous and flexural exanthema, toxic epidermal necrolysis, drug-induced erythroderma, lichenoid drug eruption, Stevens-Johnson syndrome/toxic epidermal necrolysis overlap, bullous fixed drug eruption, dapsone hypersensitivity syndrome, acneiform eruption, and fixed drug eruption. Suspected culprit drugs included ceftriaxone, omeprazole, diclofenac, antitubercular therapy, an unknown analgesic injection, ofloxacin-ornidazole, dapsone-containing multidrug therapy, and phenytoin.

Conclusion: CADRs show considerable clinical heterogeneity and require careful drug history, early recognition, prompt withdrawal of the suspected medication, and documentation for future avoidance. Severe presentations require rapid referral, multidisciplinary supportive care, and pharmacovigilance reporting.

Keywords: cutaneous adverse drug reaction; severe cutaneous adverse reaction; fixed drug eruption; toxic epidermal necrolysis; acute generalized exanthematous pustulosis; dapsone hypersensitivity syndrome.

INTRODUCTION

Cutaneous adverse drug reactions (CADRs) are unwanted drug-related effects that involve the skin, mucous membranes, hair, or nails. They form a heterogeneous clinical group, with presentations ranging from morbilliform eruptions and urticaria to severe cutaneous adverse reactions (SCARs). Because several CADRs mimic primary dermatoses, the diagnosis depends on a careful temporal

relationship between drug exposure and lesion onset, morphology-based assessment, exclusion of alternate causes, and clinical improvement after dechallenge.[1,2]

Indian and international studies have consistently reported antimicrobials, nonsteroidal anti-inflammatory drugs (NSAIDs), antiepileptics, and antitubercular drugs among important culprits.[2,3] Easy access to over-the-counter medication and self-medication practices further complicate causality assessment, particularly when patients receive injections or combination preparations from outside facilities without documentation.

SCARs such as Stevens-Johnson syndrome (SJS), toxic epidermal necrolysis (TEN), SJS-TEN overlap, drug reaction with eosinophilia and systemic symptoms, and acute generalized exanthematous pustulosis (AGEP) are uncommon but potentially fatal. Their pathogenesis is largely immune mediated, with delayed T-cell-mediated mechanisms playing a central role in several phenotypes.[4,5] Recognition of warning signs such as mucosal erosion, skin pain, extensive epidermal detachment, facial edema, fever, pustulation, and systemic symptoms is essential for early escalation of care.

The present case series describes 10 clinically distinct CADRs encountered in a tertiary care setting. The objective was to document the clinical spectrum, suspected culprit drugs, and practical diagnostic considerations, while emphasizing early withdrawal of the suspected drug, supportive care, patient counselling, and pharmacovigilance reporting.

MATERIALS AND METHODS

This descriptive case series included 10 patients who presented to the Department of Dermatology, Venereology and Leprosy with suspected CADRs. Each patient underwent detailed clinical assessment, including age, sex, presenting symptoms, morphology and distribution of lesions, mucosal involvement, timing of onset, recent drug exposure, previous similar reaction, and available treatment records.

Causality was assessed clinically using the temporal association between drug intake and symptom onset, improvement after withdrawal of the suspected drug, recurrence history where available, and exclusion of common alternative diagnoses. Formal rechallenge was not attempted because of safety concerns, particularly in severe reactions. Structured tools such as the Naranjo probability scale can support standardized causality assessment, but clinical judgement remains central in dermatological practice.[11]

Management consisted of immediate discontinuation of the suspected culprit drug, symptomatic treatment, local skin care, infection prevention where required, and systemic/supportive care according to severity. Patients with mucosal involvement, epidermal detachment, or systemic symptoms were considered to require urgent monitoring and multidisciplinary care.

RESULTS

The study included 10 patients aged between 20 and 65 years. The clinical spectrum was broad and included both localized and generalized reactions. Antibiotics, NSAIDs, antitubercular therapy, antiepileptics, proton pump inhibitors, and dapsone-containing multidrug therapy were among the suspected culprits. [Table 1] summarizes the demographic profile, implicated drug, latency, clinical diagnosis, and major clinical features.

Table 1: Clinical profile of the 10 cases of cutaneous adverse drug reactions

Case	Age/Sex	Suspected drug/exposure	Latency	Clinical diagnosis	Key clinical features
1	35/F	Injection ceftriaxone for fever	5 days	Acute generalized exanthematous pustulosis	Multiple pustular lesions with peeling over the body
2	58/F	Omeprazole for epigastric pain	5 days	Symmetrical drug-related intertriginous and flexural exanthema / baboon syndrome	Raw areas over lips with hyperpigmented lesions over axilla and groin
3	65/F	Injection diclofenac for fever	3 days	Toxic epidermal necrolysis	Generalized skin peeling with erosions over lips and extensive epidermal involvement
4	51/M	Unknown over-the-counter drug	5 days	Drug-induced erythroderma	Generalized scaling and diffuse erythematous involvement
5	55/F	Antitubercular treatment	Not specified	Lichenoid drug eruption	Generalized hyperpigmented lichenoid lesions over body
6	24/M	Unknown injection for pain	3 days	SJS-TEN overlap	Peeling of skin with widespread dusky lesions and epidermal injury
7	45/M	Ofloxacin-ornidazole	3 days	Bullous fixed drug eruption	Fluid-filled lesions leaving raw areas over lips and genitalia
8	43/M	Dapsone-containing MB-MDT blister pack for Hansen's disease	2 weeks	Dapsone hypersensitivity syndrome	Erythematous papules and plaques over face and upper limbs
9	20/M	Long-term phenytoin for epilepsy	2 weeks	Acneiform eruption	Multiple erythematous papules over back
10	35/M	Ofloxacin-ornidazole for fever and vomiting	3 days	Fixed drug eruption	Hyperpigmented patches over limbs and genitalia; previous similar episode

Three patients had severe cutaneous adverse reactions, including TEN, SJS-TEN overlap, and AGEP. Two patients had fixed drug eruption variants associated with ofloxacin-ornidazole, and one of them reported a previous similar episode, supporting recurrence at fixed sites. One patient developed features consistent with dapsone hypersensitivity syndrome after initiation of dapsone-containing multidrug therapy. Prompt withdrawal of the suspected offending drugs and supportive dermatological care were central to management.

Case Presentations

Case 1: Acute generalized exanthematous pustulosis after ceftriaxone

A 35-year-old female presented with multiple pus-filled lesions over the body and skin peeling following treatment for fever. She had received injection ceftriaxone five days before onset. The morphology of rapidly appearing pustules over erythematous and desquamating skin was consistent with AGEP, a reaction commonly associated with antibiotics.[6]

Figure 1. Acute generalized exanthematous pustulosis with widespread pustular eruption and desquamation after ceftriaxone exposure.

Case 2: Symmetrical drug-related intertriginous and flexural exanthema after omeprazole

A 58-year-old female presented with raw areas over the lips and hyperpigmented lesions involving the axilla and groin for five days after omeprazole intake for epigastric pain. The flexural distribution was compatible with symmetrical drug-related intertriginous and flexural exanthema, previously referred to as baboon syndrome.[7]

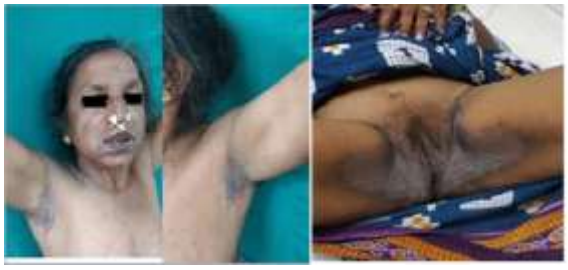

Figure 2. Flexural and intertriginous involvement compatible with symmetrical drug-related intertriginous and flexural exanthema.

Case 3: Toxic epidermal necrolysis after diclofenac injection

A 65-year-old female presented with generalized skin peeling and erosions over the lips for three days after receiving injection diclofenac from a local practitioner for fever. The extensive epidermal detachment and mucosal involvement were suggestive of TEN, a dermatological emergency requiring rapid withdrawal of the offending drug and intensive supportive care.[5]

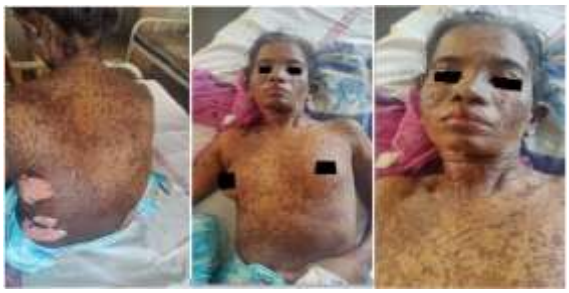

Figure 3. Toxic epidermal necrolysis showing extensive dusky lesions, epidermal detachment, and mucosal involvement after diclofenac exposure.

Case 4: Drug-induced erythroderma after unknown over-the-counter medication

A 51-year-old male presented with generalized scaling for five days after taking an unknown over-the-counter medication. The diffuse erythema and scaling were clinically diagnosed as drug-induced erythroderma. In such patients, the inability to identify the exact drug highlights the need for rational prescribing, documentation, and patient education.[1,3]

Figure 4. Drug-induced erythroderma with generalized scaling following an undocumented over-the-counter medication.

Case 5: Lichenoid drug eruption after antitubercular therapy

A 55-year-old female presented with generalized hyperpigmented lesions over the body after receiving antitubercular treatment. The clinical morphology was consistent with lichenoid drug eruption. Antitubercular agents have been reported as uncommon but recognized triggers of lichenoid eruptions.[12]

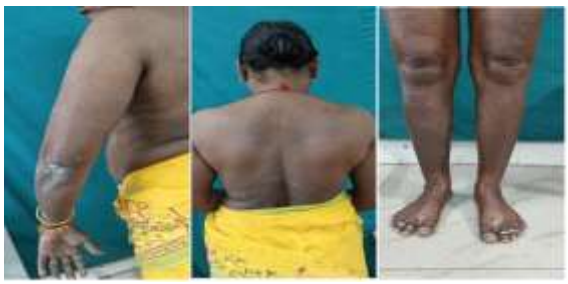

Figure 5. Lichenoid drug eruption presenting as generalized hyperpigmented lesions after antitubercular therapy.

Case 6: Stevens-Johnson syndrome/toxic epidermal necrolysis overlap after unknown analgesic injection
A 24-year-old male presented with peeling of skin for three days after an unknown injection for pain. The clinical picture was diagnosed as SJS-TEN overlap. This diagnosis is considered when epidermal detachment lies between SJS and TEN ranges, and early recognition is critical because morbidity is substantial.[5]

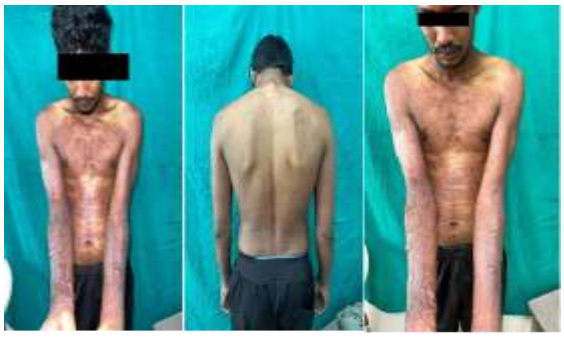

Figure 6. SJS-TEN overlap after an unknown analgesic injection, showing widespread dusky lesions and epidermal injury.

Case 7: Bullous fixed drug eruption after ofloxacin-ornidazole
A 45-year-old male presented with fluid-filled lesions that ruptured to leave raw areas over the lips and genitalia for three days. He had taken ofloxacin-ornidazole. The localized bullous lesions and genital involvement were diagnosed as bullous fixed drug eruption, a pattern known to recur at the same sites after re-exposure.[8]

Figure 7. Bullous fixed drug eruption involving mucosal and genital sites after ofloxacin-ornidazole intake.

Case 8: Dapsone hypersensitivity syndrome after multidrug therapy for Hansen's disease
A 43-year-old male diagnosed with indeterminate Hansen's disease was receiving multibacillary multidrug therapy blister packs for two weeks. He presented with erythematous papules and plaques over the face and bilateral upper limbs. The diagnosis was dapsone hypersensitivity syndrome. This syndrome is classically associated with fever, rash, lymphadenopathy, and hepatic involvement; dapsone should be stopped promptly when suspected.[9,10]

Figure 8. Dapsone hypersensitivity syndrome showing erythematous papules and plaques following dapsone-containing multidrug therapy.

Case 9: Acneiform eruption associated with long-term phenytoin therapy
A 20-year-old male receiving phenytoin for epilepsy for eight years presented with multiple erythematous papules over the back for two weeks. The lesions were diagnosed as an acneiform drug eruption. Antiepileptic medications are well-recognized contributors to CADRs and can produce both mild and severe phenotypes.[2,3]

Figure 9. Acneiform eruption with erythematous papules over the back in a patient on long-term phenytoin therapy.

Case 10: Fixed drug eruption after ofloxacin-ornidazole

A 35-year-old male presented with hyperpigmented patches over the upper limb, lower limb, and genitalia for three days after taking ofloxacin-ornidazole for fever and vomiting. He reported similar lesions in the past. The recurrent site-specific hyperpigmented lesions supported the diagnosis of fixed drug eruption.[8]

Figure 10. Fixed drug eruption with hyperpigmented patches after ofloxacin-ornidazole intake, with history of similar past reaction.

DISCUSSION

This case series illustrates the wide morphological spectrum of CADRs seen in routine dermatology practice. The patterns ranged from localized fixed drug eruption and acneiform eruption to severe reactions such as AGEP, TEN, SJS-TEN overlap, and dapsone hypersensitivity syndrome. This heterogeneity explains why CADRs remain clinically challenging, especially when the eruption resembles infection, inflammatory dermatoses, or autoimmune blistering disorders.[1]

The suspected drugs in this series were common agents used in everyday practice, including antimicrobials, NSAIDs, proton pump inhibitors, antitubercular drugs, antiepileptics, and dapsone-containing therapy. Indian studies have similarly identified antimicrobials, NSAIDs, and antiepileptics as frequent drug classes associated with CADRs.[2,3] The two ofloxacin-ornidazole-associated cases highlight the importance of documenting combination preparations, because patients often remember the indication but not the exact formulation.

Temporal association was a strong diagnostic clue in most cases. AGEP after ceftriaxone occurred within five days and was characterized by multiple pustules with desquamation, consistent with the known rapid-onset profile of AGEP.[6] The case of flexural involvement after omeprazole was clinically compatible with SDRIFE, which typically presents with symmetrical involvement of gluteal, anogenital, axillary, or other flexural areas without prominent systemic features.[7]

The severe epidermal necrolysis spectrum requires special attention. The TEN case after diclofenac injection and the SJS-TEN overlap case after an unknown analgesic injection demonstrate how serious reactions may arise after commonly used analgesics. Early discontinuation of the culprit drug, fluid and electrolyte management, wound care, pain control, infection surveillance, and ophthalmology assessment are key components of care.[5] The uncertainty around the exact analgesic in one patient also shows the clinical risk created by undocumented injections and self-medication.

The dapsone hypersensitivity case occurred after initiation of dapsone-containing multidrug therapy. Dapsone hypersensitivity syndrome is a potentially serious multiorgan drug reaction, often reported within the first several weeks of therapy. Previous work has also shown a strong pharmacogenetic association with HLA-B*13:01 in susceptible populations.[9,10] Although genetic screening is not universally available, clinicians should counsel patients starting dapsone to report fever, rash, facial swelling, jaundice, or systemic symptoms promptly.

Fixed drug eruption was represented by both bullous and non-bullous forms. The history of a previous similar eruption in one patient strongly supported recurrence, which is a characteristic diagnostic feature of fixed drug eruption.[8] Clear documentation of the culprit drug and provision of a written avoidance card are important because recurrence can be more extensive or bullous after re-exposure.

Overall, the series reinforces four practical principles: obtain a precise drug history, stop the suspected drug early, assess severity based on mucosal involvement and epidermal detachment, and report significant reactions to pharmacovigilance systems. In addition, structured causality assessment tools such as the Naranjo scale can improve consistency, although rechallenge should be avoided in severe or potentially life-threatening reactions.[11]

Limitations: This case series is limited by its small sample size and descriptive design. Laboratory confirmation, histopathology, formal causality scoring, and long-term follow-up were not uniformly available. Exact drug details were unavailable in cases where patients had received undocumented over-the-counter medication or injections. Therefore, the findings should be interpreted as clinical observations rather than incidence estimates.

CONCLUSION

CADRs can present with diverse and sometimes overlapping morphologies, from localized fixed drug eruption to life-threatening SCARs. Commonly used medications, including antimicrobials, NSAIDs, antiepileptics, antitubercular drugs, proton pump inhibitors, and dapsone-containing regimens, should be considered when evaluating new-onset eruptions. Early recognition, immediate discontinuation of the suspected culprit, supportive care, documentation of drug allergy, patient counselling, and pharmacovigilance reporting are essential to reduce recurrence, morbidity, and mortality.

REFERENCES

1. Mockenhaupt M. Epidemiology of cutaneous adverse drug reactions. Chem Immunol Allergy. 2012;97:1-17. doi:10.1159/000335612.
2. Sharma VK, Sethuraman G, Kumar B. Cutaneous adverse drug reactions: clinical pattern and causative agents--a 6 year series from Chandigarh, India. J Postgrad Med. 2001;47(2):95-99.
3. Patel TK, Thakkar SH, Sharma D. Cutaneous adverse drug reactions in Indian population: a systematic review. Indian Dermatol Online J. 2014;5(Suppl 2):S76-S86. doi:10.4103/2229-5178.146165.
4. Bellon T. Mechanisms of severe cutaneous adverse reactions: recent advances. Drug Saf. 2019;42(8):973-992. doi:10.1007/s40264-019-00825-2.
5. Frantz R, Huang S, Are A, Motaparthi K. Stevens-Johnson syndrome and toxic epidermal necrolysis: a review of diagnosis and management. Medicina (Kaunas). 2021;57(9):895. doi:10.3390/medicina57090895.
6. Szatkowski J, Schwartz RA. Acute generalized exanthematous pustulosis (AGEP): a review and update. J Am Acad Dermatol. 2015;73(5):843-848. doi:10.1016/j.jaad.2015.07.017.
7. Winnicki M, Shear NH. A systematic approach to systemic contact dermatitis and symmetric drug-related intertriginous and flexural exanthema (SDRIFE): a closer look at these conditions and an approach to intertriginous eruptions. Am J Clin Dermatol. 2011;12(3):171-180. doi:10.2165/11539080-000000000-00000.
8. Anderson HJ, Lee JB. A review of fixed drug eruption with a special focus on generalized bullous fixed drug eruption. Medicina (Kaunas). 2021;57(9):925. doi:10.3390/medicina57090925.
9. Agrawal S, Agarwalla A. Dapsone hypersensitivity syndrome: a clinico-epidemiological review. J Dermatol. 2005;32(11):883-889. doi:10.1111/j.1346-8138.2005.tb00865.x.
10. Zhang FR, Liu H, Irwanto A, Fu XA, Li Y, Yu GQ, et al. HLA-B*13:01 and the dapsone hypersensitivity syndrome. N Engl J Med. 2013;369(17):1620-1628. doi:10.1056/NEJMoa1213096.
11. Naranjo CA, Busto U, Sellers EM, Sandor P, Ruiz I, Roberts EA, et al. A method for estimating the probability of adverse drug reactions. Clin Pharmacol Ther. 1981;30(2):239-245. doi:10.1038/clpt.1981.154.
12. Byun JW, Bang CY, Choi GS, Shin J. Lichenoid eruption associated with antituberculous drug; an unusual oral and follicular involvement. Am J Dermatopathol. 2014;36(8):684-685. doi:10.1097/DAD.0b013e3182919e3a.