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Ready Reckoner in Dermatology



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CHAPTER

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Drug Reactions

INTRODUCTION

Drug reactions are among the most common dermatological emergencies encountered in inpatient and outpatient practice. They range from benign, self-limiting eruptions to life-threatening multisystem disorders like SJS/TEN or DRESS. Early recognition and prompt withdrawal of the drug are critical for reducing morbidity and mortality.

CLASSIFICATION OF DRUG REACTIONS

1. Based of onset of rash **(Table 1)**
2. Based on immunological mechanism **(Table 2)**
3. Based on morphology **(Table 3)**

TABLE 1: Classification of drug rash based on onset.

Immediate	Urticaria and anaphylaxis
Delayed	FDE, SJS, TEN

TABLE 2: Classification of drug rash based on immunological mechanism.

Type I (IgE mediated)	Urticaria, angioedema, and anaphylaxis
Type II (cytotoxic)	Drug induced hemolytic anemia, thrombocytopenia
Type III (immune-complex mediated)	Serum sickness, vasculitis
Type IV (delayed type)	FDE, SJS, TEN, Lichenoid rash

TABLE 3: Classification of drug rash based on morphology.

Subtypes	Clinical features	Drugs responsible
Exanthematous eruptions/ morbilliform rash/ maculopapular rash	• Pruritic macules and papules, usually without blistering, pustules and systemic symptoms **(Fig. 1)** • Associated with desquamation • *Rare and serious variant:* Hypersensitivity syndrome reaction or DRESS (discussed under SCAR) **(Table 4)**	B-lactams, NNRTI's, anti-epileptics
Pustular eruption	*Localized form:* • *Generalized variant:* AGEP (acute generalized exanthematous pustulosis) (discussed under SCAR)	B-lactams, anticonvulsants, sulfonamides, calcium channel blockers (diltiazem), terbinafine

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Subtypes	Clinical features	Drugs responsible
Acneiform eruption	• Comedones are absent • Pruritic follicular papules and pustules • Atypical sites like arms and legs • Halogenodermas—acne like pustules, with clear or hemorrhagic bullae	• Glucocorticoids, isoniazid, lithium, androgens, iodides, bromides, epidermal growth factor receptor inhibitors (geftinib, erlotinib) (PRIDE complex) • Halogens like topical or oral iodine, bromide
Urticarial eruption	• Can be associated with angioedema • Presents with pruritus, flushing, nausea, vomiting, diarrhea, nasal congestion • Serum sickness-like reactions—fever, urticarial rash and arthralgia (vs. true serum sickness—presence of vasculitis, renal lesions, reduced complement levels)	• NSAID's and ACE inhibitors • Cefaclor, cefprozil, bupropion, minocycline, rituximab
Fixed drug eruption	Characterized by sharply demarcated oval to round patches which occur at the same location with each exposure to the causative drug **(Fig. 2)**	Antibiotics (fluroquinolones, doxycycline, nitroimidazoles, sulfa drugs, amoxicillin-clavulanic acid, antimalarials), analgesics (naproxen, ibuprofen, mefenamic acid), antipyretics, anticonvulsants (carbamazepine, lamotrigine)
Bullous eruption	• *Bullous FDE*: Blistering may be localized and mild **(Fig. 3)**, or generalized and severe (generalized bullous FDE) • *Drug*: Induced pseudoporphyria and porphyria • *Drug induced bullous pemphigoid:* Clinically indistinguishable from classic bullous pemphigoid • Drug-induced and drug-aggravated pemphigus • Drug induced linear IgA bullous dermatosis	• Sulphonamides, co-trimoxazole, tetracycline antibiotics, terbinafine, NSAID's like etoricoxib, mefenamic acid • Naproxen causes EPP-like pseudoporphyria, other NSAID's, diuretics, retinoids (true PCT can be caused by OCP's, hormone replacement therapy, alcohol) • Diuretics, gold, penicillamine, captopril, DPP-4 inhibitors like gliptins, PD-1 immunotherapies like pembrolizumab, penicillin antibiotics • Drug induced pemphigus (thiol group of drugs) like D-penicillamine, captopril, gold, pyritinol • *Drug-aggravated pemphigus:* Beta-blockers, CCB's, beta-lactam antibiotics, phenobarbital • Vancomycin
Purpuric rash	• Clinical hallmark of cutaneous drug induced vasculitis • Can be associated with hemorrhagic bullae, ulcers and nodules	Propylthiouracil, G-CSF, GM-CSF, minocycline, phenytoin, isotretinoin, anti-TNF agents, anti-coagulants (warfarin)

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Subtypes	Clinical features	Drugs responsible
Papulosquamous eruption	• Drug-induced psoriasis • Drug-induced lupus • Drug-induced pityriasis rosea	• Beta-blockers (practolol, pindolol, atenolol), lithium, chloroquine and hydroxychloroquine, imiquimod, interferons, anti-PD1 drugs, VEGF antagonists • SLE: Procainamide, hydralazine, quinidine, isoniazid, minocycline, methyldopa • SCLE: Terbinafine, antiepileptics, omeprazole, TNF-alpha inhibitors, NSAID's • CCLE: 5-fluorouracil, voriconazole, infliximab • Bupropion, barbiturates, bismuth, clonidine, captopril, gold
Lichenoid eruption	• Pruritic purple papules over trunk and extremities • Mucous membrane and nail involvement usually absent	Beta-blockers, ACE inhibitors, penicillamine, TNF-alpha antagonists
Drug-induced hyperpigmentation	• Serpentine supravenous pattern • Flagellate pigmentation • Photo distributed rash • Reticulate pigmentation • Mucosal pigmentation • Pigmentation of teeth • Pigmentation of nails • Pigmentation of hair	• 5-fluorouracil • Bleomycin, trastuzumab, docetaxel • Minocycline, amiodarone • Diltiazem • Imatinib, sorafenib, sunitinib, retigabine • Minocycline, imatinib, cyclophosphamide • Minocycline, hydroxyurea and docetaxel (blue lunula), silver salts, zidovudine • Chloroquine, PABA, imatinib, cisplatin



FIG. 1: A child presenting with a maculopapular rash due to carbamazepine.



FIG. 2: FDE involving the lower trunk-pigmented type.

Special Mentions

Drug induced photosensitivity: It is a common cutaneous ADR occurring due to the interaction of UV-rays with drugs. It can occur with either systemic or local drug application.



FIG. 3: Bullous FDE after administration of docetaxel (chemotherapy for breast Ca).

Two sub-types are identified drug-induced phototoxic reactions (dose dependent) and drug-induced photo-allergic reactions (delayed type-IV hypersensitivity reactions). **Table 5** summarizes the drugs giving rise to these subtypes.

Drug-induced Vasculitis

Refer to **Table 6**.

CLINICAL EVALUATION AND WORK-UP

History

A detailed history of all prescription and non-prescription drugs, including details of dosage, indication of drug use, trade name

TABLE 4 : Severe cutaneous adverse reactions (SCAR's).

Syndrome	Clinical features	Common drugs
SJS	• Prodromal symptoms of fever, sore throat, conjunctivitis and myalgia • Erythematous macular rash which progresses to form purpuric centres, blisters and bullae **(Fig. 4)** • Pain and tenderness over lesions • < 10% of BSA involved • *10–30% BSA involved*: SJS-TEN overlap • *Mucosal involvement*: Oral, genital, ocular mucosa-presence of erosions and crusts	CBZ (risk factor: HLA-B*1502), phenytoin, lamotrigine, allopurinol (screen for HLA-B*5801 and renal impairment), co-trimoxazole, NSAID's, ART (nevirapine)
TEN	• >10% of BSA involved • Sheets of epidermal detachment **(Fig. 5)**	Same as drugs causing SJS
DRESS	• Fever, rash, lymphadenopathy, abnormal LFT's, renal dysfunction • Mortality due to fulminant hepatitis	CBZ, phenytoin, lamotrigine, allopurinol, dapsone, ART drugs (nevirapine, abacavir)
AGEP	• Presents with small, sterile monomorphic, non-follicular pustules over intertriginous areas and trunk; associated with fever, leukocytosis, systemic involvement—liver, lung, kidney • Generalized desquamation seen on resolution of pustules	B-lactams, anticonvulsants, sulfonamides, calcium channel blockers (diltiazem), terbinafine, quinolones, HCQs

TABLE 5 : Subtypes of drug-induced photosensitivity.

Phototoxicity subtype	Key drug classes (examples)
Hyperpigmentation/Dyschromia	Antiarrhythmics (amiodarone), calcium channel blockers (diltiazem), phenothiazine antipsychotics (chlorpromazine)
Pseudoporphyria	Tetracyclines (tetracycline), loop diuretics (furosemide), NSAIDs (naproxen), azole antifungals (voriconazole)

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Phototoxicity subtype	Key drug classes (examples)
Photo-onycholysis	Tetracyclines (doxycycline), azole antifungals (voriconazole), antifungals (griseofulvin), NSAIDs (diclofenac)
Photo-distributed telangiectasia	SSRIs (escitalopram), calcium channel blockers (amlodipine)
Pellagra-like dermatitis	Antitubercular drugs (isoniazid), antimetabolites (5-fluorouracil), immunosuppressants (azathioprine)
Photoallergy subtype	
Lichenoid Photodermatitis	Thiazide diuretics (hydrochlorothiazide), tetracyclines (doxycycline), phenothiazine antipsychotics (chlorpromazine), nsaids (naproxen)
Photo-distributed erythema multiforme	NSAIDs (naproxen, ketoprofen), chemotherapeutics (paclitaxel)
Subacute/Chronic cutaneous lupus	Antifungals (terbinafine), calcium channel blockers (diltiazem), biologics (anti-TNF-α agents)



FIG. 4: Erythematous discrete macules and papules involving 20% of BSA, with crusting of lips—a case of evolving SJS.

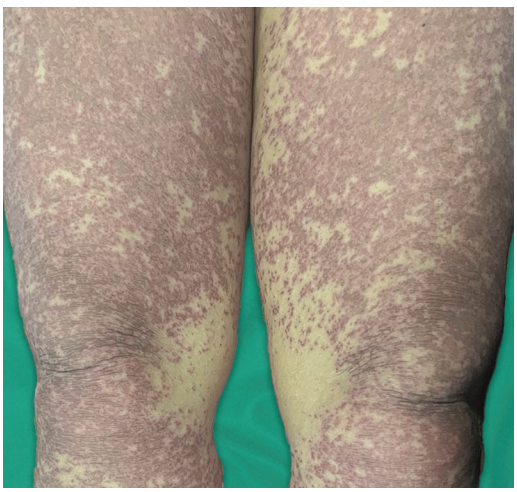

FIG. 5: Confluent papules involving more than 30% BSA suggestive of TEN.

TABLE 6 : Subtypes of drug-induced vasculitis.	
Drug-induced cutaneous leukocytoclastic vasculitis	Antitubercular drugs, NSAID's, metformin, glyburide, telmisartan, tenofovir, clopidogrel, and clomiphene
Drug-induced IgA vasculitis	Infliximab, adalimumab and etanercept, palbociclib
Drug-induced antineutrophil cytoplasmic antibodies-associated vasculitis	• Antithyroid drugs, cocaine/levamisole, and hydralazine • *Pembrolizumab (anti-PD1 inhibitor)*: Granulomatosis with polyangiitis, retinal vasculitis • *Montelukast:* Eosinophilic granulomatosis with polyangiitis
Drug-induced medium vessel vasculitis	Minocycline, vitamin K antagonist acenocoumarol, ergotamine
Drug-induced large vessel vasculitis	Bevacizumab (anti-VEGF), lenograstim (recombinant granulocyte colony-stimulating factor), ipilimumab (CTLA-4 blocker)

with batch number route of administration, frequency, duration and timing of last dose should be elicited (drug causality assessment). A history of aggravating factors like stress, food intake, alcohol consumption, UV exposure, other co-existing dermatoses like chronic urticaria, atopic dermatitis must also be documented.

Examination

A thorough cutaneous examination documenting the percentage of BSA involved, morphology of rash, predominant areas of involvement, oral involvement, involvement of other mucosal surfaces like genital mucosa and conjunctiva must be done. General physical examination including pulse rate, respiratory rate, temperature must be documented. Systemic examination of cardiovascular, pulmonary system must be included. Signs of impending cardiopulmonary collapse like laryngeal or upper airway oedema, hypotension, tachycardia, wheezing must be elicited.

Laboratory Investigations

All routine investigations including liver, renal function tests, complete blood counts, electrolyte levels, ESR, CRP, ANA profile, serum tryptase levels, complement levels, serum IgE levels, skin prick tests and intradermal tests for immediate type 1 hypersensitivity reactions like anaphylaxis and angioedema, skin biopsy for HPE and immunofluorescence studies.

Scoring Systems

The most commonly use causality and severity scoring systems are summarized in **Table 7**.

Management principles: Discontinuation of the offending drug forms the mainstay of treatment in all drug reactions. Substitution of drug must be done with caution, with drugs of unrelated chemical structures. Topical corticosteroids and oral anti-histamines may be used for symptomatic management of mild ADRs. Systemic corticosteroids, supplemented with other steroid sparing immunosuppressives can be used in cases of extensive or moderate ADRs. The management of SCAR's is discussed below.

Management of SJS/TEN

All patients diagnosed with SJS/TEN must be hospitalized, preferably in a set-up with ICU. Evaluation and monitoring of airway, breathing, circulation, urine input/output, and signs of septicemia must be done initially. Supportive therapy in the form of barrier nursing, oral and ophthalmic care, maintaining fluid and electrolyte balance, topical dressings, analgesics, antipyretics and anxiolytics, plays an extremely important role in these cases. Disease modifying therapy in the form systemic corticosteroids, cyclosporine, intravenous immunoglobulin, plasmapheresis, tumor necrosis factor-α inhibitors, granulocyte colony-stimulating factor and N-acetylcysteine have been used with variable results across the globe. High dose of systemic corticosteroids early in the course of the disease, for a short period of time is advocated in resource—poor settings. Use of cyclosporine in a dose of 3–5 mg/kg/body weight for 7–10 days has also proven to be an effective therapy.

Management of DRESS

Systemic corticosteroids are currently the most accepted treatment for DRESS. Early administration at a dose of 1 mg/kg/day, followed by gradual tapering over 4–6 weeks is recommended. The presence of any life-threatening sign (hemophagocytosis, spinal cord failure, encephalitis, liver failure, respiratory failure) warrants the addition of intravenous immunoglobulin (IVIG) at a dose of 2 g/kg for five days.

TABLE 7: Various scoring systems used for ADR's.

Score/System	Purpose	Key parameters	Interpretation
ALDEN (Algorithm of Drug Causality for Epidermal Necrolysis)	*Causality:* SJS/TEN	• Latency • Drug presence on index day • Previous reaction, continued use • Drug notoriety • Alternative causes	>6 very probable; 4–5 probable; 2–3 possible; 0–1 unlikely; <0 very unlikely
SCORTEN (Severity-of-Illness Score for Toxic Epidermal Necrolysis)	*Severity/ Prognosis:* TEN	• Age > 40 years • Presence of malignancy • Heart rate >120 beats/min • Epidermal detachment > 10% BSA on day 1 • Serum urea > 10 mmol/L (≈ 28 mg/dL) • Serum glucose > 14 mmol/L (≈ 252 mg/dL) • Serum bicarbonate < 20 mmol/L	**Score** / **Predicted mortality** 0–1 / 3.2% 2 / 12.1% 3 / 35.3% 4 / 58.3% ≥5 / >90%
Naranjo	*Causality:* ADR (general)	*Ten Qs:* 1. Previous conclusive reports? 2. Event after drug given? 3. Improvement on stopping drug? 4. Recurrence on re-challenge? 5. Alternative causes ruled out? 6. Recurrence with placebo? 7. Toxic drug levels detected? 8. Dose-response relationship seen? 9. Similar reaction in the past? 10. Objective evidence present?	≥9 = Definite; 5–8 = Probable; 1–4 = Possible; ≤0 = Doubtful
RegiSCAR (DRESS)	*Diagnosis:* DRESS	• Fever ≥38.5 °C • Enlarged lymph nodes (≥2 sites) • Eosinophilia • Atypical lymphocytes • Skin rash extent >50% body surface area • Skin rash suggesting DRESS (infiltration, purpura, scaling) • Organ involvement (1 point per organ, max 2) • Resolution ≥15 days • Investigations excluding other causes	≥6 definite; 4–5 probable; 2–3 possible; <2 no case

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Score/System	Purpose	Key parameters	Interpretation
EuroSCAR	*Diagnosis:* SCAR (AGEP, SJS/TEN)	• Clinical morphology of eruption • Latency period between drug intake and rash onset • Presence of fever • Laboratory findings (e.g., neutrophilia in AGEP, eosinophilia) • Histopathology compatible with diagnosis • Course and resolution pattern • Presence or absence of alternative causes	*Score-based:* Definite/ Probable/Possible/No case
Hartwig and Siegel	*Severity:* ADR	• *Level 1*: ADR occurred but no change in treatment required (drug continued) • *Level 2*: ADR required the drug to be stopped or changed, but no additional treatment needed • *Level 3*: ADR required drug to be stopped and additional treatment started • *Level 4(a)*: ADR required hospitalization • *Level 4(b)*: ADR occurred during hospitalization and prolonged the stay • *Level 5*: ADR caused permanent harm/disability • *Level 6*: ADR was life-threatening and required intensive care • *Level 7*: ADR directly or indirectly led to patient's death	Mild (1–2), moderate (3–4), severe (5–7)

Management of AGEP

Systemic corticosteroids, cyclosporine, acitretin, dapsone, rituximab, infliximab, and IVIG have been used.

Drug rechallenge tests or drug provocation tests: Contra-indicated in SCAR's. It is mainly used in scenarios where possible benefit is higher than the risk—patients with a hypersensitivity reaction to common antibiotics like penicillin, beta-lactams, anti-tubercular drugs, anti-retroviral drugs, NSAID's, and anti-diabetic drugs.

Multiple drug hypersensitivity syndrome (MDH): Where a person develops drug allergies to 2 or more chemically different drugs. It is suspected to have an immunological basis and must be differentiated from cross-reactivity which occurs due to structural similarities and common metabolic pathways.

Preventive strategies: HLA testing—

- *HLA-B*1502*: Carbamazepine induced SJS/TEN in Asians (Indian, Thai, and Chinese)

TABLE 8 : Quick reference table for latency periods for various drug reactions.

Hypersensitivity reaction	Time interval
Urticaria, angioedema, anaphylaxis	Within 1–12 hours of exposure
Maculopapular rash	4–14 days
AGEP	1–12 days
SJS/TEN	4–28 days
DRESS	2–8 weeks

- *HLA-B*31:01:* Carbamazepine induced DRESS
- *HLA-B*13.01:* Dapsone hypersensitivity syndrome
- *HLA-B*57:01:* Abacavir hypersensitivity
- *HLA-B*58:01:* Allopurinol induced SJS/TEN
- *HLA-A*32:01:* Vancomycin hypersensitivity

The latency periods for various drug reactions is listed in **Table 8**.

PRACTICE PEARLS

- *Early recognition and stopping culprit drug saves lives*: Always suspect a drug reaction in any new rash appearing within days to weeks of starting a medication, especially in the context of systemic symptoms.
- *Think beyond skin*: Assess for systemic involvement in all suspected drug reactions; check liver, renal, hematologic, and respiratory status.
- *SCAR red flags*: Mucosal involvement, widespread blistering, high fever, and rapidly progressive lesions mandate immediate hospitalization.
- *Avoid rechallenge in SCAR*: Never reintroduce the suspected drug in severe cutaneous adverse reactions.
- *HLA testing saves trouble*: Use genotype screening in high-risk populations before prescribing known high-risk drugs (e.g., HLA-B*1502 before carbamazepine in Asians).
- *Class over brand*: Avoid other drugs in the same chemical class once a reaction is confirmed, unless cross-reactivity is confidently excluded.
- *Document thoroughly*: Maintain detailed drug histories in the medical record to avoid future exposure and for medicolegal safety and provide written drug allergy cards listing culprit drugs and potential cross-reactive agents.

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