

Common Skin Diseases

A Clinical Approach





Brig. SP Chattopadhyay

JAYPEE

Contents

Chapter 1. Acne Rosacea 1
Bidisa Guha Niyogi

Chapter 2. Acne Vulgaris 3
SP Chattopadhyay

Chapter 3. Adverse Drug Reaction 6
Nilendu Sarma, SP Chattopadhyay

Urticarial Drug Eruptions 7; Lichenoid Drug Eruptions 7; Fixed Drug Eruptions 8; Photosensitivity Drug Eruptions 8; Photoallergic Reaction 8; Phototoxic Reaction 8; Bullous Drug Eruptions 9; Drug-Induced Pemphigus 9; Drug-Induced Pemphigoid 9; Erythema Multiforme (EM) 9; Toxic Epidermal Necrolysis (TEN) 10; Drug-Induced Lupus 11; Psoriasiform Reaction 11; Exfoliative Dermatitis/ Erythroderma 11; Other Patterns of Drug Reaction 11

Chapter 4. AIDS 13
Nilendu Sarma, SP Chattopadhyay

Acute/ Primary HIV Infection (PHI) 13; Skin 13; Oral Hairy Leukoplakia (OHL) 14; Oral Ulcer 14; Bacillary Angiomatosis 15; Kaposi's Sarcoma 15

Chapter 5. Bullous Pemphigoid 16
Nilendu Sarma

Chapter 6. Bacterial Infection (Pyoderma) 18
SP Chattopadhyay

Chapter 7. Common Tumor of Skin 20
SP Chattopadhyay

Squamous Cell Carcinoma 20; Basal Cell Carcinoma—Rodent Ulcer 21; Malignant Melanoma 21; Paget's Disease of Nipple 22; Benign Epidermal Tumor 22; Disorders of Sweat Glands 24; Disorder of Apocrine Glands 25; Bromhidrosis and Chromhidrosis 25

Chapter 8. Common Oral Conditions **26**
Rintu Guha Niyogi
Oral Lichen Planus 26; Aphthous Ulcers 27; Pemphigus 28; Other Common Oral Conditions 29

Chapter 9. Cutaneous Vasculitis **31**
Nilendu Sarma
Henoch-Schönlein Purpura 33; Cryoglobulinemic Vasculitis 34; Polyarteritis Nodosa (PAN) 35; Cutaneous Polyarteritis Nodosa 35; Wegener's Granulomatosis 36; Churg-Strauss Syndrome 36; Urticarial Vasculitis 37

Chapter 10. Disorders of Blood Vessels **38**
SP Chattopadhyay
Small Blood Vessels 38; Erythromelalgia 38; Livido Reticularis 38

Chapter 11. Diseases of Nail **40**
SP Chattopadhyay
Clubbing of Nail 40; Koilonychia 40

Chapter 12. Dermatomyositis **42**
Nilendu Sarma

Chapter 13. Eczematous Dermatitis **46**
Bidisa Guha Niyogi
Etiological Classification 46; Morphological Classification 47; Clinical Classification 47; Histopathological Features 47; Investigations 47; Different Types of Eczema 48; Discoid Eczema (Nummular Eczema) 50; Asteatotic Eczema 51; Lichen Simplex Chronicus 51; Contact Dermatitis 52; Allergic Contact Dermatitis 52

Chapter 14. Epidermal Appendages **54**
SP Chattopadhyay
Hair 54; Androgenic Alopecia 54; Alopecia 55

Chapter 15. Epidermolysis Bullosa (EB) **56**
Nilendu Sarma, SP Chattopadhyay

Chapter 16. Erythema Nodosum **59**
Nilendu Sarma
Fungal Infections 59

Chapter 17. Fungal Infection **62**
SP Chattopadhyay
Dermatophyte Infection 62; Antifungal Therapy 63; Candidiasis 64; Tinea Versicolor 64

Chapter 18. Herpes Simplex Orofacial **66**
SP Chattopadhyay

Chapter 19. Herpes Zoster and Varicella **68**
SP Chattopadhyay
Herpes Zoster 68

Chapter 20. Human Scabies **70**
SP Chattopadhyay
Human Scabies 70; Pediculosis 71; Papular Urticaria 72; Wasps and Bees 72; Onchocerciasis 72

Chapter 21. Melanin Pigmentation and its Disorder **73**
SP Chattopadhyay
Vitiligo 73

Chapter 22. Hypermelanosis of Skin **76**
SP Chattopadhyay
Color of Skin 76; Mechanism of Melanin Biosynthesis 76; Color Modulation 76; Skin Lightening Ingredients 76; Etiology of Hypermelanosis 77; Riehl's Melanosis 77;

Chapter 23. Melasma (Chloasma) **79**
SP Chattopadhyay
Melasma 79

Chapter 24. Social Prejudice of Vitiligo Around Us **81**
SP Chattopadhyay

Chapter 25. HIV Infection: AIDS and Dermatological Disorder **82**
SP Chattopadhyay
Diagnosis 82; Prevention 82; Management 83

Chapter 26. Leg Ulcers **84**
SP Chattopadhyay

Chapter 27. Leprosy **86**
SP Chattopadhyay

Chapter 28. Lichen Planus **91**
SP Chattopadhyay

Chapter 29. Lupus Erythematosus **95**
SP Chattopadhyay

Chapter 30. Molluscum Contagiosum **98**
SP Chattopadhyay
Definition 98; Epidemiology 98; Clinical Picture 98; Differential Diagnosis 98; Treatment 99

Chapter 31. Neurofibromatosis and Few Other Genetic Disorders **100**
SP Chattopadhyay
Tuberous Sclerosis 101; Incontinentia Pigmenti 101; Xeroderma Pigmentosum 102; Ehlers-Danlos Syndrome 102

Chapter 32. Newer Drugs in Dermatology **103**
Bidisa Guha Niyogy
Topical Immunosuppressives 103; Miscellaneous Drugs 104

Chapter 33. Nutritional Deficiency and Skin Disorders **106**
SP Chattopadhyay
Vitamin A 107

Chapter 34. Patch Test **109**
Nilendu Sarma, SP Chattopadhyay

Chapter 35. Pemphigus Vulgaris **111**
Nilendu Sarma
Pemphigus Vulgaris 111

Chapter 36. Pityriasis Rosea **113**
SP Chattopadhyay

Chapter 37. Physical Methods of Treatment **115**
SP Chattopadhyay

Chapter 38. Porphyria **118**
Nilendu Sarma, SP Chattopadhyay
Porphyria Variagata 118

Chapter 39. Psoriasis **120**
SP Chattopadhyay

Chapter 40. Psychocutaneous Problems **124**
SP Chattopadhyay

Chapter 41. Seborrhea and Seborrheic Dermatitis **126**
SP Chattopadhyay
Seborrheic Keratoses 127

Chapter 42. Scleroderma/Morphea **128**
Nilendu Sarma
Plaque-Type Morphea 128; Linear Lesions 129

Chapter 43. Side Room Procedures **131**
SP Chattopadhyay
Fungal Infection 131; Scabies 131; Tzanck Smear 131; Prick Test 131; Patch Test 131; Skin Biopsy 132; Gram Stain 132; Ziehl-Neelsen Staining for *M. leprae* 132; Technique for Staining 132

Chapter 44. Structure and Function of Skin **133**
SP Chattopadhyay
Ground Substance 135; Muscles 135; Vessels 135; Nerves 135; Function of Skin 135; Immunological Function of Skin 136

Chapter 45. Subcutaneous Mycosis **138**
Nilendu Sarma
Subcutaneous Mycosis 138; Sporotrichosis 138; Mycetoma/Madura Foot 139; Chromoblastomycosis 140; Pheohyphomycosis 141; Lobomycosis 141; Rhinosporidiosis 141; Subcutaneous Zygomycosis 141; Systemic Mycosis 142; Endemic Systemic Mycosis 142; Cryptococcosis 142; Aspergillosis 142; Blastomycosis 142; Histoplasmosis 143

Chapter 46. Symptomatology **144**
SP Chattopadhyay
Pruritus 144; Primary and Secondary Lesions 145

Chapter 47. Syphilis **146**
SP Chattopadhyay
Acquired Syphilis 146; Syphilis Early Primary 146; Secondary Syphilis 147; Types of Skin Lesions 147; Syphilis Congenital 148; Late Congenital Syphilis 148; Jarisch Herxheimer Reaction 148; Syphilis Latent 148; Neurosyphilis 149; Syphilis of Spinal Cord 149

Chapter 48. Systemic Diseases and Skin **150**
SP Chattopadhyay
Liver Disease 150; Renal Disease 150; Diabetes Mellitus 151; Thyroid Hormone 151; Metabolic Disorder 152

Chapter 49. Systemic Sclerosis (SSc) **153**
Nilendu Sarma
Definition 153; Pathogenesis 153; Pathology 153; Clinical Features 153; Treatment 154

Chapter 50. Topical Dermatological Preparations **155**
SP Chattopadhyay
Baths 155; Lotions 155; Paste 155; Ointments 156; Powder 156; Paints 156; Emollients 156; Shampoos 156;

Barrier Preparations 156; Sunscreens 156; Antiperspirants 156; Topical Corticosteroids 157

Chapter 51. Tuberculosis of Skin **159**
SP Chattopadhyay
Classification 159; Investigation 159; Differential Diagnosis 160; Diagnosis 161; Treatment 161

Chapter 52. Urticaria and Angioedema **162**
Nilendu Sarma
Definition 162; Classification 162

Chapter 53. Verrucae Vulgaris and Other Type of Warts **166**
SP Chattopadhyay

Chapter 54. Micro-photographic Histopathological Interpretation of Cutaneous Diseases **169**
AK Banerjee

Further Reading *172*
Index *173*

Chapter

3

Adverse Drug Reaction

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Adverse drug reaction: Unintended adverse effect of a drug at normally used doses is called adverse drug reaction.

Causal Relationship

It is not easy to prove the causal link between a administered drug and the reaction. There are many factors those need to be properly addressed and excluded before making a conclusion. The basic guidelines are:

The drug is proved to have the adverse effect.

An alternate causative factor is unlikely.

Onset of the eruption should have a temporal relationship with drug ingestion. Reaction should resolve after drug therapy is stopped.

Reaction should recur following rechallenge (should not be attempted as it is risky).

There are some special in vitro tests like radioallergosorbent testing, assay of drug-specific antibodies, and lymphocyte transformation test, etc. to confirm drug hypersensitivity but they are not available in most clinical set up.

Etiopathogenesis

Mechanisms through which the reaction occurs can be immunologic or nonimmunologic.

All four types of hypersensitivity reactions can be involved in the immunologic group.

Mechanisms vary in the different types of nonimmunologic drug reactions.

Few examples of nonimmunologic drug reaction are overdose, side effects, drug interaction, cumulative toxicity, metabolic, idiosyncrasy, intolerance, etc.

There are other factors those can complicate the adverse reaction of a drug like—

- Concurrent infection especially cytomegalovirus, human herpes virus 6 and some lymphotropic viruses.
- Connective tissue disease.
- Immune dysregulation (e.g. HIV infection).

Clinical Presentation

Skin manifestations are hallmark of most types of drug reactions although reactions without skin involvement can occur. Cutaneous lesions often closely simulate cutaneous eruptions of some systemic diseases.

There are many types of reactions classified mainly according to the skin manifestations.

Exanthematous or Maculopapular Drug Eruptions (Fig. 3.1)

- Most common form of drug eruptions.



Fig. 3.1. Exanthematous or maculopapular drug eruptions

- Can resemble to all patterns viral exanthems like scarlatiniform, rubelliform and morbilliform.
- Develop centripetally from trunk to the periphery mostly symmetrically. Frequently the face is spared but is not the rule.
- Even constitutional symptoms may be present.
- Eosinophilia in blood and in dermis on histology is often noted.
- Rapid clearance of lesions on withdrawal of the drug. But rarely progress to exfoliative dermatitis if the drug is continued.

Etiology

Most drugs can cause these types of reactions. Common offenders are Penicillin group, Phenylbutazone, Sulfonamide, Gentamicin, Anticonvulsants like Phenytoin, Carbamazepine, gold, Amphotericin B, oral hypoglycemic agents, Thiazides, Barbiturates, Benzodiazepines, Isoniazid, Phenothiazines, etc.

Differential Diagnosis

Viral exanthem, acute graft-versus-host disease (GVHD), CTD, etc.

Urticarial Drug Eruptions

- Usually the second most common form.
- Occur within 36 hours after a drug administration.
- Rarely angioedema is associated.
- Due to Type I, Type III reactions and a delayed-Type hypersensitivity reaction.
- Main inflammatory mediators are histamine and also the prostaglandins and IL-1. Also the nonimmunologic mechanisms are involved in some.

Etiology

Common drugs causing urticarial eruptions are Aspirin and Salicylates, NSAIDs, Penicillin groups, Aminoglycosides, Sulfonamide, Phenytoin and Carbamazepine, antifungal agents (i.e. Ketoconazole), oral hypoglycemic agents, captopril, radiocontrast materials, animal sera, food and drug additives, etc.

Lichenoid Drug Eruptions

- Simulate classical lichen planus. However they are more psoriasiform.
- Oral involvement is not common.
- Develop after weeks to months after taking the drug.
- Often left with marked postinflammatory hyperpigmentation.
- Resolution is slow.
- Histological features are like idiopathic LP. However focal parakeratosis and cytoid bodies in the upper two layers suggest drug as etiology.
- Lesions can be predominantly photo-distributed with some drugs, e.g. Quinine, Quinidine, Thiazide diuretics, Frusemide, Chlorpromazines, etc.

Etiology

Quinine, Quinidine, ACE inhibitors, β-blockers, Thiazide diuretics, Frusemide,

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Chlorpromazines, hypolipidemic agents, NSAIDs, antihistaminics, Methyldopa, PPI, gold, etc.

Even contact with some chemical like paraphenylenediamines can cause lichenoid eruptions.

Fixed Drug Eruptions

- Circumscribed violaceous or dusky erythematous patches.
- Recur at same sites.
- New sites can be involved in addition to the previous one at rechallenge.
- Morphology—Urticarial, bullous, eczematous, morbilliform and even generalized pigmentation. Nonpigmented lesions are rare.
- Pathogenesis—Upregulated intercellular adhesion molecule in keratinocytes and endothelial cells.
- Histology—Resembles erythema multiforme. There is interface dermatitis along with acanthosis, hypergranulosis, hyperkeratosis, and rete ridge accentuation, etc.

Etiology

Sulfonamides, Trimethoprim, Tetracycline groups, Penicillin groups, Dapsone, Antimalarials, NSAID, Metronidazole, Barbiturates, anticonvulsants, etc. Often cross-reactivity between drugs is noted.



Fig. 3.2. Furunculosis or boils (in scalp) develop in sites with preexisting folliculitis

Photosensitivity Drug Eruptions

It can be photoallergic and phototoxic reaction.

Photoallergic Reaction

Pathogenesis

Drugs absorb ultraviolet light (UVA or UVB) and become immunologically active. Rash appear through a Type IV hypersensitivity mechanism.

Clinical Feature

It takes around 24 hours. Although rash is localized to the exposed areas rarely, it may become generalized later.

Phototoxic Reaction

Pathogenesis

Skin is directly damaged, via reactive species generation.

Clinical Feature

- Lesions appears rapidly even after first exposure.
- Consist of blister, desquamation, and hyperpigmentation.
- Confined exclusively to sun-exposed skin.

Both types of reaction can be present in a same individual.

Etiology

Amiodarone, Phenothiazines, Psoralens, Sulfonamides, Thiazide diuretics, NSAID,

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Quinidine, oral hypoglycemics, sunscreens, etc.

Bullous Drug Eruptions (Fig. 3.3)

There are multiple entities those can present with blisters and many of these are dermatological emergency.



Fig. 3.3. Bullous drug eruptions

The list consists of fixed drug eruption, erythema multiforme, Stevens-Johnson syndrome, toxic epidermal necrolysis, drug-induced autoimmune bullous disorders (like pemphigus, pemphigoid, linear IgA bullous dermatosis) and pseudoporphyria cutanea tarda.

Drug-induced Pemphigus

- Can resemble pemphigus vulgaris, pemphigus herpetiformis, subcorneal pustular dermatosis, etc.
- Morbilliform or urticarial eruption followed by a bullous eruption strongly suggests a drug-induced pemphigus.

Pathogenesis

Drugs containing a thiol group or those transform to a thiol form after metabolism in body are common etiology (80% of all cases) than drug without thiol group.

Etiology

- Thiol (sulfhydryl) group containing drugs like Captopril, Penicillamine, and gold sodium thiomalate.
- Nonthiol drugs are Penicillin groups, Rifampicin, Cefadroxyl, Piroxicam, Phenobarbital, Phenylbutazone, etc.

Drug-induced Pemphigoid

Clinically mimic classical bullous pemphigoid, cicatricial pemphigoid or epidermolysis bullosa (EBA).

Etiology

Furosemide, Penicillamine, Penicillin, Salicylazosulfapyridine, Sulfasalazine, and Phenacetin, etc.

Drug-induced Porphyria Cutanea Tarda/ Pseudo PCT

Drug-induced PCT is called pseudo PCT. They have similar clinical feature, light microscopic, and direct immunofluorescent findings. However porphyrin metabolism in patients with pseudo PCT is usually normal. They are seen after high dose frusemide, NSAIDs, sulfonylureas, etc.

Erythema Multiforme (EM)

There are two subtypes—Erythema multiforme minor and major.

Erythema multiforme minor

- This is the majority.
- Small-sized papules or maculopapules with gradual increase upto sizes of 1–3

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cm appear in crops of lesions with acral preponderance.
- Target lesion is diagnostic. It is classically less than 3 cm in diameter, circular with three clinically obvious zones—Central purpura or erythema, middle pale edematous area and outer erythematosus zone. Atypical target lesions are also present.
- Lesions usually heal within 2 weeks.
- Usually mucous membrane and systemic involvement and vesicobullous lesions are absent.

Erythema Multiforme Major

- Severe variety.
- After a prodrome, disease starts with sudden onset, rapidly progress with successive crops that continue even upto 4 weeks.
- Mucous membrane and systemic involvement is present in most of the cases.
- Bullous lesions, target lesions. are almost always present.

Differential Diagnosis

Stevens-Johnson syndrome (SJS) (Fig. 3.4), urticarial vasculitis, pemphigoid, Kawasaki's disease, SLE.

Toxic Epidermal Necrolysis (TEN)

- Usually there is a prodrome.
- Small macular erythema or morbilliform erythema rapidly progress and coalesce to involve extensive areas with erythema and marked skin tenderness.
- Most distinctive features are the easy separation of skin. This can be elicited with mild sliding force especially over a bony prominence called Nikolsky sign.
- Blisters are not distinctive feature.
- Mucosa like oral mucosa, conjunctiva, genital mucosa are involved in almost all cases.
- Prognosis is poor unless treated in ICU like a burn case.
- Blindness can develop from corneal and conjunctival involvement, which is preventable and advice is to be taken from ophthalmologist.

Offending Drugs

Antiepileptics, Sulfonamides, NSAIDs, ATD, etc.

Differential Diagnosis

SSSS
TSS.



Fig. 3.4. Stevens-Johnson syndrome (Drug reactions differential diagnosis)

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Drug-induced Lupus

- Unlike most of the drug reactions those mimic systemic disease, skin disease is less frequent in drug-induced LE than in idiopathic LE. Features of discoid LE are rare.
- Pulmonary manifestations are more common.

Pathogenesis

- In most of cases the mechanism is unknown.
- Procainamide and Hydralazine inhibit T-cell DNA methylation. This stimulates T-helper lymphocytes leading to antibody production against histones or histone DNA complexes.

Etiology

Procainamide and Hydralazine, β-blocker, Methyldopa, Penicillamine, Isoniazid, Phenytoin, Carbamazepine, Thiazide diuretics, Terbinafine, etc.

Psoriasiform Reaction

Psoriasiform lesions can be induced in patients without any history of psoriasis or an existing psoriasis can be exacerbated.

Etiology

Lithium, β-blocker, antimalarials, NSAIDs (frequently used in psoriatic arthritis), anticonvulsants, arsenic, iodine, Penicillamine, Interferon, gold, sudden withdrawal of systemic corticosteroid or even potent topical corticosteroid, etc.

Exfoliative Dermatitis/Erythroderma

- This is a serious reaction.
- Can start as eczematous eruption or extensive erythema from onset. Even severe contact dermatitis can progress to generalized erythroderma.

Etiology

Common drugs are Sulfonamide, Quinidine, Penicillim, Isoniazide, Phenytoin, Carbamazepine, Thiocetazone, Captopril, etc.

Other Patterns of Drug Reaction

Drug-associated vasculitis.

Leukocytoclastic Vasculitis

Phenylbutazone, Indomethacin, Allopurinol, Thiazide diuretics, Phenytoin, Penicillins, etc.

Henoch-Schönlein Purpura

Acetylsalicylic acid, gold, Penicillins, Quinine, etc.

Pigmented Purpura

Bromhexine, Carbromal, Captopril, etc.

Polyarteritis Nodosa

Acetylsalicylic acid, Sulfasalazine, Allopurinol, Sulfonamides, Potassium, etc.

Ichthyosis

Allopurinol, Clofazimine, lipid-lowering agents, Hydrochlorothiazide, Niacin, etc.

Pityriasis Rosea like Lesions

Gold, Griseofulvin, Metronidazole, Isotretinoin, Omeprazole, Captopril, etc.

Treatment

Identification of the drug is the primary thing. A basic knowledge on common drugs causing reaction and specific reaction pattern is useful because most of the time no records are available.

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Stopping the suspected or better, all the medicines, if possible, as early as possible is the next important step.

Whenever a serious reaction is present or suspected, patients should be urgently hospitalized.

Symptomatic and routine management is of utmost importance like maintenance of body temperature, fluid, electrolyte balance, nutrition, etc.

Medications

- Antihistamines (very rarely they can be a cause of drug reaction)
- Topical corticosteroid in mild cases
- Systemic corticosteroid—Controversial, should be used judiciously in severe cases, can be life-saving but can cause catastrophe if used improperly.

Basic Treatment Guideline for Specific Conditions

Treatment for toxic epidermal necrolysis (TEN)

- Symptomatic.
- Stop all medication. Use medicine as minimum as possible.
- Any friction can lead to skin loss so handling of patients should be with utmost care. Air-fluidized bed is useful.
- Dressing with biological dressing materials.
- Care in a burn unit is very useful to prevent secondary systemic infection like pneumonia, septicemia as these can be fatal.
- Temperature, fluid, electrolyte, should be closely monitored.
- Oral intake is difficult due to oropharyngeal mucosal involvement. So parenteral nutrition is required.
- Eye specialist should always be consulted from very first day. Conjunctival and corneal erosion and scaring should be avoided. Artificial tear is helpful.
- Role of systemic corticosteroid is not proved and it is better avoided.
- Intravenous IgG Immunoglobulin 0.5 gm/kg of body weight for 4–5 days has been found to be life-saving in majority cases.

Treatment for exfoliative dermatitis/ erythroderma

- Symptomatic.
- Temperature, fluid, electrolyte, nutrition should be monitored.
- Infection, venous thrombosis, heart failure are common complication and should be prevented.
- A systemic steroid is needed in severe cases.

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Common Skin Diseases
A Clinical Approach

Salient Features

- Useful for students studying MBBS, BSc Nursing, DVD, MD courses as well as for the practitioners.
- Chapters contain clinical photographs and histopathological microphotographs.
- Provides some of the recent advances as well.
- Highlights of the book are illustrations of procedures, therapy and clinical descriptions.



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