

Lesson Plan (60 Minutes)

Topic: Structure and Functions of the Skin

Target Audience: 5th Year MBBS Students

Department: Dermatology & Venereology

Duration: 60 Minutes

Teaching Method: Interactive Lecture with Clinical Correlation

General Learning Objective

By the end of the session, students will be able to describe the structure, histology, and functions of the skin and correlate normal skin anatomy with common dermatological diseases.

Specific Learning Objectives

At the end of the lecture, students should be able to:

1. Define the skin as the largest organ of the body.
2. Describe the layers of the skin.
3. Explain the histology of the epidermis and dermis.
4. Identify the different cells of the epidermis.
5. Describe skin appendages and their functions.
6. Explain the physiological functions of the skin.
7. Correlate skin structure with common dermatological disorders.

Teaching Aids

- PowerPoint presentation
- Whiteboard
- Skin models
- Histology slides
- Clinical photographs

One-Hour Lesson Plan

Time in minutes	Topic	Teaching Method	Teaching Aids
0–5	Introduction and importance of the skin	Interactive discussion	Title slide
5–15	Structure of the skin (epidermis, dermis, subcutaneous tissue)	Diagram-based lecture	Skin cross-sectional diagram
15–25	Histology of the epidermis and dermis	Histology demonstration	Histology slides
25–35	Skin appendages (hair, nails, sebaceous and sweat glands)	Clinical correlation	Clinical photographs
35–45	Functions of the skin	Interactive lecture	Flowchart
45–55	Clinical relevance of skin structure	Case-based discussion	Clinical images
55–60	Summary and formative assessment	MCQs and oral questions	Summary slide

Key Teaching Points

Layers of the Skin

1. Epidermis

- Stratified squamous keratinized epithelium
- Five layers:
 - Stratum basale
 - Stratum spinosum
 - Stratum granulosum
 - Stratum lucidum (palms and soles)
 - Stratum corneum

Cells of the Epidermis

- Keratinocytes
- Melanocytes
- Langerhans cells
- Merkel cells

2. Dermis

- Papillary layer
- Reticular layer
- Contains:
 - Collagen and elastic fibers
 - Blood vessels
 - Lymphatics
 - Nerves
 - Hair follicles
 - Sweat glands
 - Sebaceous glands

3. Subcutaneous Tissue (Hypodermis)

- Adipose tissue
- Loose connective tissue
- Large blood vessels and nerves
- Provides insulation and energy storage

Skin Appendages

- Hair follicles
- Sebaceous glands
- Eccrine sweat glands
- Apocrine sweat glands
- Nails

Functions of the Skin

- Protection against trauma, microorganisms, and chemicals
- Prevention of water loss
- Thermoregulation (sweating and vasomotor control)
- Sensation (touch, pain, pressure, temperature)
- Vitamin D synthesis
- Immune defense
- Wound healing
- Cosmetic and social function

Clinical Correlation

Structure

Epidermis
Dermo-epidermal junction
Melanocytes
Hair follicles
Sebaceous glands
Sweat glands

Disease

Psoriasis, Pemphigus vulgaris
Bullous pemphigoid
Vitiligo, Melanoma
Acne vulgaris, Folliculitis
Acne
Hyperhidrosis, Miliaria

Teaching Methods

- Interactive lecture
- Diagram-based explanation
- Histology slide demonstration
- Clinical image discussion
- Question-and-answer session

Formative Assessment

Oral Questions

1. Name the five layers of the epidermis.
2. What are the four main cell types of the epidermis?
3. What are the functions of melanocytes?
4. Differentiate eccrine and apocrine sweat glands.
5. What are the major functions of the skin?
6. Which skin layer is involved in bullous pemphigoid?

Take-Home Messages

- The skin is the largest organ of the human body and serves as the first line of defense.
- It consists of three layers: epidermis, dermis, and subcutaneous tissue.
- Keratinocytes, melanocytes, Langerhans cells, and Merkel cells are the principal epidermal cells.
- Skin appendages play essential roles in protection, thermoregulation, and sensation.
- Understanding normal skin structure and function is fundamental to diagnosing and managing dermatological diseases.

Lesson Plan (60 Minutes)

Topic: Cutaneous Signs and Symptoms – Primary and Secondary Skin Lesions

Target Audience: 5th Year MBBS Students

Department: Dermatology & Venereology

Duration: 60 Minutes

Teaching Method: Interactive Lecture with Clinical Image-Based Discussion

General Learning Objective

By the end of the session, students will be able to identify, describe, and classify primary and secondary skin lesions and use correct dermatological terminology in clinical practice.

Specific Learning Objectives

At the end of the lecture, students should be able to:

1. Define cutaneous signs and symptoms.
2. Describe the common symptoms of skin diseases.
3. Define and identify primary skin lesions.
4. Define and identify secondary skin lesions.
5. Describe skin lesions using standard dermatological terminology.
6. Correlate different skin lesions with common dermatological diseases.
7. Develop an approach to the examination of skin lesions.

One-Hour Lesson Plan

Time min	Topic	Learning Objectives	Teaching Method	Teaching Aids
0–5	Introduction	Define skin lesions and explain the importance of morphology in diagnosis	Interactive questioning	Title slide
5–10	Cutaneous Signs & Symptoms	Discuss itching, pain, burning, tenderness, photosensitivity, and sensory changes	Lecture	Symptom chart
10–25	Primary Skin Lesions	Define and identify macule, patch, papule, plaque, nodule, tumor, vesicle, bulla, pustule, wheal, cyst	Clinical image discussion	Clinical photographs
25–38	Secondary Skin Lesions	Explain scale, crust, erosion, ulcer, fissure, excoriation, lichenification, atrophy, scar, sclerosis	Clinical image discussion	Clinical photographs
38–45	Morphological Description of Skin Lesions	Learn how to describe size, shape, color, border, arrangement, distribution, and configuration	Demonstration	Flowchart
45–55	Clinical Correlation	Relate lesions to diseases (psoriasis, eczema, pemphigus, bullous pemphigoid, acne, scabies, fungal infections)	Case-based discussion	Clinical cases
55–60	Summary & Assessment	Review with MCQs and image-based questions	Interactive quiz	Summary slide

Key Teaching Points

Common Cutaneous Symptoms

- Pruritus (itching)
- Pain
- Burning sensation
- Tenderness
- Photosensitivity
- Numbness
- Hyperesthesia
- Hypoesthesia

Primary Skin Lesions

Lesion	Definition	Example
Macule	Flat lesion <1 cm	Freckle
Patch	Flat lesion >1 cm	Vitiligo
Papule	Raised lesion <1 cm	Lichen planus
Plaque	Raised flat-topped lesion >1 cm	Psoriasis
Nodule	Solid lesion extending into dermis	Dermatofibroma
Tumor	Large solid lesion	Skin neoplasm
Vesicle	Small fluid-filled blister <1 cm	Herpes simplex
Bulla	Large blister >1 cm	Bullous pemphigoid
Pustule	Pus-filled lesion	Acne vulgaris
Wheal	Transient edematous lesion	Urticaria
Cyst	Encapsulated cavity with fluid/semisolid material	Epidermoid cyst

Secondary Skin Lesions

Lesion	Definition	Example
Scale	Accumulated flakes of stratum corneum	Psoriasis
Crust	Dried serum, blood, or pus	Impetigo
Erosion	Partial loss of epidermis	Pemphigus vulgaris
Ulcer	Loss of epidermis and dermis	Venous ulcer
Fissure	Linear crack in skin	Heel fissure
Excoriation	Scratch mark	Scabies
Lichenification	Thickened skin with exaggerated skin markings	Chronic eczema
Atrophy	Thinning of skin	Long-term topical steroid use
Scar	Fibrous tissue replacing normal skin	Burn scar
Sclerosis	Hardening of skin	Systemic sclerosis

Describing a Skin Lesion

Students should systematically describe:

- Site
- Number
- Size
- Shape
- Color
- Surface

- Margin
- Border
- Consistency
- Configuration
- Distribution
- Arrangement
- Associated findings

Clinical Correlation

Disease

Psoriasis
 Acne vulgaris
 Pemphigus vulgaris
 Bullous pemphigoid
 Scabies
 Tinea corporis
 Lichen planus
 Vitiligo
 Urticaria

Characteristic Lesion

Plaques with silvery scales
 Comedones, papules, pustules
 Flaccid bullae and erosions
 Tense bullae
 Papules, burrows, excoriations
 Annular scaly plaque
 Violaceous papules
 Depigmented patches
 Wheals

Teaching Methods

- Interactive lecture
- Clinical image interpretation
- Demonstration of lesion description
- Case-based discussion
- Question-and-answer session

Teaching Aids

- PowerPoint presentation
- Whiteboard
- High-quality clinical photographs
- Dermatology atlas
- Skin lesion charts

Formative Assessment

Oral Questions

1. Differentiate a macule from a papule.
2. What is the difference between a vesicle and a bulla?
3. Define plaque and give two examples.
4. What is lichenification?
5. Differentiate erosion from an ulcer.
6. Describe the primary lesions of acne vulgaris.
7. Name three secondary skin lesions.
8. How would you systematically describe a skin lesion during examination?

Take-Home Messages

- Accurate identification of primary and secondary skin lesions is the foundation of dermatological diagnosis.
- Most dermatological diseases can be diagnosed by careful history and detailed morphological examination.
- Always describe skin lesions using standard dermatological terminology (site, size, shape, color, border, surface, configuration, and distribution).
- Recognizing lesion morphology helps formulate an appropriate differential diagnosis and management plan.
- Developing strong observational skills is essential for every MBBS student and future clinician in dermatology.

Lesson Plan (60 Minutes)

Topic: Cutaneous Signs and Symptoms – Primary and Secondary Skin Lesions

Target Audience: 5th Year MBBS Students

Department: Dermatology & Venereology

Duration: 60 Minutes

Teaching Method: Interactive Lecture with Clinical Image-Based Discussion

General Learning Objective

By the end of the session, students will be able to identify, describe, and classify primary and secondary skin lesions and use correct dermatological terminology in clinical practice

Specific Learning Objectives

At the end of the lecture, students should be able to:

1. Define cutaneous signs and symptoms.
2. Describe the common symptoms of skin diseases.
3. Define and identify primary skin lesions.
4. Define and identify secondary skin lesions.
5. Describe skin lesions using standard dermatological terminology.
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One-Hour Lesson Plan

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25–38 min	Secondary Skin Lesions	Explain scale, crust, erosion, ulcer, fissure, excoriation, lichenification, atrophy, scar, sclerosis	Clinical image discussion	Clinical photographs
38–45 min	Morphological Description of Skin Lesions	Learn how to describe size, shape, color, border, arrangement, distribution, and configuration	Demonstration	Flowchart
45–55 min	Clinical Correlation	Relate lesions to diseases (psoriasis, eczema, pemphigus, bullous pemphigoid, acne, scabies, fungal infections)	Case-based discussion	Clinical cases
55–60 min	Summary & Assessment	Review with MCQs and image-based questions	Interactive quiz	Summary slide

Key Teaching Points

Common Cutaneous Symptoms

- Pruritus (itching)
- Pain
- Burning sensation
- Tenderness
- Photosensitivity
- Numbness
- Hyperesthesia
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Primary Skin Lesions

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Describing a Skin Lesion

Students should systematically describe:

- Site
- Number
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- Color
- Surface
- Margin

- Border
- Consistency
- Configuration
- Distribution
- Arrangement
- Associated findings

Clinical Correlation

Disease	Characteristic Lesion
Psoriasis	Plaques with silvery scales
Acne vulgaris	Comedones, papules, pustules
Pemphigus vulgaris	Flaccid bullae and erosions
Bullous pemphigoid	Tense bullae
Scabies	Papules, burrows, excoriations
Tinea corporis	Annular scaly plaque
Lichen planus	Violaceous papules
Vitiligo	Depigmented patches
Urticaria	Wheals

Teaching Methods

- Interactive lecture
- Clinical image interpretation
- Demonstration of lesion description
- Case-based discussion
- Question-and-answer session

Teaching Aids

- PowerPoint presentation
- Whiteboard
- High-quality clinical photographs
- Dermatology atlas
- Skin lesion charts

Formative Assessment

Oral Questions

1. Differentiate a macule from a papule.
2. What is the difference between a vesicle and a bulla?
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4. What is lichenification?
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Take-Home Messages

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- Most dermatological diseases can be diagnosed by careful history and detailed morphological examination.
- Always describe skin lesions using standard dermatological terminology (site, size, shape, color, border, surface, configuration, and distribution).
- Recognizing lesion morphology helps formulate an appropriate differential diagnosis and management plan.
- Developing strong observational skills is essential for every MBBS student and future clinician in dermatology.

Lesson Plan (60 Minutes)

Topic: Cutaneous Drug Reactions (Adverse Cutaneous Drug Reactions)

Target Audience: 5th Year MBBS Students

Department: Dermatology & Venereology

Duration: 60 Minutes

Teaching Method: Interactive Lecture with Case-Based Discussion

General Learning Objective

By the end of the session, students will be able to recognize, diagnose, investigate, classify, and manage common cutaneous drug reactions, including severe cutaneous adverse reactions (SCARs).

Specific Learning Objectives

At the end of the lecture, students should be able to:

1. Define cutaneous drug reaction (CDR) and adverse drug reaction (ADR).
2. Classify common cutaneous drug eruptions.
3. Explain the immunopathogenesis of drug reactions.
4. Recognize the clinical features of common and severe drug reactions.
5. Identify high-risk drugs commonly implicated.
6. Discuss investigations and causality assessment.
7. Outline evidence-based management.
8. Recognize dermatological emergencies requiring urgent referral.

One-Hour Lesson Plan

Time	Topic	Learning Objectives	Teaching Method	Teaching Aids
0–5 min	Introduction	Define ADR and cutaneous drug reaction; discuss clinical importance	Interactive questioning	Title slide, clinical photographs
5–10 min	Classification	Classify cutaneous drug reactions into mild and severe forms	Lecture	Classification chart
10–18 min	Etiology & Pathogenesis	Explain immune mechanisms (Type I–IV hypersensitivity) and risk factors	Diagram-based lecture	Immunology flowchart
18–32 min	Clinical Features	Describe exanthematous eruptions, urticaria, fixed drug eruption (FDE), photosensitivity, DRESS, AGEP, Stevens–Johnson syndrome (SJS), and toxic epidermal necrolysis (TEN)	Clinical image discussion	Clinical photographs
32–40 min	Diagnosis	Discuss drug history, clinical evaluation, laboratory tests, skin biopsy, and causality assessment	Case-based discussion	Diagnostic algorithm
40–50 min	Management	Explain withdrawal of the offending drug, supportive care, topical/systemic therapy, and management of SCARs	Treatment algorithm	Drug charts
50–55 min	Prevention	Drug allergy documentation, pharmacovigilance, patient counseling, and prevention of re-exposure	Interactive discussion	Prevention flowchart
55–60 min	Summary & Assessment	Review key points using MCQs and clinical scenarios	Interactive quiz	Summary slide

Key Teaching Points

Definition

A cutaneous drug reaction is an undesirable skin or mucosal reaction caused by a medication, ranging from mild rashes to life-threatening conditions.

Classification

Mild to Moderate Reactions

- Morbilliform (maculopapular) eruption

- Urticaria
- Angioedema
- Fixed drug eruption (FDE)
- Photosensitivity
- Acneiform eruption
- Pigmentary changes

Severe Cutaneous Adverse Reactions (SCARs)

- Stevens–Johnson syndrome (SJS)
- Toxic epidermal necrolysis (TEN)
- Drug Reaction with Eosinophilia and Systemic Symptoms (DRESS)
- Acute Generalized Exanthematous Pustulosis (AGEP)

Common Offending Drugs

- Antibiotics (especially sulfonamides, penicillins)
- Antiepileptics (carbamazepine, phenytoin, lamotrigine)
- Allopurinol
- NSAIDs
- Antiretroviral drugs

Investigations

- Complete blood count
- Liver and renal function tests
- Skin biopsy (when indicated)
- Serum electrolytes (SCARs)
- Causality assessment (e.g., Naranjo algorithm, WHO-UMC system)

Management

General Principles

- Stop the suspected drug immediately.
- Assess severity.
- Provide symptomatic treatment.
- Document the drug allergy.
- Report serious reactions through pharmacovigilance systems.

Mild Reactions

- Oral antihistamines
- Topical corticosteroids
- Emollients

Severe Reactions (SJS/TEN/DRESS)

- Hospitalization (burn or intensive care unit if indicated)
- Fluid and electrolyte management
- Wound and eye care
- Nutritional support
- Infection prevention
- Systemic therapy according to current institutional/professional guidelines and specialist advice

Prevention

- Careful drug history
- Avoid re-exposure to the culprit drug
- Drug allergy card/medical alert identification
- Patient education
- Pharmacovigilance reporting

Teaching Methods

- Interactive lecture
- Clinical image interpretation
- Case-based discussion
- Question-and-answer session
- Short MCQ quiz

Teaching Aids

- PowerPoint presentation
- Whiteboard
- Clinical photographs
- Drug reaction classification chart
- SCAR management algorithm
- Histopathology images (optional)

Formative Assessment

Oral Questions

1. What is the most common type of cutaneous drug reaction?
 2. Name four drugs commonly associated with Stevens–Johnson syndrome.
 3. Differentiate fixed drug eruption from a morbilliform drug eruption.
 4. What are the cardinal features of DRESS syndrome?
 5. What is the first step in managing a suspected drug reaction?
 6. Why is early recognition of SJS/TEN important?
-

Take-Home Messages

- Cutaneous drug reactions are common and vary from mild self-limiting eruptions to life-threatening dermatological emergencies.
- A detailed drug history is the cornerstone of diagnosis.
- Immediate withdrawal of the suspected drug is the most important initial management step.
- Early recognition of SJS, TEN, DRESS, and AGEP can be life-saving.
- Proper documentation, patient counseling, and avoidance of future exposure are essential to prevent recurrence.

Lesson plan for cutaneous tuberculosis.

Lesson Plan: Eczema, Contact Dermatitis, Seborrheic Dermatitis & Atopic Dermatitis

Class: 5th Year MBBS

Department: Dermatology & Venereology

Duration: 60 minutes

Teaching method: Interactive lecture + clinical case discussion + clinical images

Topic: Eczema and Common Eczematous Dermatoses

Learning Objectives

By the end of the session, students should be able to:

1. Define eczema/dermatitis and describe its basic pathophysiology.
2. Classify the major types of eczema.
3. Describe the clinical features and stages of eczema.
4. Differentiate irritant and allergic contact dermatitis.
5. Identify common occupational and environmental allergens.
6. Describe the clinical features and management of seborrheic dermatitis.
7. Explain the pathogenesis and diagnostic criteria of atopic dermatitis.
8. Identify common complications and triggers of atopic dermatitis.
9. Formulate a basic management plan for these conditions.
10. Differentiate eczema from common mimickers such as psoriasis, tinea and scabies.

1. Time Distribution

Time in minutes	Topic	Teaching activity
0–5	Introduction to eczema	Interactive questioning
5–12	Definition, classification & pathogenesis	Mini lecture
12–22	Clinical features & diagnosis of eczema	Image-based discussion
22–32	Contact dermatitis	Case discussion
32–40	Seborrheic dermatitis	Clinical demonstration
40–52	Atopic dermatitis	Lecture + case discussion
52–57	Differential diagnosis & complications	Interactive discussion
57–60	Summary + MCQs	Assessment

Part A - Eczema

2. Definition

Eczema/dermatitis refers to a group of inflammatory skin disorders characterized by varying combinations of:

- Erythema
- Pruritus
- Papules
- Vesicles
- Oozing
- Crusting
- Scaling
- Lichenification

3. Classification

Introduce students to:

Endogenous eczema

- Atopic dermatitis
- Seborrheic dermatitis
- Nummular eczema
- Dyshidrotic eczema
- Venous/stasis eczema

Exogenous eczema

- Irritant contact dermatitis
- Allergic contact dermatitis

4. Clinical Stages of Eczema

Acute eczema

- Erythema
- Edema
- Papules
- Vesicles
- Weeping/oozing
- Crusting

Subacute eczema

- Erythema
- Scaling
- Crusting
- Less vesiculation

Chronic eczema

- Dryness
- Scaling
- Lichenification
- Fissuring
- Excoriation

Part B - Contact Dermatitis

5. Definition

Contact dermatitis is an inflammatory dermatitis resulting from exposure to an external substance.

Two major types:

A. Irritant Contact Dermatitis — ICD

- Direct damage to the skin
- Does not require prior sensitization
- More common than allergic contact dermatitis

Common irritants:

- Soaps
- Detergents
- Solvents
- Acids/alkalis
- Cement
- Frequent hand washing
- Wet work

B. Allergic Contact Dermatitis — ACD

- Type IV delayed hypersensitivity
- Requires prior sensitization

Common allergens:

- Nickel
- Fragrances
- Preservatives
- Hair dyes—especially PPD
- Rubber chemicals
- Cosmetics
- Topical medications
- Plants

6. Clinical Features of Contact Dermatitis

Typical features:

- Erythema
- Edema
- Papules
- Vesicles
- Oozing
- Crusting
- Scaling
- Pruritus

Distribution often provides an important diagnostic clue.

Examples:

Hands → occupational exposure, detergents, wet work

Eyelids → cosmetics/nail products/fragrances

Neck → perfume/jewelry

Feet → footwear allergens

Face/scalp → cosmetics/hair products

7. Diagnosis

History

About:

- Occupation
- New cosmetics
- Soaps/detergents

- Jewelry
- Hair dye
- Footwear
- Topical medications
- Workplace exposures

Patch testing

Patch test is the standard investigation for suspected allergic contact dermatitis

8. Management

General measures

- Identify and avoid the causative agent
- Protective measures
- Gloves/barrier protection where appropriate
- Regular emollient use

Pharmacological treatment

- Topical corticosteroids
- Emollients
- Treatment of secondary infection when present
- Systemic therapy for severe/widespread disease under specialist supervision

Key message:

The most important treatment of contact dermatitis is identification and avoidance of the offending exposure.

Part C - Seborrheic Dermatitis

9. Definition

A common chronic inflammatory dermatosis affecting sebaceous gland-rich areas.

Common sites:

- Scalp
- Eyebrows

- Glabella
- Nasolabial folds
- Postauricular areas
- Presternal region

10. Pathogenesis

three major factors:

Malassezia yeast + sebum + host inflammatory response

Other contributing factors:

- Genetic susceptibility
- Neuropsychiatric disease
- Immunosuppression

11. Clinical Features

Scalp

- Dandruff
- Erythema
- Greasy/yellowish scales

Face

- Erythematous patches
- Greasy scales
- Nasolabial folds
- Eyebrows
- Glabella
- Postauricular region

Infants

Cradle cap

12. Management

Scalp

- Antifungal shampoos, e.g. ketoconazole
- Other medicated shampoos when appropriate

- Short courses of topical corticosteroids for significant inflammation

Face

- Topical antifungal treatment
- Mild topical corticosteroid for short-term control when needed
- Steroid-sparing topical agents for recurrent facial disease

Important teaching point:

Seborrheic dermatitis is chronic and relapsing, so maintenance therapy may be required.

Part D -Atopic Dermatitis

13. Definition

Atopic dermatitis is a chronic, pruritic, inflammatory skin disease associated with epidermal barrier dysfunction and immune dysregulation.

Atopic tendency may be associated with:

- Asthma
- Allergic rhinitis
- Atopic dermatitis

14. Pathogenesis: simple model:

Genetic/barrier dysfunction

↓

Increased transepidermal water loss

↓

Dry skin + increased penetration of irritants/allergens

↓

Immune dysregulation

↓

Chronic inflammation + pruritus

filaggrin/barrier dysfunction as an important concept.

15. Clinical Features

Infants

- Face
- Scalp
- Extensor surfaces

Older children

- Flexural areas
- Antecubital fossae
- Popliteal fossae
- Neck

Adults

- Flexural eczema
- Hands
- Face/neck
- Lichenification

Cardinal symptom

Pruritus

A useful teaching phrase:

“Itch that rashes” is a characteristic clinical concept in atopic dermatitis.

16. Associated Features

- Xerosis
- Dennie–Morgan folds
- Keratosis pilaris
- Pityriasis alba
- Hyperlinear palms
- Ichthyosis vulgaris
- Recurrent skin infection

17. Complications

- Secondary bacterial infection
- Viral infection, particularly eczema herpeticum
- Sleep disturbance
- Impaired quality of life
- Chronic lichenification

Emergency teaching point

Eczema herpeticum should be suspected when a patient with atopic dermatitis develops:

- Sudden painful vesicles/erosions
- Monomorphic lesions
- Fever or systemic illness

It requires urgent antiviral treatment and medical assessment.

18. Management of Atopic Dermatitis

Step 1 — Skin care

- Regular emollients
- Avoid excessive bathing/harsh soaps
- Identify individual triggers
- Maintain skin barrier

Step 2 — Control inflammation

- Appropriate-potency topical corticosteroids
- Topical calcineurin inhibitors in selected sites/patients
- Other topical anti-inflammatory treatments according to severity

Step 3 — Moderate/severe disease

Specialist-directed treatment may include:

- Phototherapy
- Biologic therapy
- JAK inhibitors
- Other systemic immunomodulatory therapy

Step 4 — Treat complications

- Secondary bacterial infection
- Viral infection
- Sleep disturbance
- Other associated problems

19. Differential Diagnosis

Disease	Helpful distinguishing feature
Psoriasis	Well-demarcated plaques with silvery scale
Tinea corporis	Annular lesion with active peripheral border
Scabies	Severe nocturnal itch + typical sites/contacts
Seborrheic dermatitis	Seborrhoeic distribution with greasy scale
Allergic contact dermatitis	Exposure-related distribution
Atopic dermatitis	Chronic pruritus + typical age/site pattern

20. Case-Based Learning

Case 1 - Contact dermatitis

A 35-year-old woman develops itchy, erythematous, scaly lesions around the ears and neck after wearing new artificial jewelry.

Questions:

1. What is the likely diagnosis?
2. What allergen is commonly responsible?
3. What type of hypersensitivity is involved?
4. What investigation can confirm suspected allergy?

Answers:

- Allergic contact dermatitis
- Nickel is a common allergen
- Type IV delayed hypersensitivity
- Patch testing

Case 2 - Atopic dermatitis

A 7-year-old child has severe itching, dry skin and recurrent eczematous lesions predominantly over the antecubital and popliteal fossae.

Questions:

1. Most likely diagnosis?
2. What is the cardinal symptom?
3. Mention two associated atopic conditions.
4. What is the cornerstone of long-term skin care?

Answers:

- Atopic dermatitis
- Pruritus
- Asthma/allergic rhinitis
- Regular emollient/barrier care

Case 3 - Seborrheic dermatitis

A young adult has recurrent erythema and greasy scales over the scalp, eyebrows and nasolabial folds.

Questions:

1. Diagnosis?
2. Important yeast associated with the condition?
3. Mention two treatment options.

Answers:

- Seborrheic dermatitis
- *Malassezia*
- Antifungal shampoo/topical antifungal; short course of appropriate topical anti-inflammatory therapy when indicated.

21. Assessment - MCQs

1. The most characteristic symptom of atopic dermatitis is:

- A. Pain
- B. Pruritus
- C. Paresthesia
- D. Ulceration

Answer: B

2. Allergic contact dermatitis is primarily:

- A. Type I hypersensitivity
- B. Type II hypersensitivity
- C. Type III hypersensitivity
- D. Type IV hypersensitivity

Answer: D

3. Patch testing is mainly used to diagnose:

- A. Atopic dermatitis
- B. Allergic contact dermatitis
- C. Seborrheic dermatitis
- D. Psoriasis

Answer: B

4. Seborrheic dermatitis commonly affects:

- A. Palms and soles
- B. Sebaceous gland-rich areas
- C. Only the genitalia
- D. Nails only

Answer: B

5. A serious viral complication of atopic dermatitis is:

- A. Eczema herpeticum
- B. Tinea versicolor
- C. Impetigo contagiosa only
- D. Molluscum contagiosum only-----Answer: A

22. Take-Home Messages

- Eczema is a clinical pattern of inflammatory dermatitis, not a single disease.
- Contact dermatitis: always look for an external exposure.
- Allergic contact dermatitis → Type IV hypersensitivity → patch testing.
- Seborrheic dermatitis → sebaceous areas + greasy scale + Malassezia-associated inflammation.
- Atopic dermatitis → itch + xerosis + barrier dysfunction + characteristic distribution.
- Emollients and appropriate anti-inflammatory therapy are central to eczema management.
- Always look for infection and other complications, particularly eczema herpeticum in atopic dermatitis.

Lesson Plan (60 Minutes)

Topic: Superficial Fungal Infections (Superficial Mycoses)

Target Audience: 5th Year MBBS Students

Department: Dermatology & Venereology

Duration: 60 Minutes

Teaching Method: Interactive Lecture with Clinical Case-Based Discussion

General Learning Objective

By the end of the session, students will be able to recognize, diagnose, investigate, differentiate, and manage common superficial fungal infections encountered in clinical practice.

Specific Learning Objectives

At the end of the lecture, students should be able to:

1. Define superficial fungal infections.
2. Classify superficial fungal infections.
3. Identify the common causative fungi.
4. Describe the clinical features of dermatophytosis, candidiasis, and pityriasis versicolor.
5. Perform an appropriate diagnostic workup.
6. Outline evidence-based treatment and prevention.
7. Recognize complications and indications for referral.

One-Hour Lesson Plan

Time in minutes	Topic	Teaching Method	Teaching Aids
0–5	Introduction	Interactive discussion	Title slide
5–10	Classification & Etiology	Lecture	Classification chart
10–25	Clinical Features	Clinical image discussion	Clinical photographs
25–35	Diagnosis & Investigations	Demonstration and case discussion	KOH microscopy images, Wood's lamp images
35–50	Management	Treatment algorithm discussion	Drug chart
50–55	Prevention & Complications	Interactive discussion	Prevention flowchart
55–60	Summary & Assessment	MCQs and oral questions	Summary slide

Key Teaching Points

Definition

Superficial fungal infections are infections confined to the stratum corneum, hair, and nails, caused mainly by dermatophytes, *Candida* species, and *Malassezia* species.

Classification

A. Dermatophytosis (Tinea)

- Tinea corporis
- Tinea cruris
- Tinea pedis
- Tinea manuum
- Tinea faciei
- Tinea capitis
- Tinea barbae
- Tinea unguium (onychomycosis)

B. Cutaneous Candidiasis

- Intertrigo
- Paronychia
- Oral candidiasis
- Vulvovaginal candidiasis

C. Pityriasis (Tinea) Versicolor

- Caused by *Malassezia* species

Etiological Agents

Dermatophytes

- *Trichophyton*
- *Microsporum*
- *Epidermophyton*

Yeasts

- *Candida albicans*
- *Malassezia* species

Clinical Features

Dermatophytosis

- Itching (most common symptom)
- Annular erythematous scaly plaques
- Central clearing
- Raised active border

Candidiasis

- Moist erythematous plaques
- Satellite pustules
- Common in skin folds

Pityriasis Versicolor

- Hypopigmented or hyperpigmented macules
- Fine branny scales
- Mild itching
- Common on the trunk, neck, and upper arms

Investigations

- Clinical diagnosis
- 10–20% KOH microscopy (first-line investigation)
- Fungal culture (selected cases)
- Wood's lamp examination (helpful in some infections)
- Dermoscopy (where available)
- Nail clipping/hair microscopy for onychomycosis or tinea capitis

Differential Diagnosis

- Psoriasis
- Seborrhoeic dermatitis
- Eczema
- Pityriasis rosea
- Vitiligo (for hypopigmented lesions)
- Erythrasma

Management

General Measures

- Keep the affected area clean and dry
- Avoid sharing towels and clothing
- Treat infected family members or close contacts when appropriate
- Avoid misuse of topical steroid combinations

Topical Antifungal Therapy

- Clotrimazole
- Miconazole
- Ketoconazole
- Terbinafine
- Luliconazole
- Sertaconazole

Systemic Antifungal Therapy

Indications

- Extensive dermatophytosis
- Tinea capitis
- Onychomycosis
- Recurrent or resistant disease

Drugs

- Terbinafine
- Itraconazole
- Fluconazole
- Griseofulvin (especially for many cases of tinea capitis)

Complications

- Secondary bacterial infection
- Chronic or recurrent infection
- Permanent scarring alopecia (untreated inflammatory tinea capitis)
- Nail dystrophy
- Majocchi granuloma (rare)

Prevention

- Maintain good personal hygiene
- Keep skin folds dry
- Wear breathable clothing
- Avoid walking barefoot in communal wet areas
- Avoid sharing combs, towels, and footwear
- Prompt treatment of infected individuals and pets (when relevant)

Teaching Methods

- Interactive lecture
- Clinical image interpretation
- Case-based discussion

- Demonstration of KOH microscopy
- Question-and-answer session

Teaching Aids

- PowerPoint presentation
- Whiteboard
- Clinical photographs
- KOH microscopy images
- Wood's lamp photographs
- Treatment algorithm
- Dermatology atlas

Formative Assessment

Oral Questions

1. Define superficial fungal infection.
2. Name the three common genera of dermatophytes.
3. What is the most common symptom of dermatophytosis?
4. What is the characteristic lesion of tinea corporis?
5. What is the first-line investigation for superficial fungal infections?
6. Which fungus causes pityriasis versicolor?
7. When is systemic antifungal therapy indicated?
8. Why should topical steroid–antifungal combination creams be avoided?

Suggested Clinical Cases

- Case 1: A 28-year-old man presents with an itchy annular scaly plaque with central clearing on the forearm. (*Diagnosis: Tinea corporis*)
- Case 2: A 45-year-old woman with diabetes has itchy, moist erythematous plaques with satellite pustules in the groin. (*Diagnosis: Cutaneous candidiasis*)
- Case 3: A 20-year-old student develops multiple hypopigmented, finely scaly macules over the upper back during summer. (*Diagnosis: Pityriasis versicolor*)

Take-Home Messages

- Superficial fungal infections are among the most common dermatological disorders and are usually diagnosed clinically with support from KOH microscopy.
- Dermatophytes infect keratinized tissues (skin, hair, and nails), *Candida* commonly affects moist skin folds, and *Malassezia* causes pityriasis versicolor.
- Appropriate use of topical or systemic antifungal agents, patient education, and good hygiene are essential for successful treatment and prevention of recurrence.
- Avoid inappropriate use of topical corticosteroid-containing combination creams, as they can mask infection and lead to tinea incognito.

Lesson Plan (60 Minutes)

Topic: Bacterial Skin Infections

Target Audience: 5th Year MBBS Students

Department: Dermatology & Venereology

Duration: 60 Minutes

Teaching Method: Interactive Lecture with Case-Based Discussion

General Learning Objective

By the end of the session, students will be able to recognize, diagnose, investigate, and manage common bacterial skin infections encountered in clinical practice.

Specific Learning Objectives

At the end of the lecture, students should be able to:

1. Define bacterial skin infections (pyodermas).
2. Identify the common causative organisms.
3. Classify primary and secondary bacterial skin infections.
4. Describe the clinical features of common bacterial skin diseases.
5. Discuss the investigations used for diagnosis.
6. Outline evidence-based treatment and prevention.
7. Recognize complications and indications for referral.

One-Hour Lesson Plan

Time in minutes	Topic	Learning Objectives	Teaching Method	Teaching Aids
0–5	Introduction	Define bacterial skin infections and discuss their importance	Interactive questioning	Title slide
5–10	Etiology & Classification	Discuss common pathogens and classify primary vs secondary pyodermas	Lecture	Classification chart
10–25	Common Bacterial Skin Infections	Describe impetigo, ecthyma, folliculitis, furuncle, carbuncle, cellulitis, erysipelas, paronychia	Clinical image discussion	Clinical photographs
25–35	Diagnosis & Investigations	Explain clinical diagnosis, Gram stain, pus culture, sensitivity testing, blood tests	Case-based discussion	Diagnostic algorithm

Time in minutes	Topic	Learning Objectives	Teaching Method	Teaching Aids
35–48	Management	Discuss hygiene, topical and systemic antibiotics, incision and drainage, supportive care	Treatment algorithm	Drug charts
48–55	Complications & Prevention	Explain abscess formation, sepsis, post-streptococcal complications, prevention strategies	Interactive discussion	Prevention flowchart
55–60	Summary & Assessment	Review key points through MCQs and clinical cases	Interactive quiz	Summary slide

Key Teaching Points

Definition

Bacterial skin infections are infections of the skin and soft tissues caused mainly by *Staphylococcus aureus* and *Streptococcus pyogenes*.

Classification

Primary Bacterial Skin Infections

- Impetigo
- Ecthyma
- Folliculitis
- Furuncle (boil)
- Carbuncle
- Cellulitis
- Erysipelas

Secondary Bacterial Skin Infections

- Infected eczema
- Infected scabies
- Infected ulcers
- Secondary infection of wounds

Common Organisms

- *Staphylococcus aureus*
- *Streptococcus pyogenes* (Group A Streptococcus)
- *Pseudomonas aeruginosa* (hot-tub folliculitis)
- *Corynebacterium minutissimum* (erythrasma)

Clinical Features

Impetigo

- Honey-colored crusts
- Common in children
- Bullous and non-bullous forms

Ecthyma

- Ulcerative form of impetigo
- Extends into the dermis
- Heals with scarring

Folliculitis

- Pustules centered on hair follicles

Furuncle

- Painful, deep follicular abscess

Carbuncle

- Multiple interconnected furuncles
- Often associated with diabetes mellitus

Cellulitis

- Painful, warm, erythematous swelling with poorly defined margins
- May be associated with fever

Erysipelas

- Bright red, raised plaque with sharply defined borders
- Usually caused by *Streptococcus pyogenes*

Investigations

- Clinical diagnosis
- Gram stain
- Pus culture and antibiotic sensitivity
- CBC and inflammatory markers (selected cases)
- Blood culture in severe infection

Management

General Measures

- Good hygiene
- Wound care
- Drain abscesses when indicated

Topical Antibiotics

- Mupirocin
- Fusidic acid (where appropriate)

Systemic Antibiotics

- Flucloxacillin or cloxacillin
- Cephalexin
- Amoxicillin-clavulanate (selected cases)
- Clindamycin or doxycycline if MRSA is suspected, according to local guidelines

Surgical Treatment

- Incision and drainage for abscesses, furuncles, and carbuncles when indicated

Complications

- Abscess
- Cellulitis
- Lymphangitis
- Osteomyelitis
- Sepsis
- Acute post-streptococcal glomerulonephritis (after streptococcal skin infection)

Prevention

- Personal hygiene
- Early treatment of skin trauma
- Hand washing
- Diabetes control
- Avoid sharing towels and personal items
- Appropriate wound care

Teaching Methods

- Interactive lecture

- Clinical image interpretation
- Case-based discussion
- Question-and-answer session
- Short MCQ quiz

Teaching Aids

- PowerPoint presentation
- Whiteboard
- Clinical photographs
- Gram stain images
- Treatment flowcharts
- Antibiotic guideline summary

Formative Assessment

Oral Questions

1. What are the two most common bacteria causing skin infections?
2. Differentiate impetigo from ecthyma.
3. What is the difference between a furuncle and a carbuncle?
4. How do cellulitis and erysipelas differ clinically?
5. When is incision and drainage indicated?
6. What are the common complications of bacterial skin infections?

Take-Home Messages

- Staphylococcus aureus and Streptococcus pyogenes are the leading causes of bacterial skin infections.
- Impetigo, folliculitis, furuncle, carbuncle, cellulitis, and erysipelas are common conditions that should be recognized clinically.
- Diagnosis is mainly clinical, with microbiological investigations reserved for selected or severe cases.
- Early treatment with appropriate antibiotics and drainage of abscesses prevents complications.
- Good hygiene, wound care, and control of underlying diseases are essential for prevention and reducing recurrence.

Lesson Plan (60 Minutes)

Topic: Common Viral Skin Diseases

Focus: Herpes Zoster, Herpes Simplex, Molluscum Contagiosum, Chickenpox (Varicella), and Viral Warts

Target Audience: 5th Year MBBS Students

Department: Dermatology & Venereology

Duration: 60 Minutes

Teaching Method: Interactive Lecture with Clinical Case Discussion

General Learning Objective

By the end of the session, students will be able to recognize, diagnose, differentiate, and manage common viral skin infections encountered in dermatology.

Specific Learning Objectives

At the end of the lecture, students should be able to:

1. Classify common viral skin infections.
2. Identify the causative viruses.
3. Describe the clinical features of herpes simplex, herpes zoster, chickenpox, molluscum contagiosum, and viral warts.
4. Differentiate these viral skin diseases clinically.
5. Discuss appropriate investigations.
6. Outline evidence-based management and preventive measures.
7. Recognize complications requiring urgent referral.

One-Hour Lesson Plan

Time in Minutes	Topic	Teaching Method	Teaching Aids
0–5	Introduction and classification of viral skin diseases	Interactive lecture	Title slide
5–15	Herpes Simplex Virus (HSV) infection	Clinical image discussion	Clinical photographs
15–25	Herpes Zoster (Shingles)	Case-based discussion	Clinical photographs
25–35	Chickenpox (Varicella)	Interactive lecture	Clinical images
35–42	Molluscum Contagiosum	Clinical image discussion	Clinical photographs
42–50	Viral Warts (Human Papillomavirus)	Clinical demonstration	Images of different wart types
50–55	Diagnosis, investigations, treatment, and prevention	Flowchart discussion	Treatment algorithm
55–60	Summary and formative assessment	MCQs and oral questions	Summary slide

Key Teaching Points

Classification

- Herpesviridae
 - Herpes simplex virus (HSV-1, HSV-2)
 - Varicella-zoster virus (Chickenpox and Herpes Zoster)
- Poxvirus
 - Molluscum contagiosum
- Human Papillomavirus (HPV)
 - Viral warts

1. Herpes Simplex Virus Infection

Clinical Features

- Painful grouped vesicles on an erythematous base
- Recurrent episodes
- Orofacial (HSV-1) and genital (HSV-2) disease
- Burning or tingling before lesions appear

Investigations

- Usually clinical
- PCR (preferred where available)
- Tzanck smear (less specific)
- Viral culture (selected cases)

Treatment

- Oral acyclovir, valacyclovir, or famciclovir
- Early treatment shortens the duration and severity

2. Herpes Zoster

Clinical Features

- Reactivation of varicella-zoster virus
- Painful unilateral dermatomal vesicular eruption
- Thoracic dermatomes commonly involved
- Post-herpetic neuralgia is a common complication

Treatment

- Oral antiviral therapy within 72 hours of rash onset when possible
- Adequate analgesia
- Eye involvement (herpes zoster ophthalmicus) requires urgent ophthalmology referral

3. Chickenpox (Varicella)

Clinical Features

- Fever and malaise
- Generalized itchy vesicular rash
- Lesions at different stages (macules, papules, vesicles, crusts)

Treatment

- Symptomatic care in uncomplicated cases
- Antiviral therapy for selected high-risk patients
- Isolation until all lesions have crusted

4. Molluscum Contagiosum

Clinical Features

- Smooth, dome-shaped, pearly papules
- Central umbilication

- Common in children and immunocompromised individuals

Treatment

- Often self-limiting
- Cryotherapy, curettage, or topical therapies when indicated

5. Viral Warts

Clinical Features

- Caused by Human Papillomavirus (HPV)
- Types:
 - Common wart (verruca vulgaris)
 - Plantar wart
 - Flat wart
 - Filiform wart

Treatment

- Salicylic acid preparations
- Cryotherapy
- Electrocautery or curettage (selected cases)
- Other therapies for resistant lesions

Investigations

- Mainly clinical diagnosis
- PCR for HSV/VZV when diagnosis is uncertain
- Skin biopsy for atypical lesions
- HIV testing in patients with extensive, recurrent, or atypical viral infections when clinically indicated

Complications

- Secondary bacterial infection
- Post-herpetic neuralgia
- Herpes zoster ophthalmicus
- Eczema herpeticum
- Disseminated viral infection in immunocompromised patients

Prevention

- Varicella vaccination
- Herpes zoster vaccination in eligible adults
- Hand hygiene
- Avoid direct contact with active lesions

- Patient education on transmission and recurrence

Teaching Methods

- Interactive lecture
- Clinical image interpretation
- Case-based discussion
- Question-and-answer session
- Short MCQ quiz

Teaching Aids

- PowerPoint presentation
- Whiteboard
- Clinical photographs
- Treatment algorithms
- Dermatology atlas

Formative Assessment

Oral Questions

1. Differentiate herpes simplex from herpes zoster.
2. Which virus causes chickenpox and shingles?
3. What is the characteristic lesion of molluscum contagiosum?
4. Name four types of viral warts.
5. What is the most common complication of herpes zoster?
6. When should antiviral therapy be started in herpes zoster?
7. What is the causative virus of viral warts?

Take-Home Messages

- Viral skin infections are common and are often diagnosed clinically.
- HSV causes recurrent painful grouped vesicles, while herpes zoster presents as a painful unilateral dermatomal vesicular eruption.
- Chickenpox presents with generalized vesicles at different stages of development.
- Molluscum contagiosum is recognized by its umbilicated papules, and viral warts are caused by human papillomavirus (HPV).
- Early recognition, appropriate antiviral therapy when indicated, patient education, and vaccination (where available) help reduce complications and transmission.

Lesson Plan: Psoriasis

Audience: 5th Year MBBS Students

Duration: 60 minutes

Teaching Method: Interactive lecture with clinical images.

General Learning Objective

At the end of the session, students will be able to recognize, diagnose, and outline the basic management of psoriasis and understand its systemic implications.

Time in minutes	Topic	Learning Objectives	Teaching Method	Teaching Aids
0–5	Introduction	Define psoriasis and understand its importance as a chronic inflammatory disease	Interactive questioning	Title slide, clinical image
5–10	Epidemiology & Etiology	Describe prevalence, risk factors, genetics, and triggering factors	Lecture	Flowchart
10–18	Pathogenesis	Explain the immune mechanisms (T-cell mediated disease, IL-23/IL-17 axis)	Diagram-based lecture	Immunopathogenesis diagram
18–30	Clinical Features	Identify morphology, Auspitz sign, Koebner phenomenon, common sites, nail changes, and different clinical types	Clinical image discussion	High-quality photographs
30–38	Diagnosis & Severity Assessment	Discuss diagnosis, differential diagnosis,	Case-based discussion	Chart
38–50	Management	Outline topical therapy, phototherapy, systemic agents, biologics, and lifestyle measures	Algorithm-based lecture	Treatment flowchart
50–55	Complications	Explain psoriatic arthritis, metabolic syndrome, cardiovascular risk, and psychological impact	Discussion	Summary slide
55–60	Recap & Assessment	Reinforce key learning points through MCQs and case scenario	Interactive quiz	MCQ slides

Specific Learning Objectives

By the end of the lecture, students should be able to:

1. Define psoriasis.
2. Describe its epidemiology and risk factors.
3. Explain the immunopathogenesis.
4. Recognize the clinical features and different variants.
5. Identify nail and joint involvement.
6. Differentiate psoriasis from common differential diagnoses.
7. Outline evidence-based management.
8. Recognize systemic comorbidities.
9. Counsel patients regarding long-term follow-up and lifestyle modification.

Teaching Methods

- Interactive lecture
- Case-based discussion
- Clinical image interpretation
- Question-and-answer session
- Short MCQ quiz

Audio-Visual Aids

- PowerPoint presentation
- Clinical photographs
- Whiteboard
- Laser pointer
- Treatment algorithm

Formative Assessment (End of Class)

Asked about:

1. What is the Auspitz sign?
2. What is the Koebner phenomenon?
3. Name three clinical variants of psoriasis.
4. Which cytokines play the major role in psoriasis?
5. What are the indications for systemic therapy?

Take-Home Messages

- Psoriasis is a chronic immune-mediated inflammatory skin disease.
- Diagnosis is mainly clinical.
- Always look for nail changes and psoriatic arthritis.
- Assess disease severity before initiating treatment.
- Treatment should be individualized according to severity and patient factors.
- Psoriasis is associated with important systemic comorbidities, requiring holistic care.

Lesson Plan: HIV and AIDS

Target Audience: 5th Year MBBS Students

Department: Dermatology & Venereology

Duration: 60 Minutes

Teaching Method: Interactive lecture with case discussion

General Learning Objective

By the end of the session, students will be able to understand the epidemiology, pathogenesis, clinical manifestations, diagnosis, treatment, prevention, and dermatological aspects of HIV/AIDS.

Specific Learning Objectives

At the end of the lecture, students should be able to:

1. Define HIV infection and AIDS.
2. Describe the epidemiology of HIV globally and in Bangladesh.
3. Explain the structure, transmission, and life cycle of HIV.
4. Describe the pathogenesis and stages of HIV infection.
5. Recognize the clinical manifestations, including common dermatological diseases.
6. Interpret investigations used in HIV diagnosis and monitoring.
7. Outline the principles of antiretroviral therapy (ART).
8. Discuss opportunistic infections and their prevention.
9. Explain preventive strategies, including PEP and PrEP.
10. Counsel patients regarding prevention, stigma reduction, and follow-up.

Lesson Schedule (60 Minutes)

Time in Minutes	Topic	Learning Objective	Teaching Method	Teaching Aid
0–5	Introduction	Define HIV and AIDS; explain the importance of the disease	Interactive questioning	Title slide
5–10	Epidemiology	Discuss global and Bangladesh HIV burden and risk groups	Lecture	Epidemiology graphs/maps
10–18	HIV Structure & Pathogenesis	Explain HIV structure, life cycle, CD4 cell destruction, and disease progression	Diagram-based lecture	HIV life cycle diagram
18–25	Modes of Transmission	Explain sexual, blood-borne, vertical transmission, and myths about HIV	Interactive discussion	Flowchart
25–35	Clinical Features	Describe Clinical features, AIDS-defining illnesses, and WHO clinical stages	Case discussion	Clinical photographs
35–43	Diagnosis	Explain HIV testing algorithm, ELISA, rapid tests, confirmatory tests, CD4 count, and viral load	Lecture	Diagnostic algorithm
43–50	Management	Outline ART, first-line regimens, adherence, monitoring, and adverse effects	Treatment algorithm	National guideline summary
50–55	Prevention	Discuss condom use, screening, PMTCT, PEP, PrEP, infection control, and counseling	Interactive lecture	Prevention flowchart
55–60	Summary & Assessment	Review key messages and assess understanding with MCQs	Quiz and Q&A	Summary slide

Teaching Methods

- Interactive lecture
- Case-based discussion
- Clinical image interpretation
- Question-and-answer session
- Short MCQ quiz

Audio-Visual Aids

- PowerPoint presentation
- Whiteboard
- Clinical photographs
- HIV life cycle diagram
- WHO staging chart
- ART treatment algorithm

Student Assessment

During the Lecture

- Oral questions
- Case-based discussion
- Image recognition

End of Class (MCQs/Short Questions)

1. What is the difference between HIV infection and AIDS?
2. Name four modes of HIV transmission.
3. What is the role of the CD4 count?
4. Name three common opportunistic infections in AIDS.
5. What are PEP and PrEP?
6. What is the goal of antiretroviral therapy?

Key Dermatological Manifestations to Emphasize

- Oral candidiasis
- Herpes zoster
- Seborrhoeic dermatitis
- Pruritic papular eruption
- Extensive dermatophytosis
- Molluscum contagiosum
- Human papillomavirus (genital warts)
- Kaposi sarcoma
- Bacillary angiomatosis
- Drug eruptions associated with ART

Take-Home Messages

- HIV is a chronic, manageable viral infection when diagnosed early and treated appropriately.
- Early diagnosis and prompt initiation of ART improve survival and reduce transmission.
- Dermatological manifestations are often among the earliest clinical clues to HIV infection.
- Prevention through safe sexual practices, blood safety, PMTCT, PEP, and PrEP is essential.
- Reducing stigma and discrimination is a key component of comprehensive HIV care.
- Lifelong follow-up, adherence to ART, and regular monitoring are critical for successful outcomes.

Lesson Plan (60 Minutes)

Topic: Overview of Sexually Transmitted Diseases (STDs) and Gonococcal Infection

Target Audience: 5th Year MBBS Students

Department: Dermatology & Venereology

Duration: 60 Minutes

Teaching Method: Interactive Lecture with Case-Based Discussion

General Learning Objective

By the end of the session, students will be able to understand the epidemiology, classification, clinical presentation, diagnosis, treatment, and prevention of sexually transmitted diseases (STDs), with special emphasis on gonococcal infection.

Specific Learning Objectives

At the end of the lecture, students should be able to:

1. Define sexually transmitted diseases (STDs).
2. Describe the epidemiology and public health importance of STDs.
3. Classify STDs according to the causative organism.
4. Explain the etiology and pathogenesis of gonococcal infection.
5. Recognize the clinical features of gonorrhoea in males and females.
6. Discuss investigations and diagnosis.
7. Outline current treatment and prevention strategies.
8. Describe complications and partner management.

One-Hour Lesson Plan

Time in minutes	Topic	Learning Objectives	Teaching Method	Teaching Aids
0–5	Introduction	Define STDs and explain their importance	Interactive discussion	Title slide
5–12	Epidemiology & Classification	Classify STDs and discuss disease burden	Lecture	Classification chart
12–18	Overview of Common STDs	Briefly review syphilis, gonorrhoea, chlamydia, chancroid, genital herpes, HPV, HIV, trichomoniasis	Interactive lecture	Flowchart
18–35	Gonococcal Infection	Etiology, transmission, pathogenesis, clinical features in males and females	Clinical case discussion	Clinical photographs & diagrams

Time in minutes	Topic	Learning Objectives	Teaching Method	Teaching Aids
35–45	Diagnosis	Gram stain, NAAT, culture, antimicrobial susceptibility testing	Algorithm-based lecture	Diagnostic flowchart
45–53	Treatment & Prevention	Current treatment, partner management, prevention, antimicrobial resistance	Treatment algorithm	Drug chart
53–60	Summary & Assessment	MCQs, case discussion, oral questions	Interactive quiz	Summary slide

Key Teaching Points

Definition

Sexually transmitted diseases (STDs), also called sexually transmitted infections (STIs), are infections transmitted primarily through sexual contact, including vaginal, anal, and oral intercourse.

Classification of STDs

Bacterial

- Gonorrhoea
- Syphilis
- Chlamydial infection
- Chancroid
- Lymphogranuloma venereum
- Granuloma inguinale

Viral

- Genital herpes
- Human papillomavirus (HPV)
- HIV infection
- Hepatitis B

Protozoal

- Trichomoniasis

Fungal

- Genital candidiasis (not usually classified as an STI but may be sexually associated)

Gonococcal Infection

Etiology

- *Neisseria gonorrhoeae*
- Gram-negative intracellular diplococcus

Transmission

- Sexual contact
- Vertical transmission during childbirth

Clinical Features

In Males

- Urethral discharge
- Dysuria
- Epididymitis
- Proctitis (in some patients)

In Females

- Cervicitis
- Vaginal discharge
- Dysuria
- Pelvic inflammatory disease (PID)
- Many infections are asymptomatic

Extragenital Infection

- Pharyngitis
- Rectal gonorrhoea
- Conjunctivitis

Disseminated Gonococcal Infection (DGI)

- Fever
- Migratory polyarthritits
- Tenosynovitis
- Pustular skin lesions

Investigations

- Gram stain (especially useful in symptomatic men)
- Nucleic Acid Amplification Test (NAAT) – preferred diagnostic test
- Culture with antimicrobial susceptibility testing (important where resistance is suspected)
- Screening for coexisting STIs (HIV, syphilis, chlamydia, hepatitis B)

Treatment (Current Principles)

- Treat according to current national or international STI guidelines, as recommendations change due to antimicrobial resistance.
- Ceftriaxone is the cornerstone of treatment in most current guidelines.
- If chlamydial infection has not been excluded, provide appropriate additional treatment for chlamydia according to guidelines.
- Counsel the patient to abstain from sexual activity until treatment is completed and symptoms have resolved.
- Evaluate and treat all recent sexual partners.

Prevention

- Safe sexual practices and consistent condom use
- Early diagnosis and treatment
- Partner notification and treatment
- Regular screening in high-risk groups
- Health education

Complications

- Pelvic inflammatory disease
- Infertility
- Ectopic pregnancy
- Chronic pelvic pain
- Epididymo-orchitis
- Disseminated gonococcal infection
- Neonatal ophthalmia

Teaching Methods

- Interactive lecture
- Clinical case discussion
- Problem-based learning
- Question-and-answer session
- Short MCQ quiz

Teaching Aids

- PowerPoint presentation
- Whiteboard
- Clinical photographs
- Gram stain images
- Diagnostic and treatment algorithms
- National STI guideline summary

Formative Assessment

Oral Questions

1. What is the causative organism of gonorrhoea?
2. What is the preferred diagnostic test for gonorrhoea?
3. Why is antimicrobial resistance an important concern in gonorrhoea?
4. Name four complications of untreated gonococcal infection.
5. What is disseminated gonococcal infection?
6. Why is partner management essential in STI control?
7. Which STIs should be screened for in a patient diagnosed with gonorrhoea?

Suggested Clinical Cases

- Case 1: A 24-year-old man presents with purulent urethral discharge and dysuria for three days after unprotected sexual intercourse.
- Case 2: A 22-year-old woman has lower abdominal pain, cervical motion tenderness, and mucopurulent cervical discharge, raising suspicion of gonococcal pelvic inflammatory disease.

Take-Home Messages

- STDs remain a major global public health problem, with gonorrhoea being one of the most common bacterial STIs.
- NAAT is the preferred diagnostic test, while culture remains important for monitoring antimicrobial resistance.
- Prompt diagnosis, guideline-based treatment, partner management, and screening for coexisting STIs are essential to prevent complications and reduce transmission.
- Prevention through safe sexual practices, patient education, and early treatment remains the cornerstone of STI control.

Lesson Plan (60 Minutes)

Topic: Acne Vulgaris

Target Audience: 5th Year MBBS Students

Department: Dermatology & Venereology

Duration: 60 Minutes

Teaching Method: Interactive Lecture with Clinical Case Discussion

General Learning Objective

By the end of the session, students will be able to understand the etiopathogenesis, clinical features, diagnosis, grading, management, and prevention of acne vulgaris.

Specific Learning Objectives

At the end of the lecture, students should be able to:

1. Define acne vulgaris.
2. Describe the epidemiology and risk factors.
3. Explain the etiopathogenesis of acne.
4. Classify acne lesions and grade disease severity.
5. Recognize the clinical features and complications.
6. Differentiate acne vulgaris from acneiform eruptions.
7. Outline evidence-based management according to disease severity.
8. Counsel patients regarding treatment adherence and prevention of scarring.

One-Hour Lesson Plan

Time	Topic	Learning Objectives	Teaching Method	Teaching Aids
0–5	Introduction	Define acne vulgaris and discuss its burden in adolescents and young adults	Interactive questioning	Title slide, clinical photographs
5–10	Epidemiology & Risk Factors	Describe prevalence, age distribution, hormonal influence, genetics, diet, and other risk factors	Lecture	Epidemiology charts
10–20	Etiopathogenesis	Explain the four major pathogenic factors: follicular hyperkeratinization, increased sebum production, <i>Cutibacterium acnes</i> proliferation, and inflammation	Diagram-based lecture	Pathogenesis flowchart

Time	Topic	Learning Objectives	Teaching Method	Teaching Aids
20–30	Clinical Features & Classification	Identify lesions (comedones, papules, pustules, nodules, cysts), grading, and common sites	Clinical image discussion	Clinical photographs
30–38	Diagnosis & Differential Diagnosis	Explain clinical diagnosis, indications for investigations, and differentiate acne vulgaris from rosacea, folliculitis, acneiform drug eruptions, and perioral dermatitis	Case-based discussion	Diagnostic algorithm
38–50	Management	Discuss topical therapy, systemic therapy, hormonal treatment, isotretinoin, and scar prevention	Treatment algorithm	Drug charts
50–55	Complications & Patient Counseling	Explain acne scars, post-inflammatory hyperpigmentation, psychosocial impact, adherence, and skin care	Discussion	Counseling checklist
55–60	Summary & Assessment	Review key points through MCQs and case scenarios	Interactive quiz	Summary slide

Key Teaching Points

Definition

Acne vulgaris is a chronic inflammatory disorder of the pilosebaceous unit characterized by comedones, papules, pustules, nodules, and, in severe cases, cysts and scarring.

Epidemiology

- Commonest skin disease in adolescents.
- Peak age:
 - Females: 14–17 years
 - Males: 16–19 years
- May persist into adulthood, especially in women.

Etiopathogenesis (The Four Major Factors)

1. Follicular hyperkeratinization.
2. Increased sebum production (androgen-mediated).
3. Proliferation of *Cutibacterium acnes*.
4. Inflammation.

Clinical Features

Primary Lesions

- Open comedones (blackheads)
- Closed comedones (whiteheads)

Inflammatory Lesions

- Papules
- Pustules
- Nodules
- Cysts

Common Sites

- Face
- Chest
- Upper back
- Shoulders

Grading

- Grade I: Comedonal acne
- Grade II: Papular acne
- Grade III: Papulopustular acne
- Grade IV: Nodulocystic/conglobate acne

Differential Diagnosis

- Rosacea
- Bacterial folliculitis
- Perioral dermatitis
- Acneiform drug eruption
- Malassezia (Pityrosporum) folliculitis

Investigations

- Usually clinical diagnosis.
- Hormonal evaluation in selected patients (e.g., signs of hyperandrogenism).
- Pregnancy test before isotretinoin in women of childbearing potential.

Management

Topical Therapy

- Benzoyl peroxide

- Topical retinoids (adapalene, tretinoin)
- Topical clindamycin (preferably combined with benzoyl peroxide)
- Azelaic acid

Systemic Therapy

- Doxycycline
- Minocycline (where appropriate)
- Oral isotretinoin (severe or refractory acne)

Hormonal Therapy (Selected Females)

- Combined oral contraceptives
- Spironolactone

Complications

- Atrophic scars (ice-pick, boxcar, rolling)
- Hypertrophic scars and keloids
- Post-inflammatory hyperpigmentation
- Psychological distress (anxiety, depression, reduced self-esteem)

Patient Counseling

- Acne is treatable but improvement takes several weeks.
- Adherence to treatment is essential.
- Avoid squeezing or picking lesions.
- Use gentle cleansers and non-comedogenic cosmetics.
- Regular follow-up is important, especially during systemic therapy.

Teaching Methods

- Interactive lecture
- Clinical image interpretation
- Case-based discussion
- Question-and-answer session
- Short MCQ quiz

Teaching Aids

- PowerPoint presentation
- Whiteboard
- Clinical photographs
- Acne grading chart
- Treatment algorithms
- Drug comparison table

Formative Assessment

Oral Questions

1. What are the four major pathogenic factors in acne vulgaris?
2. What is the primary lesion of acne?
3. Differentiate open and closed comedones.
4. What are the indications for oral isotretinoin?
5. Why should topical antibiotics not be used as monotherapy?
6. Name three complications of acne vulgaris.

Take-Home Messages

- Acne vulgaris is a chronic inflammatory disease of the pilosebaceous unit.
- The four key pathogenic mechanisms are follicular hyperkeratinization, excess sebum production, *Cutibacterium acnes* proliferation, and inflammation.
- Management depends on severity, using topical agents for mild disease and systemic therapies for moderate-to-severe disease.
- Oral isotretinoin is the most effective treatment for severe nodulocystic or refractory acne but requires careful monitoring.
- Early treatment and patient education help prevent permanent scarring and reduce psychosocial impact.

Lesson Plan (60 Minutes)

Topic: Urticaria

Target Audience: 5th Year MBBS Students

Department: Dermatology & Venereology

Duration: 60 Minutes

Teaching Method: Interactive Lecture with Case-Based Discussion

General Learning Objective

By the end of the session, students will be able to recognize, diagnose, classify, investigate, and manage urticaria and identify dermatological emergencies such as angioedema and anaphylaxis.

Specific Learning Objectives

At the end of the lecture, students should be able to:

1. Define urticaria and angioedema.
2. Classify acute and chronic urticaria.
3. Explain the etiopathogenesis and common triggers.
4. Recognize the clinical features of different types of urticaria.
5. Discuss the diagnostic approach and appropriate investigations.
6. Outline evidence-based management.
7. Recognize indications for emergency treatment and referral.

One-Hour Lesson Plan

Time in minutes	Topic	Learning Objectives	Teaching Method	Teaching Aids
0–5	Introduction	Define urticaria and explain its clinical importance	Interactive questioning	Title slide, clinical photographs
5–10	Epidemiology & Classification	Differentiate acute and chronic urticaria; spontaneous vs inducible urticaria	Lecture	Classification chart
10–18	Etiology & Pathogenesis	Explain mast cell activation, histamine release, and common triggers	Diagram-based lecture	Pathogenesis flowchart
18–30	Clinical Features	Describe wheals, angioedema, physical urticarias, and warning signs of anaphylaxis	Clinical image discussion	Clinical photographs
30–38	Diagnosis	Explain history, physical examination, investigations, and differential diagnosis	Case-based discussion	Diagnostic algorithm
38–50	Management	Discuss trigger avoidance, second-generation antihistamines, dose escalation, omalizumab, cyclosporine, and emergency management of anaphylaxis	Treatment algorithm	Drug chart
50–55	Patient Education & Prognosis	Counsel on avoidance of triggers, adherence, and follow-up	Interactive discussion	Counseling checklist
55–60	Summary & Assessment	Review key concepts using MCQs and clinical cases	Interactive quiz	Summary slide

Key Teaching Points

Definition

Urticaria (hives) is characterized by transient, intensely itchy wheals, with or without angioedema, caused by mast cell mediator release.

Classification

According to Duration

- Acute urticaria: Less than 6 weeks
- Chronic urticaria: 6 weeks or longer

According to Cause

- Chronic spontaneous urticaria
- Inducible (physical) urticaria:
 - Dermographism
 - Cold urticaria
 - Cholinergic urticaria
 - Pressure urticaria
 - Solar urticaria
 - Aquagenic urticaria (rare)

Clinical Features

- Itchy wheals
- Individual lesions usually disappear within 24 hours
- Angioedema may involve lips, eyelids, tongue, or airway
- Recurrent episodes are common in chronic urticaria

Investigations

- Usually clinical diagnosis
- CBC, ESR/CRP if indicated
- Thyroid function and thyroid autoantibodies (selected chronic cases)
- Physical provocation tests for inducible urticaria
- Allergy testing only when suggested by history

Management

First-Line

- Avoid known triggers
- Second-generation H1 antihistamines (e.g., cetirizine, loratadine, fexofenadine)

If Symptoms Persist

- Increase the dose of second-generation antihistamines (up to fourfold under guideline recommendations)
- Omalizumab for antihistamine-refractory chronic spontaneous urticaria
- Cyclosporine in selected refractory cases

Acute Severe Urticaria

- Short course of systemic corticosteroids may be considered in selected patients

Anaphylaxis

- Immediate intramuscular adrenaline (epinephrine) is the first-line treatment
- Airway, breathing, circulation support
- Urgent referral/hospital management

Teaching Methods

- Interactive lecture
- Clinical image interpretation
- Case-based discussion
- Question-and-answer session
- Short MCQ quiz

Teaching Aids

- PowerPoint presentation
- Whiteboard
- Clinical photographs
- Diagnostic and treatment algorithms
- Urticaria classification chart

Formative Assessment

Oral Questions

1. Define urticaria.
2. What is the difference between acute and chronic urticaria?
3. Name four physical (inducible) types of urticaria.
4. What is the first-line treatment for chronic urticaria?
5. When is omalizumab indicated?
6. What is the first-line emergency treatment for anaphylaxis?

Take-Home Messages

- Urticaria is characterized by transient pruritic wheals, often with angioedema.
- Most cases are diagnosed clinically.
- Second-generation antihistamines are the cornerstone of treatment.
- Refractory chronic urticaria may require omalizumab or other specialist therapies.
- Always recognize anaphylaxis promptly and treat immediately with intramuscular adrenaline, as it is a life-threatening emergency.

Lesson Plan: Chronic Arsenicosis

Topic: Arsenic Exposure and Chronic Arsenicosis

Target Audience: 5th Year MBBS Students

Department: Dermatology & Venereology / Community Medicine

Duration: 60 Minutes

Teaching Method: Interactive Lecture with Clinical Case Discussion

General Learning Objective

At the end of the session, students will be able to understand the sources, pathogenesis, clinical manifestations, diagnosis, management, prevention, and public health importance of chronic arsenicosis, particularly in Bangladesh.

Specific Learning Objectives

By the end of the lecture, students should be able to:

1. Define arsenic and chronic arsenicosis.
2. Describe the sources and routes of arsenic exposure.
3. Explain the epidemiology and public health significance of arsenicosis in Bangladesh and worldwide.
4. Discuss the pathogenesis and toxic effects of chronic arsenic exposure.
5. Recognize the characteristic dermatological and systemic manifestations.
6. Diagnose chronic arsenicosis using clinical criteria and investigations.
7. Outline management strategies and preventive measures.
8. Identify complications, including arsenic-induced malignancies.

One-Hour Lesson Plan

Time in minutes	Topic	Learning Objectives	Teaching Method	Teaching Aids
0–5	Introduction	Define arsenic and chronic arsenicosis; explain its importance in Bangladesh	Interactive questioning	Title slide, clinical photographs
5–10	Epidemiology	Discuss global and Bangladesh prevalence, affected regions, and public health burden	Lecture	Maps, epidemiological charts
10–18	Sources & Pathogenesis	Explain sources of arsenic, routes of exposure, metabolism, and mechanisms of toxicity	Diagram-based lecture	Flowchart of arsenic metabolism
18–35	Clinical Manifestations	Describe dermatological, neurological, gastrointestinal, cardiovascular, hepatic, respiratory, endocrine, and hematological manifestations	Clinical image discussion	Clinical photographs
35–43	Diagnosis	Explain WHO diagnostic criteria, history, examination, arsenic estimation, skin biopsy, and differential diagnosis	Case-based discussion	Diagnostic algorithm
43–52	Management	Discuss exposure cessation, nutritional support, symptomatic treatment, management of skin lesions, surveillance for malignancy	Treatment algorithm	Management flowchart
52–57	Prevention & Public Health	Explain safe drinking water, mitigation strategies, health education, and community screening	Interactive lecture	Prevention flowchart
57–60	Summary & Assessment	Recap key concepts and assess students with MCQs and short questions	Interactive quiz	Summary slide

Key Teaching Points

Definition

- Chronic arsenicosis is a chronic multisystem disorder caused by prolonged ingestion or exposure to arsenic, most commonly through contaminated groundwater.

Sources of Arsenic

- Contaminated tube-well groundwater
- Mining and smelting industries
- Pesticides and herbicides
- Industrial pollution
- Food irrigated with contaminated water

Pathogenesis

- Oxidative stress
- DNA damage
- Enzyme inhibition
- Impaired DNA repair
- Carcinogenesis
- Microvascular injury

Clinical Manifestations

Dermatological Features (Hallmark)

- Diffuse melanosis
- Raindrop pigmentation
- Leucomelanosis
- Palmar and plantar keratosis
- Hyperkeratotic plaques
- Mee's lines (white transverse nail bands)

Systemic Manifestations

- Peripheral neuropathy
- Chronic cough
- Peripheral vascular disease
- Hypertension
- Diabetes mellitus
- Chronic liver disease
- Chronic kidney disease

Malignancies

- Bowen's disease
- Squamous cell carcinoma
- Basal cell carcinoma
- Lung cancer
- Bladder cancer
- Liver cancer

Diagnosis

- History of long-term arsenic exposure
- Characteristic skin lesions
- Arsenic estimation in:
 - Drinking water
 - Urine (recent exposure)
 - Hair and nails (chronic exposure)
- Skin biopsy when indicated

Management

- Eliminate arsenic exposure
- Safe drinking water
- Nutritional improvement (adequate protein and antioxidant-rich diet)
- Keratolytics and emollients for hyperkeratosis
- Treatment of complications
- Regular follow-up and cancer surveillance

Prevention

- Arsenic-free water supply
- Deep tube wells and treated surface water
- Rainwater harvesting where appropriate
- Routine screening in endemic areas
- Community awareness and health education

Teaching Methods

- Interactive lecture
- Clinical image interpretation
- Case-based discussion
- Question-and-answer session
- Short MCQ quiz

Teaching Aids

- PowerPoint presentation
- Whiteboard
- Clinical photographs of arsenicosis
- Bangladesh arsenic distribution map
- WHO/Bangladesh guideline summary
- Diagnostic and management flowcharts

Formative Assessment

Oral Questions

1. Define chronic arsenicosis.
2. What are the major sources of arsenic exposure in Bangladesh?
3. What are the characteristic skin manifestations of chronic arsenicosis?
4. Name three systemic complications of chronic arsenicosis.
5. Which cancers are associated with chronic arsenic exposure?
6. How can chronic arsenicosis be prevented?

Take-Home Messages

- Chronic arsenicosis is a preventable environmental disease and remains an important public health challenge in Bangladesh.
- Diffuse melanosis, raindrop pigmentation, and palmoplantar keratosis are the hallmark dermatological signs.
- Chronic arsenic exposure affects multiple organ systems and increases the risk of cutaneous and internal malignancies.
- Diagnosis depends on exposure history, characteristic clinical findings, and arsenic testing.
- The cornerstone of management is eliminating arsenic exposure, treating complications, improving nutrition, and ensuring long-term surveillance.
- Prevention through safe drinking water and community education is the most effective strategy.

Lesson Plan (60 Minutes)

Topic: Bullous Skin Diseases (Pemphigus Vulgaris and Bullous Pemphigoid)

Target Audience: 5th Year MBBS Students

Department: Dermatology & Venereology

Duration: 60 Minutes

Teaching Method: Interactive Lecture with Clinical Case-Based Discussion

General Learning Objective

By the end of the session, students will be able to recognize, classify, diagnose, investigate, and manage common autoimmune bullous diseases, with emphasis on Pemphigus Vulgaris (PV) and Bullous Pemphigoid (BP).

Specific Learning Objectives

At the end of the lecture, students should be able to:

1. Define bullous skin diseases.
2. Classify autoimmune and non-autoimmune blistering disorders.
3. Explain the pathogenesis of pemphigus vulgaris and bullous pemphigoid.
4. Describe the clinical features of common bullous disorders.
5. Differentiate pemphigus vulgaris from bullous pemphigoid.
6. Interpret the role of skin biopsy, histopathology, and immunofluorescence.
7. Outline evidence-based management.
8. Recognize complications and indications for urgent referral.

One-Hour Lesson Plan

Time in minutes	Topic	Learning Objectives	Teaching Method	Teaching Aids
0–5	Introduction	Define bullous diseases and discuss their importance	Interactive questioning	Title slide
5–10	Classification	Classify autoimmune and inherited bullous disorders	Lecture	Classification chart
10–20	Pemphigus Vulgaris	Explain pathogenesis, clinical features, diagnosis	Clinical image discussion	Clinical photographs
20–30	Bullous Pemphigoid	Explain pathogenesis, clinical features, diagnosis	Clinical image discussion	Clinical photographs
30–40	Diagnosis	Skin biopsy, histopathology, direct and indirect immunofluorescence, ELISA	Case-based discussion	Histopathology & DIF images
40–50	Management	Topical and systemic corticosteroids, immunosuppressants, biologics, supportive care	Treatment algorithm	Drug chart
50–55	Complications & Prognosis	Infection, fluid loss, nutritional deficiency, relapse, mortality	Interactive discussion	Flowchart
55–60	Summary & Assessment	MCQs, image-based questions, case discussion	Interactive quiz	Summary slide

Key Teaching Points

Definition

Bullous skin diseases are disorders characterized by the formation of vesicles and bullae due to separation within the epidermis or at the dermoepidermal junction.

Classification

Autoimmune Bullous Diseases

- Pemphigus vulgaris
- Pemphigus foliaceus
- Bullous pemphigoid
- Mucous membrane pemphigoid
- Linear IgA bullous dermatosis
- Dermatitis herpetiformis

Genetic Bullous Disorders

- Epidermolysis bullosa

Pemphigus Vulgaris (PV)

Autoantigen

- Desmoglein 3 $\pm$ Desmoglein 1

Level of Split

- Intraepidermal (suprabasal)

Clinical Features

- Flaccid bullae
- Painful erosions
- Oral mucosal involvement (often the first manifestation)
- Positive Nikolsky sign
- Positive Asboe-Hansen sign

Histopathology

- Suprabasal acantholysis
- "Row of tombstones" appearance

Direct Immunofluorescence (DIF)

- Intercellular IgG and C3 in a "fish-net/chicken-wire" pattern

Bullous Pemphigoid (BP)

Autoantigen

- BP180 (Type XVII collagen)
- BP230

Level of Split

- Subepidermal

Clinical Features

- Elderly patients
- Tense bullae
- Severe pruritus
- Mucosal involvement is uncommon

- Nikolsky sign usually negative

Histopathology

- Subepidermal blister with eosinophils

Direct Immunofluorescence

- Linear IgG and C3 along the basement membrane zone

Comparison of Pemphigus Vulgaris and Bullous Pemphigoid

Feature	Pemphigus Vulgaris	Bullous Pemphigoid
Age	Middle-aged	Elderly
Blister	Flaccid	Tense
Level of split	Intraepidermal	Subepidermal
Mucosal involvement	Common	Rare
Nikolsky sign	Positive	Usually negative
Autoantigen	Desmoglein 1 & 3	BP180 & BP230
DIF	Intercellular IgG	Linear BMZ IgG

Investigations

- Skin biopsy for histopathology
- Perilesional biopsy for direct immunofluorescence
- Indirect immunofluorescence
- ELISA for desmoglein and BP180/BP230 antibodies
- CBC, blood glucose, liver and renal function tests before systemic therapy

Management

General Measures

- Admit severe cases
- Wound care
- Pain control
- Nutritional support
- Infection prevention
- Oral care for mucosal disease

Medical Treatment

Pemphigus Vulgaris

- Systemic corticosteroids
- Rituximab (first-line steroid-sparing therapy in moderate to severe disease)
- Azathioprine
- Mycophenolate mofetil
- Cyclophosphamide or methotrexate (selected patients)
- IVIG or plasmapheresis in refractory cases

Bullous Pemphigoid

- High-potency topical corticosteroids for localized disease
- Systemic corticosteroids for extensive disease
- Doxycycline (selected mild/moderate cases)
- Azathioprine, methotrexate, or mycophenolate mofetil as steroid-sparing agents
- Biologic therapy (e.g., rituximab, dupilumab) in selected refractory patients

Complications

- Secondary infection
- Sepsis
- Fluid and electrolyte imbalance
- Malnutrition
- Adverse effects of long-term corticosteroids
- Relapse

Teaching Methods

- Interactive lecture
- Clinical photograph interpretation
- Histopathology review
- Case-based learning
- Small-group discussion
- Question-and-answer session

Teaching Aids

- PowerPoint presentation
- Whiteboard
- Clinical photographs
- Histopathology slides
- Direct immunofluorescence images
- Diagnostic and treatment algorithms

Formative Assessment

Oral Questions

1. Define a bulla and differentiate it from a vesicle.
2. What are the autoantigens in pemphigus vulgaris?
3. Why is the Nikolsky sign positive in pemphigus vulgaris?
4. Differentiate pemphigus vulgaris from bullous pemphigoid.
5. What is the gold standard investigation for autoimmune bullous diseases?
6. Describe the direct immunofluorescence findings in PV and BP.
7. What is the current first-line steroid-sparing treatment for moderate-to-severe pemphigus vulgaris?
8. List two complications of long-term corticosteroid therapy.

Suggested Clinical Cases

1. Case 1: A 45-year-old woman with painful oral ulcers followed by flaccid blisters over the trunk and a positive Nikolsky sign. (Diagnosis: Pemphigus vulgaris)
2. Case 2: A 75-year-old man with intense itching and widespread tense bullae but no oral lesions. (Diagnosis: Bullous pemphigoid)

Take-Home Messages

- Autoimmune bullous diseases are potentially life-threatening if not diagnosed and treated promptly.
- Pemphigus vulgaris causes intraepidermal flaccid blisters with frequent mucosal involvement, whereas bullous pemphigoid causes subepidermal tense bullae that usually spare the mucosa.
- Skin biopsy with direct immunofluorescence is the cornerstone of diagnosis.
- Early treatment with corticosteroids and appropriate steroid-sparing agents (especially rituximab for pemphigus vulgaris) significantly improves outcomes.
- Long-term follow-up is essential to monitor disease activity, treatment response, and adverse effects.

Lesson Plan (60 Minutes)

Topic: Cutaneous Tuberculosis

Target Audience: 5th Year MBBS Students

Department: Dermatology & Venereology

Duration: 60 Minutes

Teaching Method: Interactive Lecture with Case-Based Discussion

General Learning Objective

By the end of the session, students will be able to recognize, diagnose, classify, investigate, and manage cutaneous tuberculosis and understand its public health significance.

Specific Learning Objectives

At the end of the lecture, students should be able to:

1. Define cutaneous tuberculosis (CTB).
2. Describe the epidemiology and routes of infection.
3. Classify the different types of cutaneous tuberculosis.
4. Explain the pathogenesis and spectrum of disease.
5. Recognize the clinical features of common forms of CTB.
6. Discuss appropriate investigations and diagnostic methods.
7. Outline treatment according to national and WHO tuberculosis guidelines.
8. Recognize complications and preventive measures.

One-Hour Lesson Plan

Time in minutes	Topic	Learning Objectives	Teaching Method	Teaching Aids
0–5	Introduction	Define cutaneous tuberculosis and discuss its importance	Interactive questioning	Title slide, clinical photographs
5–10	Epidemiology	Discuss global and Bangladesh burden, risk factors, and routes of infection	Lecture	Epidemiology charts
10–18	Etiology & Pathogenesis	Explain <i>Mycobacterium tuberculosis</i> , routes of spread, host immunity, and disease spectrum	Diagram-based lecture	Pathogenesis flowchart
18–32	Classification & Clinical Features	Discuss lupus vulgaris, scrofuloderma, tuberculosis verrucosa cutis (TVC), tuberculous chancre, miliary tuberculosis, and tuberculids	Clinical image discussion	Clinical photographs
32–40	Diagnosis	Explain Mantoux test, skin biopsy, histopathology, Ziehl–Neelsen stain, culture, PCR/CBNAAT, chest X-ray, and other investigations	Case-based discussion	Diagnostic algorithm
40–50	Management	Discuss standard anti-tubercular therapy (ATT), indications for surgery, follow-up, and monitoring	Treatment algorithm	National TB guideline summary
50–55	Complications & Prevention	Discuss scarring, deformity, malignant transformation (rare), BCG vaccination, contact tracing, and public health measures	Discussion	Prevention flowchart
55–60	Summary & Assessment	Review key concepts using MCQs and clinical cases	Interactive quiz	Summary slide

Key Teaching Points

Definition

Cutaneous tuberculosis is a chronic skin infection caused by *Mycobacterium tuberculosis*, occurring through exogenous inoculation, contiguous spread, hematogenous dissemination, or lymphatic spread.

Classification

Multibacillary Forms

- Scrofuloderma
- Tuberculous chancre
- Acute miliary tuberculosis

Paucibacillary Forms

- Lupus vulgaris
- Tuberculosis verrucosa cutis (TVC)
- Tuberculids (e.g., lichen scrofulosorum, papulonecrotic tuberculid)

Clinical Features

Lupus Vulgaris

- Most common form in many regions
- Slowly enlarging reddish-brown plaques
- "Apple-jelly" nodules on diascopy
- Common on the face and neck

Scrofuloderma

- Extension from underlying infected lymph nodes, bone, or joints
- Nodules that soften to form ulcers and sinus tracts
- Heals with characteristic scarring

Tuberculosis Verrucosa Cutis (TVC)

- Warty plaque
- Usually on hands or feet
- Occurs after exogenous inoculation in previously sensitized individuals

Tuberculids

- Hypersensitivity reactions associated with an underlying focus of tuberculosis
- Symmetric papular lesions with few or no detectable bacilli

Diagnosis

- Detailed history and physical examination
- Mantoux (tuberculin skin) test
- Skin biopsy and histopathology (granulomatous inflammation)
- Ziehl–Neelsen staining for acid-fast bacilli
- Mycobacterial culture
- PCR/CBNAAT where available

- Chest X-ray and evaluation for systemic tuberculosis

Treatment

- Standard anti-tubercular therapy (ATT) according to national/WHO guidelines:
 - Intensive phase: Isoniazid, Rifampicin, Pyrazinamide, Ethambutol
 - Continuation phase: Isoniazid and Rifampicin
- Surgical intervention only for selected cases (e.g., drainage, reconstruction after healing)
- Regular follow-up to assess response and monitor drug toxicity

Complications

- Permanent scarring
- Contractures
- Cosmetic deformity
- Secondary bacterial infection
- Rarely, squamous cell carcinoma developing in long-standing lupus vulgaris scars

Teaching Methods

- Interactive lecture
- Clinical image interpretation
- Case-based discussion
- Question-and-answer session
- Short MCQ quiz

Teaching Aids

- PowerPoint presentation
- Whiteboard
- Clinical photographs
- Histopathology slides
- Diagnostic algorithms
- National TB treatment guidelines

Formative Assessment

Oral Questions

1. What is cutaneous tuberculosis?

2. Name the common clinical types of cutaneous tuberculosis.
3. What is the characteristic finding on diascopy in lupus vulgaris?
4. How does scrofuloderma develop?
5. Which investigations help confirm the diagnosis of cutaneous tuberculosis?
6. What is the standard treatment for cutaneous tuberculosis?

Take-Home Messages

- Cutaneous tuberculosis is an uncommon but important extrapulmonary manifestation of tuberculosis.
- Lupus vulgaris, scrofuloderma, and tuberculosis verrucosa cutis are the most commonly encountered forms.
- Diagnosis relies on clinical suspicion, supported by histopathology, microbiological tests, and molecular methods where available.
- Early initiation of standard anti-tubercular therapy results in excellent outcomes and helps prevent complications.
- Patients should always be evaluated for systemic tuberculosis, and management should follow national tuberculosis control guidelines.

Lesson Plan — Leprosy (Hansen Disease)

Target: 5th Year MBBS Students

Department: Dermatology & Venereology

Duration: 60 minutes

Teaching method: Interactive lecture + clinical image interpretation + case-based discussion

General Learning Objective

By the end of the session, students will be able to recognize, classify, diagnose, investigate, treat, and prevent leprosy, with particular emphasis on early detection of peripheral nerve involvement and prevention of disability.

Specific Learning Objectives

Students should be able to:

1. Define leprosy and identify its causative organism.
2. Describe transmission and risk factors.
3. Explain the spectrum and classification of leprosy.
4. Recognize the characteristic skin and nerve manifestations.
5. Demonstrate the basic examination of peripheral nerves and sensory function.
6. State the diagnostic criteria and appropriate investigations.
7. Describe WHO classification and multidrug therapy (MDT).
8. Recognize Type 1 and Type 2 lepra reactions and their management principles.
9. Identify complications and methods of disability prevention.

60-Minute Teaching Plan

Time in minutes	Topic	Learning Objectives	Method	Teaching Aids
0–5	Introduction & clinical importance	Define leprosy; understand its public-health significance	Interactive questioning	Opening slide
5–10	Etiology, transmission & epidemiology	Identify <i>M. leprae</i> and modes of transmission	Short lecture	Diagram
10–20	Clinical spectrum & classification	Understand tuberculoid → lepromatous spectrum and WHO PB/MB classification	Lecture + images	Spectrum chart
20–32	Clinical features	Identify skin lesions, sensory loss, nerve thickening and deformities	Clinical image demonstration	Photographs
32–40	Diagnosis & examination	Apply cardinal signs; examine peripheral nerves and sensation	Demonstration	Nerve examination chart
40–48	Treatment	Understand MDT for PB and MB disease	Treatment algorithm	WHO MDT chart
48–54	Lepra reactions	Recognize Type 1 and Type 2 reactions and their importance	Case discussion	Clinical cases
54–57	Complications & prevention of disability	Identify ulcers, deformities, eye complications and self-care measures	Interactive discussion	Images
57–60	Summary & assessment	Consolidate key points	MCQs/oral questions	Summary slide

Key Teaching Content

1. Definition & Etiology

Leprosy (Hansen disease) is a chronic infectious disease caused mainly by *Mycobacterium leprae*, primarily affecting:

- Skin
- Peripheral nerves
- Upper respiratory mucosa
- Eyes
- Other tissues in advanced disease

2. Cardinal Clinical Signs

Emphasize the three cardinal signs:

1. Hypopigmented or erythematous skin lesion with definite loss of sensation
2. Peripheral nerve involvement with sensory loss, weakness or paralysis
3. Demonstration of acid-fast bacilli in slit-skin smear

MBBS examination pearl: A patient can be diagnosed clinically when characteristic skin lesions with sensory loss and/or typical nerve involvement are present; bacteriological confirmation is not mandatory in every case.

3. Classification

Ridley–Jopling Spectrum

- Tuberculoid (TT)
- Borderline tuberculoid (BT)
- Mid-borderline (BB)
- Borderline lepromatous (BL)
- Lepromatous (LL)

WHO Operational Classification

Paucibacillary (PB)

- Usually 1–5 skin lesions
- No demonstrated bacilli on smear

Multibacillary (MB)

- More than 5 skin lesions, and/or
- Positive slit-skin smear

4. Clinical Features

Tuberculoid Leprosy

- Few, well-defined hypopigmented or erythematous plaques
- Marked sensory loss
- Hair and sweating may be lost
- Asymmetrical nerve involvement
- Thickened peripheral nerves

Lepromatous Leprosy

- Numerous, symmetrical lesions
- Macules, papules, plaques and nodules

- Diffuse infiltration of skin
- Loss of eyebrows/eyelashes
- Multiple nerve involvement
- Sensory loss may initially be less obvious
- Advanced disease may produce leonine facies

Nerve Involvement

Important nerves to demonstrate:

- Ulnar
- Common peroneal
- Posterior tibial
- Greater auricular
- Facial nerve

Assess:

- Thickening/tenderness
- Sensory function
- Motor function
- Autonomic function

5. Important Investigations

- Slit-skin smear for acid-fast bacilli
- Skin biopsy when diagnosis is uncertain
- Histopathology
- Fite-Faraco staining where available
- Nerve function assessment
- Baseline investigations before systemic therapy when indicated

6. Treatment

Multidrug Therapy (MDT)

For adults, the WHO-recommended regimen uses:

Rifampicin + Dapsone + Clofazimine

- PB: rifampicin + dapsone + clofazimine for 6 months under current WHO guidance.
- MB: the same three-drug regimen for 12 months.

Treatment should follow the current national/WHO leprosy programme protocol, particularly for children, pregnancy, drug intolerance, or special clinical circumstances.

Important Teaching Point

Leprosy reactions are not treatment failure. MDT should generally be continued during reactions.

7. Leprosy Reactions

Type 1 Reaction — Reversal Reaction

Usually occurs in borderline disease.

Features:

- Existing lesions become red, swollen and tender
- New lesions may appear
- Neuritis
- New sensory or motor impairment

Management principle:

- Continue MDT
- Systemic corticosteroids when significant neuritis or nerve function impairment is present
- Frequent nerve-function monitoring

Type 2 Reaction — Erythema Nodosum Leprosum (ENL)

More common in BL/LL disease.

Features:

- Fever
- Tender erythematous nodules
- Malaise
- Neuritis
- Arthritis
- Orchitis
- Iritis/uveitis
- Other systemic involvement

Management: specialist-directed anti-inflammatory/immunomodulatory treatment, commonly including corticosteroids; clofazimine or other agents may be used depending on severity and recurrence.

8. Complications

- Peripheral nerve damage
- Claw hand
- Foot drop
- Lagophthalmos
- Plantar ulcers
- Contractures
- Resorption of digits
- Blindness
- Secondary bacterial infection
- Social stigma and psychological problems

9. Prevention of Disability

Teach patients:

- Daily inspection of anesthetic hands and feet
- Protective footwear
- Avoid burns and trauma
- Regular moisturizing of dry skin
- Proper wound care
- Eye protection in lagophthalmos
- Physiotherapy and occupational therapy
- Early reporting of new nerve symptoms

Case-Based Discussion

Case 1

A 25-year-old man has three hypopigmented plaques on the forearm. The lesions are anesthetic and the ulnar nerve is thickened.

Questions:

- What is your diagnosis?
- Which cardinal signs are present?
- How would you classify the disease?
- What treatment would you prescribe?

Case 2

A 55-year-old man with multiple skin lesions develops fever, painful red nodules and tender nerves.

Questions:

- What type of lepra reaction is likely?

- What systemic complications should be looked for?
- Should MDT be stopped?

Formative Assessment

1. What is the causative organism of leprosy?
2. What are the three cardinal signs?
3. Name the five Ridley–Jopling groups.
4. What distinguishes PB from MB leprosy?
5. Name four peripheral nerves commonly affected.
6. Differentiate Type 1 reaction from ENL.
7. What is the current MDT regimen for adult MB disease?
8. Why is early detection of nerve involvement important?

Take-Home Messages

- Think of leprosy whenever a skin lesion has definite sensory loss.
- Always examine the peripheral nerves, not just the skin.
- Leprosy is curable with MDT, but nerve damage can become permanent if treatment is delayed.
- Recognize Type 1 reaction and ENL early, particularly when there is new nerve dysfunction.
- Disability prevention through early diagnosis, nerve-function monitoring and self-care is as important as killing the bacilli.

Lesson Plan (60 Minutes)

Topic: Pigmentary Disorders of the Skin – Vitiligo and Melasma

Target Audience: 5th Year MBBS Students

Department: Dermatology & Venereology

Duration: 60 Minutes

Teaching Method: Interactive Lecture with Clinical Case Discussion

General Learning Objective

By the end of the session, students will be able to recognize, diagnose, differentiate, investigate, and manage the two most common pigmentary disorders—vitiligo and melasma.

Specific Learning Objectives

At the end of the lecture, students should be able to:

1. Define pigmentary disorders.
2. Classify disorders of hyperpigmentation and hypopigmentation.
3. Explain the etiopathogenesis of vitiligo and melasma.
4. Recognize the clinical features and classify vitiligo.
5. Differentiate vitiligo from other hypopigmented disorders.
6. Discuss the diagnosis and assessment of melasma.
7. Outline evidence-based treatment of vitiligo and melasma.
8. Counsel patients regarding prognosis and prevention.

One-Hour Lesson Plan

Time	Topic	Learning Objectives	Teaching Method	Teaching Aids
0–5 min	Introduction	Define pigmentary disorders and discuss their clinical importance	Interactive questioning	Title slide
5–10 min	Classification	Classify hyperpigmentation and hypopigmentation disorders	Lecture	Classification chart
10–22 min	Vitiligo	Etiology, pathogenesis, clinical features, classification, diagnosis	Clinical image discussion	Clinical photographs, Wood's lamp images
22–32 min	Management of Vitiligo	Medical therapy, phototherapy, surgical treatment, counseling	Treatment algorithm	Drug chart
32–45 min	Melasma	Epidemiology, etiopathogenesis, clinical types, diagnosis, MASI score	Case-based discussion	Clinical photographs
45–55 min	Management of Melasma	Sun protection, topical agents, chemical peels, lasers, oral tranexamic acid	Treatment algorithm	Drug chart
55–60 min	Summary & Assessment	Review with MCQs and case scenarios	Interactive quiz	Summary slide

Key Teaching Points

Pigmentary Disorders

Hypopigmentation

- Vitiligo
- Pityriasis alba
- Post-inflammatory hypopigmentation
- Albinism

Hyperpigmentation

- Melasma
- Post-inflammatory hyperpigmentation
- Drug-induced pigmentation
- Addison disease

Vitiligo

Definition

An acquired autoimmune depigmenting disorder characterized by the selective destruction of melanocytes.

Etiopathogenesis

- Autoimmune mechanisms
- Genetic susceptibility
- Oxidative stress
- Neurogenic factors

Clinical Features

- Well-defined depigmented macules and patches
- Symmetrical distribution
- Leukotrichia
- Koebner phenomenon
- Usually asymptomatic

Classification

- Non-segmental vitiligo
- Segmental vitiligo
- Mixed vitiligo
- Universal vitiligo

Diagnosis

- Clinical examination
- Wood's lamp examination
- Dermoscopy
- Thyroid function tests (selected patients)
- Autoimmune screening when indicated

Treatment

- Patient counseling
- Broad-spectrum sunscreen
- Topical corticosteroids
- Topical tacrolimus
- Narrowband UVB phototherapy
- Excimer laser (localized disease)
- Surgical grafting for stable vitiligo
- Depigmentation therapy in extensive disease (selected cases)

Melasma

Definition

An acquired hypermelanosis presenting as symmetrical brown facial macules and patches.

Risk Factors

- Sun exposure
- Pregnancy
- Oral contraceptives
- Hormonal therapy
- Genetic predisposition

Clinical Types

- Centrofacial
- Malar
- Mandibular

Wood's Lamp Classification

- Epidermal
- Dermal
- Mixed

Assessment

- MASI (Melasma Area and Severity Index)

Treatment

- Sun avoidance
- Broad-spectrum sunscreen
- Triple combination cream (hydroquinone + tretinoin + corticosteroid)
- Azelaic acid
- Kojic acid
- Topical retinoids
- Oral tranexamic acid (selected patients)
- Chemical peels
- Laser and light-based therapies (selected cases)

Differential Diagnosis

Vitiligo

- Pityriasis versicolor
- Pityriasis alba
- Post-inflammatory hypopigmentation
- Chemical leukoderma

Melasma

- Post-inflammatory hyperpigmentation
- Drug-induced pigmentation
- Lichen planus pigmentosus
- Exogenous ochronosis

Teaching Methods

- Interactive lecture
- Clinical image interpretation
- Case-based discussion
- Question-and-answer session
- Short MCQ quiz

Teaching Aids

- PowerPoint presentation
- Whiteboard
- Clinical photographs
- Wood's lamp images
- Dermoscopy images
- MASI scoring chart
- Vitiligo assessment tools (e.g., VASI)

Formative Assessment

Oral Questions

1. What is the most common cause of acquired depigmentation?
2. What is the role of the Wood's lamp in vitiligo?
3. Name the clinical types of vitiligo.
4. What are the major risk factors for melasma?
5. What is the MASI score used for?
6. Name two topical treatments for vitiligo.
7. What is the first step in the management of melasma?
8. Which phototherapy is preferred for generalized vitiligo?

Take-Home Messages

- Vitiligo is an autoimmune depigmenting disorder caused by melanocyte loss, while melasma is an acquired hyperpigmentation disorder strongly associated with ultraviolet exposure and hormonal influences.
- Diagnosis of both conditions is primarily clinical, supported by tools such as Wood's lamp, dermoscopy, VASI (for vitiligo), and MASI (for melasma).
- Narrowband UVB phototherapy is the treatment of choice for generalized vitiligo, whereas strict photoprotection and topical depigmenting agents are the cornerstones of melasma management.
- Patient education, realistic expectations, and long-term follow-up are essential because both disorders often have a chronic or relapsing course.

Lesson Plan: Syphilis

Target Audience: 5th Year MBBS Students

Department: Dermatology & Venereology

Duration: 60 Minutes

Teaching Method: Interactive Lecture with Clinical Case Discussion

General Learning Objective

By the end of the session, students will be able to recognize, diagnose, investigate, treat, and prevent syphilis according to current clinical guidelines.

Specific Learning Objectives

At the end of the lecture, students should be able to:

1. Define syphilis and identify its causative organism.
 2. Describe the epidemiology and modes of transmission.
 3. Explain the natural history and stages of syphilis.
 4. Recognize the clinical manifestations of primary, secondary, latent, and tertiary syphilis.
 5. Discuss congenital syphilis and its prevention.
 6. Interpret serological tests used for diagnosis.
 7. Outline current treatment regimens and follow-up.
 8. Explain partner notification and prevention strategies.
-

One-Hour Lesson Plan

Time in minutes	Topic	Learning Objective	Teaching Method	Teaching Aid
0–5	Introduction	Define syphilis; identify <i>Treponema pallidum</i> and discuss disease importance	Interactive questioning	Title slide, clinical image
5–10	Epidemiology	Discuss global and Bangladesh burden, risk groups, and public health significance	Lecture	Epidemiology charts
10–15	Etiology & Transmission	Explain microbiology and modes of transmission (sexual, vertical, blood exposure)	Diagram-based lecture	Transmission flowchart
15–30	Clinical Stages	Describe primary, secondary, latent, tertiary, and congenital syphilis with characteristic findings	Case-based discussion	Clinical photographs
30–40	Diagnosis	Explain dark-field microscopy (where available), NAAT (if available), non-treponemal (VDRL/RPR), and treponemal (TPHA/TPPA, FTA-ABS) tests	Interactive lecture	Diagnostic algorithm
40–50	Treatment	Discuss benzathine penicillin regimens, neurosyphilis treatment, penicillin allergy, pregnancy, and follow-up	Treatment algorithm	Guideline summary
50–55	Prevention & Complications	Explain partner notification, congenital syphilis prevention, HIV testing, and complications	Discussion	Flowchart
55–60	Summary & Assessment	Review key messages using MCQs and a short clinical scenario	Interactive quiz	Summary slide

Key Teaching Points

Definition

- Syphilis is a **chronic systemic sexually transmitted infection** caused by *Treponema pallidum* subsp. *pallidum*.

Clinical Stages

- Primary syphilis: Painless chancre, regional lymphadenopathy.
- Secondary syphilis: Generalized rash (including palms and soles), mucous patches, condyloma lata, generalized lymphadenopathy.

- Latent syphilis: Asymptomatic; early and late latent stages.
- Tertiary syphilis: Gummatous disease, cardiovascular syphilis, neurosyphilis.
- Congenital syphilis: Early and late manifestations due to vertical transmission.

Diagnosis

- Direct detection (dark-field microscopy or NAAT where available).
- Non-treponemal tests: VDRL, RPR (screening and monitoring).
- Treponemal tests: TPHA/TPPA, FTA-ABS (confirmation).

Treatment (Core Principles)

- **Benzathine penicillin G** is the treatment of choice for most stages.
- Neurosyphilis requires aqueous crystalline penicillin G.
- Pregnant women with syphilis should receive penicillin, with desensitization if allergic.
- Follow serological response with quantitative non-treponemal titers.

Teaching Methods

- Interactive lecture
- Clinical image interpretation
- Case-based discussion
- Question-and-answer session
- Short MCQ quiz

Teaching Aids

- PowerPoint presentation
- Whiteboard
- Clinical photographs
- Diagnostic algorithms
- Treatment flowcharts
- WHO/National STI guideline summary

Formative Assessment

Oral Questions

1. What is the causative organism of syphilis?
2. Describe the features of a primary chancre.
3. What are the manifestations of secondary syphilis?
4. Differentiate non-treponemal and treponemal tests.
5. What is the treatment of choice for early syphilis?
6. How can congenital syphilis be prevented?

Take-Home Messages

- Syphilis is a multistage systemic infection with diverse clinical manifestations.
- Early diagnosis and treatment prevent serious long-term complications and transmission.
- Serological testing is the cornerstone of diagnosis and follow-up.
- Penicillin remains the gold-standard treatment for syphilis.
- Always screen for HIV and other STIs, notify and treat sexual partners, and ensure follow-up.
- Universal antenatal screening and timely treatment are essential to prevent congenital syphilis.

Lesson Plan (60 Minutes)

Topic: Cutaneous Manifestations of Systemic Diseases

Target Audience: 5th Year MBBS Students

Department: Dermatology & Venereology

Duration: 60 Minutes

Teaching Method: Interactive Lecture with Case-Based Discussion

General Learning Objective

By the end of the session, students will be able to recognize common skin manifestations of systemic diseases, correlate them with underlying pathology, and formulate an appropriate diagnostic and management approach.

Specific Learning Objectives

At the end of the lecture, students should be able to:

1. Define cutaneous manifestations of systemic diseases.
2. Explain why the skin is considered a "window to systemic disease."
3. Identify common skin signs associated with endocrine, renal, hepatic, hematological, rheumatological, gastrointestinal, infectious, and malignant diseases.
4. Correlate skin findings with underlying systemic disorders.
5. Outline the diagnostic approach and appropriate investigations.
6. Discuss principles of management and referral.

One-Hour Lesson Plan

Time in minutes	Topic	Learning Objectives	Teaching Method	Teaching Aids
0–5	Introduction	Importance of skin as a marker of systemic disease	Interactive discussion	Title slide
5–10	Classification	Categorize cutaneous manifestations by organ system	Lecture	Classification chart
10–20	Endocrine Disorders	Recognize diabetic dermopathy, acanthosis nigricans, necrobiosis lipoidica, pretibial myxedema	Clinical photographs	Clinical images
20–30	Connective Tissue & Autoimmune Disorders	Identify malar rash, discoid lupus, heliotrope rash, Gottron papules, scleroderma	Case discussion	Clinical photographs
30–38	Hepatic, Renal & Hematological Disorders	Recognize jaundice, spider angioma, palmar erythema, calciphylaxis, pallor, purpura	Interactive lecture	Flowchart
38–46	Infectious Diseases & Malignancy	HIV-related skin lesions, Kaposi sarcoma, paraneoplastic dermatoses	Clinical image discussion	Clinical photographs
46–54	Diagnostic Approach	History, examination, investigations, skin biopsy, laboratory tests	Algorithm-based lecture	Diagnostic algorithm
54–60	Summary & Assessment	MCQs, image-based questions, discussion	Interactive quiz	Summary slide

Key Teaching Points

Endocrine Diseases

- Diabetes mellitus:
 - Diabetic dermopathy
 - Acanthosis nigricans
 - Necrobiosis lipoidica
 - Diabetic bullae
- Thyroid disease:
 - Pretibial myxedema
 - Dry skin
 - Alopecia
 - Vitiligo

Connective Tissue Diseases

- Systemic lupus erythematosus:
 - Malar rash
 - Photosensitivity
 - Oral ulcers
- Dermatomyositis:
 - Heliotrope rash
 - Gottron papules
 - Shawl sign
- Systemic sclerosis:
 - Sclerodactyly
 - Skin tightening
 - Telangiectasia
 - Salt-and-pepper pigmentation

Liver Diseases

- Jaundice
- Spider angiomas
- Palmar erythema
- Pruritus
- Terry's nails

Renal Diseases

- Uremic pruritus
- Xerosis
- Calciphylaxis
- Half-and-half nails

Hematological Disorders

- Pallor
- Petechiae
- Purpura
- Ecchymosis
- Koilonychia

Gastrointestinal Disorders

- Dermatitis herpetiformis (celiac disease)
- Erythema nodosum (inflammatory bowel disease)
- Pyoderma gangrenosum (ulcerative colitis/Crohn's disease)

Infectious Diseases

- HIV/AIDS:
 - Oral candidiasis
 - Herpes zoster
 - Seborrheic dermatitis
 - Molluscum contagiosum
 - Kaposi sarcoma

Internal Malignancies

- Acanthosis nigricans (malignant type)
- Leser–Trélat sign
- Dermatomyositis
- Paraneoplastic pemphigus

Diagnostic Approach

- Detailed history
- Complete skin examination
- General physical examination
- Laboratory tests (CBC, blood glucose, thyroid profile, liver and renal function tests, autoimmune markers)
- Skin biopsy when indicated
- Imaging or specialist referral based on suspected systemic disease

Teaching Methods

- Interactive lecture
- Clinical image interpretation
- Case-based discussion
- Small-group problem solving
- Question-and-answer session

Teaching Aids

- PowerPoint presentation
- Whiteboard
- Clinical photographs
- Histopathology images
- Diagnostic algorithms

Formative Assessment

Oral Questions

1. Why is the skin called the "window to systemic disease"?
2. Name four cutaneous manifestations of diabetes mellitus.
3. What are the characteristic skin findings in systemic lupus erythematosus?
4. Which skin lesions are associated with chronic liver disease?
5. Name three skin manifestations of HIV infection.
6. Which skin conditions may indicate an underlying malignancy?
7. What investigations would you request in a patient with suspected systemic sclerosis?
8. What is the importance of skin biopsy in systemic diseases?

Suggested Clinical Cases

- Case 1: A 52-year-old man with velvety hyperpigmented plaques on the neck and axillae, later found to have type 2 diabetes mellitus.
- Case 2: A 24-year-old woman with a malar rash, photosensitivity, and oral ulcers suggestive of systemic lupus erythematosus.
- Case 3: A 60-year-old patient with generalized pruritus, jaundice, and spider angiomas due to chronic liver disease.

Take-Home Messages

- The skin often provides the earliest clue to an underlying systemic disease.
- Careful dermatological examination can lead to early diagnosis of endocrine, autoimmune, infectious, hepatic, renal, hematological, gastrointestinal, and malignant disorders.
- Recognition of characteristic skin signs improves diagnostic accuracy and patient outcomes.
- A systematic approach combining history, examination, investigations, and multidisciplinary management is essential in patients with cutaneous manifestations of systemic diseases.