



ASHFAQ ACADEMY OF CLINICAL MEDICINE

Dermaesthetic OPD Masterclass

Sample notes · Day 2: Pigmentary Disorders

This is a short preview of the student notes and clinical reference you receive with the course. The full Day 2 notes cover pigmentary disorders and hair disorders, from melasma and vitiligo to hair loss.

Shown here: the approach to pigmentary disorders, Wood's lamp examination, the MASI score and the opening pages on melasma.

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PREVIEW EDITION

TOPIC 1 · FOUNDATIONAL FRAMEWORK

Approach to Pigmentary Disorders

1.1 Melanin Biology — The Basis for Everything in This Session

Melanocytes reside in the basal layer of the epidermis and in the hair follicle bulb. They synthesise melanin within specialised organelles (melanosomes) via the rate-limiting enzyme tyrosinase, which converts tyrosine to DOPA and then to dopaquinone — the precursor for both eumelanin (brown-black) and pheomelanin (red-yellow). Melanosomes are transferred to surrounding keratinocytes via dendritic processes. Pigmentary disease results from a disturbance at any point in this pathway: melanocyte number, melanocyte function/tyrosinase activity, melanosome transfer, or melanin distribution/degradation.

1.2 Wood's Lamp Examination — Technique & Interpretation

Wood's lamp emits long-wave UV-A (365nm) and, examined in a completely darkened room, reveals pigment depth by differential fluorescence — information not obtainable by naked-eye examination alone.

Finding	Depth	Prognosis	Example
Accentuated (darker)	Epidermal	Good — responds well to topicals	Superficial melasma, epidermal PIH
No change	Dermal	Poor — slow, partial response	Dermal melasma, established PIH
Partial accentuation	Mixed	Intermediate	Most common melasma pattern clinically
Chalk-white/blue-white	Absent melanin	N/A — total pigment loss	Vitiligo
Yellow-green	Surface (fungal)	N/A	Pityriasis versicolor
Coral-red	Surface (bacterial)	N/A	Erythrasma

★ CLINICAL PEARL

Wood's lamp accentuation correlates directly with topical treatment response. Always Wood's-lamp a pigmentary patient BEFORE setting treatment expectations — a dermal-predominant melasma patient told to expect the same result as an epidermal patient will be disappointed and lose confidence in the treatment plan.

1.3 Classification Map

Category	Definition	Examples
Hyperpigmentation	Excess melanin/pigment deposition	PIH, melasma, drug-induced (minocycline, antimalarials, amiodarone), Addison's disease, haemochromatosis, lichen planus pigmentosus
Hypopigmentation	Reduced but not absent	Pityriasis versicolor, post-inflammatory hypopigmentation,

	melanin	pityriasis alba
Depigmentation	Complete absence of melanin	Vitiligo, chemical leukoderma

1.4 MASI Score (Melasma Area and Severity Index)

Scores 4 facial regions — forehead, right malar, left malar, chin — for area of involvement (0–6) and for darkness + homogeneity (0–4 each). Formula: $(0.3 \times \text{forehead}) + (0.3 \times \text{R malar}) + (0.3 \times \text{L malar}) + (0.1 \times \text{chin})$, where each regional score = area $\times$ (darkness+homogeneity). Maximum score 48. Photograph and score at baseline and every 12 weeks; a reduction of ≥ 4 points is considered clinically meaningful improvement and should be used to justify continuing or escalating therapy.

1.5 When to Investigate Beyond Wood's Lamp

Clinical Scenario	Investigation	Purpose
Suspected drug-induced pigmentation	Detailed medication history $\pm$ biopsy	Confirms causative agent; biopsy shows drug/pigment deposits in dermis
Suspected systemic cause (diffuse hyperpigmentation)	Morning cortisol/ACTH, ferritin/iron studies, TFTs	Screens Addison's disease, haemochromatosis, thyroid dysfunction
Hypopigmented patch, endemic area	Sensation testing (light touch/pinprick/temperature)	Excludes leprosy — mandatory, not optional, in South Asia
Diagnostic uncertainty	Skin biopsy	Histology distinguishes LPP, vitiligo, PIH, and rare causes definitively

TOPIC 2 · COMPLETE CLINICAL REFERENCE

Melasma

2.1 Definition & Epidemiology

Melasma is a chronic, acquired, symmetrical facial hypermelanosis. Extremely common in South Asia — prevalence studies suggest up to 40% of women in this demographic have some degree of disease. Driven by the interaction of UV radiation, hormonal influence, and genetic predisposition.

2.2 Pathophysiology

Driver	Mechanism
UV radiation	The most potent trigger. UV-A and UV-B upregulate melanocyte-stimulating hormone and tyrosinase activity. Even brief, low-level daily exposure (walking to the car, sitting near a window) sustains active disease and undoes weeks of treatment progress.
Hormonal influence	Oestrogen and progesterone stimulate melanogenesis via MSH receptors on melanocytes — explains precipitation by pregnancy (chloasma) and combined oral contraceptives.
Genetic predisposition	Family history in up to 50% of patients. Darker Fitzpatrick types are significantly more susceptible — both the genetic substrate and ambient UV exposure are high in our population.
Visible light	Blue-violet visible light (400–450nm) independently triggers melanogenesis via opsin-3 receptors on melanocytes — a mechanism only established in the past decade, explaining why standard chemical UV filters alone are sometimes insufficient.

2.3 Clinical Classification

Distribution Pattern	Areas Involved	Notes
Centrofacial	Forehead, nose, upper lip, chin	Most common pattern
Malar	Cheeks, nose	
Mandibular	Jawline, ramus	Associated with OCP-triggered disease

Depth classification (epidermal/dermal/mixed) is assessed by Wood's lamp as above. Dermal-predominant melasma has a materially worse prognosis — counsel accordingly before starting treatment.

2.4 Investigations

Investigation	Finding / Purpose
Wood's lamp	Depth classification (epidermal/dermal/mixed) — predicts treatment response
Dermoscopy	Pseudo-network pigmentation pattern; helps exclude other diagnoses
MASI scoring	Baseline severity and objective monitoring of treatment response

Hormonal work-up (selected cases)	If associated with irregular cycles, hirsutism, or other hyperandrogenic signs — screen for PCOS
Patch testing (selected cases)	If a specific cosmetic/contactant trigger is suspected (rare)

2.5 Complications

- Significant psychological and quality-of-life impact — often disproportionate to objective severity, particularly in professional and pre-marital contexts in South Asian culture.
- Treatment-induced complications: ochronosis (paradoxical blue-grey discolouration from prolonged uninterrupted hydroquinone use), rebound hyperpigmentation from inappropriate laser/peel use, contact dermatitis from topical agents.

2.6 Management — Photoprotection (The Non-Negotiable Foundation)

⊕ PRESCRIBING BLUEPRINT: Sunscreen Protocol for Melasma

Formulation: Broad-spectrum SPF 50+ with UVA protection (PPD ≥ 16 or 4-star UVA). Mineral filters (zinc oxide/titanium dioxide) preferred; on their own (untinted) they give little visible-light protection. Iron oxide tint essential for visible-light protection.

Application: $2\text{mg}/\text{cm}^2 \approx \frac{1}{4}$ teaspoon for face+neck. Apply 20–30 min before sun exposure, as the LAST skincare step before makeup.

Reapplication: Every 2 hours of sun exposure; once each morning sufficient if indoors near windows only.

Duration: Lifelong daily habit — not a treatment course.

2.7 Management — Topical Agents (Exact Prescribing)

Agent	Conc.	Schedule	Duration / Key Rule
Hydroquinone	4%	Nocte	Max 12 wks continuous, then 4-wk break. Ochronosis risk with uninterrupted use.
Triple combo (Kligman)	HQ4%+Tret0.05%+Fluocinolone0.01%	Nocte	12-wk course only. Expect peeling/irritation weeks 1–4 — warn in advance.
Tranexamic acid (topical)	5%	BD	Blocks UV-melanocyte plasminogen signalling; layer under sunscreen AM
Tranexamic acid (oral)	250mg	BD × 12wks	Screen clotting/VTE history first; contraindicated in pregnancy
Azelaic acid	15–20%	BD	Safe in pregnancy; excellent maintenance agent
Niacinamide	4–5%	BD	Adjunct — blocks melanosome transfer, not synthesis
Kojic acid	2–4%	BD	Weaker tyrosinase inhibitor; can sensitise with prolonged use
Cysteamine	5%	15min contact,	Newer antioxidant option; third-line