



CLINICA
PUBLICATION

STRATUM CORNEUM

EPIDERMIS

DERMIS

HYPODERMIS

DNB Dermatology, Venereology & Leprosy

Solved Previous-Year Questions

FREE SAMPLE • VOLUMES 1, 2 & 3

A 100-page preview of the complete three-volume set

VOLUME 1

Basic Science, Diagnostics & Therapeutics

Parts 1–6 · 130 answers

VOLUME 2

Clinical Dermatology

Parts 7–16 · 210 answers

VOLUME 3

Venereology & HIV, Leprosy, Dermatosurgery & Aesthetics, Recent Advances

Parts 17–23 · 160 answers

REVIEWED BY

Dr Soumik Dey
DNB (Dermatology)

Dr Sirsa Ghosh
DNB (Dermatology)

UPDATED TILL OCTOBER 2026

Order the full set at www.clinicapublication.com

FREE SAMPLE

About this sample

This 100-page preview gives you a look inside all three volumes of **DNB Dermatology, Venereology & Leprosy — Solved Previous-Year Questions**. For each volume you will find its cover, its complete index — every question in that volume, with the papers and years in which it was asked — and a selection of full answers exactly as they appear in the book.

Pages keep their original page numbers, so you can match them against the index. Pages not included here are marked in the index but are available only in the full set, which contains **500 solved answers** across 23 parts.

What's inside

	CONTENTS	BOOK PAGES
Vol 1	Cover and complete index	3–10
Vol 1	Answers 1–3 · Keratinization, DEJ & desmosomes	11–19
Vol 1	Answers 74–76 · Dermoscopy	220–227
Vol 1	Answer 114 · JAK inhibitors	294–297
Vol 2	Cover and complete index	3–13
Vol 2	Answers 131–133 · Psoriasis	14–21
Vol 2	Answers 160–161 · DRESS and SJS/TEN	80–89
Vol 2	Answer 243 · Hidradenitis suppurativa	287–291
Vol 3	Cover and complete index	3–12
Vol 3	Answer 343 · Congenital syphilis	19–24
Vol 3	Answer 431 · ENL	225–231
Vol 3	Answer 442 · WHO MDT regimens	257–258
Vol 3	Answers 481–482 · Botulinum toxin & filler complications	347–352

The complete set

VOLUME 1

Basic Science, Diagnostics & Therapeutics

Parts 1–6 · 130 answers

VOLUME 2

Clinical Dermatology

Parts 7–16 · 210 answers

VOLUME 3

Venereology & HIV, Leprosy, Dermatosurgery & Aesthetics, Recent Advances

Parts 17–23 · 160 answers

REVIEWED BY

Dr Soumik Dey

DNB (Dermatology)

Dr Sirsa Ghosh

DNB (Dermatology)

TO ORDER THE FULL SET, VISIT

www.clinicapublication.com



CLINICA
PUBLICATION

STRATUM CORNEUM

EPIDERMIS

DERMIS

HYPODERMIS

DNB Dermatology, Venereology & Leprosy

Solved Previous-Year Questions

VOLUME 1

Basic Science, Diagnostics & Therapeutics

- Part 1 · Structure and Function of the Skin and Appendages
- Part 2 · Immunology, Physiology, Photobiology and Genetics
- Part 3 · Dermatopathology and Diagnostic Techniques
- Part 4 · Topical and Systemic Therapeutics
- Part 5 · Biologics, Small Molecules, Newer Drugs and Phototherapy
- Part 6 · Severity Scores, Research Methods and Emergency Care

REVIEWED BY

Dr Soumik Dey
DNB (Dermatology)

Dr Sirsa Ghosh
DNB (Dermatology)

*Based on Rook's Textbook of Dermatology (10th ed.), IADVL Textbook of Dermatology (5th ed.),
IAL Textbook of Leprosy (3rd ed.) and current Indian & international guidelines*

UPDATED TILL OCTOBER 2026

Order at www.clinicapublication.com

Index

HOW TO USE

Each answer groups every paper and year in which the question was asked (badge = how often) and answers exactly the parts asked, sized to the marks. The amber Management-basis strip names the guideline used (Indian programme or IADVL first, then international). [W#] tags mark guideline/web facts, listed in the Web-Sourced Facts Register. Book figures are reproduced with their book, volume and page; diagram questions also carry a schematic you can draw in the exam. Each answer ends with its Sources (book, chapter and pages).

A • Parts

Part 1 — Structure and Function of the Skin and Appendages	23 answers	11
Part 2 — Immunology, Physiology, Photobiology and Genetics	32 answers	82
Part 3 — Dermatopathology and Diagnostic Techniques	28 answers	169
Part 4 — Topical and Systemic Therapeutics	25 answers	237
Part 5 — Biologics, Small Molecules, Newer Drugs and Phototherapy	16 answers	287
Part 6 — Severity Scores, Research Methods and Emergency Care	6 answers	317
Part 7 — Papulosquamous and Eczematous Disorders	Volume 2	—
Part 8 — Urticaria, Drug Reactions and Mastocytosis	Volume 2	—
Part 9 — Bullous and Connective Tissue Diseases	Volume 2	—
Part 10 — Vasculitis, Neutrophilic, Granulomatous and Vascular Disorders	Volume 2	—
Part 11 — Pigmentary, Hair, Nail and Adnexal Disorders	Volume 2	—
Part 12 — Infections and Infestations	Volume 2	—
Part 13 — Genetic, Metabolic and Nutritional Disorders; Skin in Systemic Disease	Volume 2	—
Part 14 — Paediatric, Pregnancy, Erythroderma and Photodermatoses	Volume 2	—
Part 15 — Cutaneous Oncology	Volume 2	—
Part 16 — Psychodermatology, Environmental and Miscellaneous Dermatoses	Volume 2	—
Part 17 — Genital Anatomy and Sexually Transmitted Infections	Volume 3	—
Part 18 — Syndromic Management, HIV and STI Programmes	Volume 3	—
Part 19 — Leprosy: Basic Science, Classification and Diagnosis	Volume 3	—
Part 20 — Leprosy: Reactions, Neuritis, Treatment, Disability and Programme	Volume 3	—
Part 21 — Dermatotomy and Lasers	Volume 3	—
Part 22 — Aesthetic Dermatology	Volume 3	—
Part 23 — Recent Advances: AI, Tele dermatology and COVID-19	Volume 3	—

B • This volume, question by question

1. Keratinization: keratins & cornified cell envelope	2×	11
Asked in: Jun 2021 · Paper I · Q6 (10 marks) · Jun 2022 · Paper I · Q1 (10 marks)		
2. Dermo-epidermal junction / basement membrane zone: labelled diagram	4×	15
Asked in: Jun 2020 · Paper I · Q1(a) (5 marks) · Dec 2020 · Paper I · Q1(a) (5 marks) · Oct 2023 · Paper I · Q9(a) (5 marks) · May 2024 · Paper I · Q7(a) (2 marks)		
3. Desmosomes, cell junctions & desmosomal diseases	3×	17
Asked in: Jun 2021 · Paper I · Q3(a) (5 marks) · Dec 2022 · Paper I · Q1 (10 marks) · Apr 2023 · Paper I · Q7(a) (5 marks)		
4. Dermis: cells, fibres & extracellular matrix	1×	20
Asked in: Jun 2022 · Paper I · Q4 (10 marks)		
5. Collagen synthesis	1×	23
Asked in: Dec 2025 · Paper I · Q2(b) (5 marks)		
6. Skin barrier: structure, function & impaired barrier	2×	26
Asked in: Dec 2021 · Paper I · Q1 (10 marks) · Oct 2023 · Paper I · Q2 (10 marks)		

7. Melanogenesis & its regulation	3×	30
Asked in: Jun 2021 · Paper I · Q9(b) (5 marks) · Dec 2021 · Paper I · Q4(a) (4 marks) · May 2024 · Paper I · Q8(a) (6 marks)		
8. Epidermal melanin unit	2×	36
Asked in: Jun 2021 · Paper I · Q9(a) (5 marks) · Dec 2021 · Paper I · Q4(b) (3 marks)		
9. Epidermal vs follicular melanocytes	1×	37
Asked in: Jun 2022 · Paper I · Q9(a) (3 marks)		
10. Eumelanin & melanin types	1×	39
Asked in: Dec 2025 · Paper I · Q5(b) (5 marks)		
11. Hair cycle (diagram) & factors affecting it	4×	41
Asked in: Jun 2020 · Paper I · Q2(a) (5 marks) · Dec 2022 · Paper I · Q5(a) (4 marks) · Apr 2023 · Paper I · Q1(a) (5 marks) · Oct 2023 · Paper I · Q9(b) (5 marks)		
12. Hair keratins, hair colour & colouring agents	2×	45
Asked in: Dec 2022 · Paper I · Q5(b) (3 marks) · Dec 2022 · Paper I · Q5(c) (3 marks)		
13. Hair follicle: longitudinal section (diagram)	1×	47
Asked in: Oct 2024 · Paper I · Q4(a) (5 marks)		
14. Nail apparatus: diagram, development, parts & functions	6×	49
Asked in: Jun 2021 · Paper I · Q10(a) (5 marks) · Jun 2021 · Paper I · Q10(b) (5 marks) · Dec 2021 · Paper I · Q2(a) (3 marks) · May 2024 · Paper I · Q5(a) (6 marks) · Jun 2025 · Paper I · Q4(a) (3 marks) · Jun 2025 · Paper I · Q4(b) (3 marks)		
15. Hyperhidrosis: primary & management	3×	52
Asked in: Jun 2021 · Paper I · Q1(c) (5 marks) · Dec 2021 · Paper II · Q5(a) (3 marks) · Dec 2022 · Paper IV · Q4(a) (5 marks)		
16. Sweat glands (eccrine & apocrine): structure & function	4×	56
Asked in: Jun 2021 · Paper I · Q1(a) (3 marks) · Oct 2023 · Paper I · Q1 (10 marks) · Oct 2024 · Paper I · Q1(a) (3 marks) · Oct 2024 · Paper I · Q1(b) (3 marks)		
17. Sweat function tests	2×	62
Asked in: Jun 2021 · Paper I · Q1(b) (2 marks) · Oct 2024 · Paper I · Q7(b) (4 marks)		
18. Fox–Fordyce disease	1×	65
Asked in: Oct 2024 · Paper I · Q1(c) (4 marks)		
19. Pilosebaceous unit, sebum composition & sebaceous gland disorders	1×	67
Asked in: Jun 2020 · Paper I · Q4 (10 marks)		
20. Skin ageing: intrinsic & extrinsic	3×	71
Asked in: Jun 2022 · Paper IV · Q6(c) (4 marks) · May 2024 · Paper I · Q3 (10 marks) · Jun 2025 · Paper I · Q6(a) (5 marks)		
21. Histology of normal oral mucosa	1×	76
Asked in: Dec 2020 · Paper I · Q4(a) (4 marks)		
22. Muscles of facial expression & innervation	1×	78
Asked in: Dec 2022 · Paper I · Q3(a) (5 marks)		
23. Sensory innervation of hand & foot	1×	80
Asked in: Apr 2023 · Paper I · Q9(a) (5 marks)		
24. Antimicrobial peptides	3×	82
Asked in: Dec 2020 · Paper I · Q6(a) (3 marks) · Dec 2022 · Paper I · Q3(b) (3 marks) · May 2024 · Paper I · Q4 (10 marks)		
25. Toll-like receptors & innate immunity	3×	86
Asked in: Jun 2021 · Paper I · Q2(a) (5 marks) · Oct 2023 · Paper I · Q10(b) (5 marks) · Dec 2025 · Paper I · Q2(a) (5 marks)		
26. Regulatory T cells & resident memory T cells	3×	89
Asked in: Jun 2021 · Paper I · Q2(b) (5 marks) · May 2024 · Paper I · Q2(a) (5 marks) · May 2024 · Paper I · Q2(b) (5 marks)		
27. Adhesion molecules	1×	92
Asked in: Dec 2020 · Paper I · Q6(c) (4 marks)		
28. Langerhans cells, dendritic cells & antigen-presenting cells	3×	94
Asked in: Jun 2021 · Paper I · Q8(b) (5 marks) · Oct 2024 · Paper I · Q2 (10 marks) · Jun 2025 · Paper I · Q1(a) (5 marks)		
29. Mast cells	2×	97
Asked in: Dec 2020 · Paper I · Q6(b) (3 marks) · Oct 2024 · Paper I · Q10(a) (3 marks)		

30. Skin immune network: dendritic cells, mast cells & macrophages Asked in: Jun 2022 · Paper I · Q8 (10 marks)	1×	99
31. Merkel cells Asked in: Jun 2025 · Paper I · Q1(b) (5 marks)	1×	102
32. Plasma cells Asked in: Dec 2025 · Paper I · Q10(b) (5 marks)	1×	104
33. Cutaneous stem cells: properties, types & role Asked in: Dec 2020 · Paper I · Q2 (10 marks) · Jun 2022 · Paper I · Q5 (10 marks) · Dec 2022 · Paper I · Q4(a) (4 marks) · Dec 2025 · Paper I · Q5(a) (5 marks)	4×	105
34. Stem cell–based therapies Asked in: Jun 2021 · Paper I · Q3(b) (5 marks) · Dec 2022 · Paper I · Q4(b) (3 marks) · May 2024 · Paper IV · Q9(a) (3 marks)	3×	109
35. Gene therapy & gene editing Asked in: Dec 2022 · Paper I · Q4(c) (3 marks) · May 2024 · Paper IV · Q7(b) (3 marks)	2×	112
36. Wound healing: principles/stages, abnormal healing & newer therapies Asked in: Jun 2020 · Paper I · Q8 (10 marks) · Dec 2020 · Paper I · Q8 (10 marks) · Jun 2022 · Paper I · Q2 (10 marks) · Apr 2023 · Paper I · Q3(a) (5 marks) · Apr 2023 · Paper I · Q3(b) (5 marks) · Dec 2025 · Paper I · Q4 (10 marks)	6×	113
37. Skin substitutes in wound care Asked in: May 2024 · Paper IV · Q7(a) (4 marks)	1×	118
38. Itch: neurophysiology, mediators & anti-itch agents Asked in: Dec 2020 · Paper I · Q7(a) (6 marks) · Dec 2020 · Paper I · Q7(b) (4 marks) · Oct 2023 · Paper I · Q4 (10 marks) · May 2024 · Paper I · Q1(b) (6 marks) · Oct 2024 · Paper I · Q8(a) (5 marks) · Dec 2025 · Paper I · Q7(a) (6 marks)	6×	119
39. Pruritus: clinical types & senile pruritus Asked in: May 2024 · Paper I · Q1(a) (2 marks) · May 2024 · Paper I · Q1(c) (2 marks)	2×	123
40. Management of pruritus Asked in: Oct 2024 · Paper I · Q8(b) (5 marks)	1×	124
41. Skin as an endocrine organ Asked in: Dec 2020 · Paper I · Q5(a) (6 marks) · Dec 2025 · Paper I · Q3 (10 marks)	2×	126
42. Vitamin D synthesis in skin Asked in: Dec 2020 · Paper I · Q5(b) (4 marks)	1×	130
43. Skin as a thermoregulatory organ Asked in: May 2024 · Paper I · Q10(a) (4 marks)	1×	132
44. Effects of UV radiation on skin Asked in: Dec 2021 · Paper I · Q10(a) (3 marks) · Oct 2024 · Paper II · Q5(b) (3 marks) · Jun 2025 · Paper I · Q9(b) (5 marks)	3×	135
45. Solar / UV spectrum, sources & measurement Asked in: May 2024 · Paper I · Q10(b) (3 marks) · Oct 2024 · Paper II · Q5(a) (2 marks) · Jun 2025 · Paper I · Q9(a) (5 marks)	3×	137
46. Sunscreens & sun protection factor (SPF) Asked in: Apr 2023 · Paper I · Q4(a) (5 marks) · Dec 2025 · Paper I · Q8(b) (5 marks)	2×	141
47. Fitzpatrick skin phototypes Asked in: Dec 2021 · Paper I · Q10(c) (3 marks)	1×	142
48. Skin microbiome & normal flora (site-specific) Asked in: Jun 2021 · Paper I · Q8(a) (5 marks) · Dec 2021 · Paper I · Q5(a) (4 marks) · Oct 2023 · Paper I · Q3 (10 marks) · Dec 2025 · Paper I · Q1 (10 marks)	4×	143
49. Biofilms in skin disease Asked in: Dec 2021 · Paper I · Q5(c) (3 marks) · Oct 2023 · Paper II · Q6(b) (5 marks)	2×	147
50. Cutaneous mosaicism Asked in: Jun 2020 · Paper I · Q10(a) (5 marks) · Jun 2022 · Paper I · Q9(c) (3 marks) · Oct 2023 · Paper I · Q8(a) (5 marks) · Dec 2025 · Paper I · Q10(a) (5 marks)	4×	148
51. Pigmentary demarcation lines & linear lesions Asked in: Jun 2020 · Paper I · Q10(b) (5 marks) · Oct 2023 · Paper I · Q8(b) (5 marks)	2×	151

52. Prenatal diagnosis of genodermatoses	2×	153
Asked in: Dec 2020 · Paper I · Q9(a) (4 marks) · May 2024 · Paper I · Q7(c) (5 marks)		
53. Patterns of inheritance & genome sequencing	1×	155
Asked in: Jun 2022 · Paper I · Q3 (10 marks)		
54. Primary & secondary skin lesions; configuration & distribution	2×	160
Asked in: Jun 2022 · Paper I · Q10 (10 marks) · Jun 2025 · Paper I · Q3 (10 marks)		
55. Koebner (isomorphic) phenomenon	1×	164
Asked in: Jun 2020 · Paper I · Q7 (10 marks)		
56. Interface dermatitis & lichenoid tissue reaction	5×	169
Asked in: Jun 2020 · Paper I · Q9(a) (6 marks) · Jun 2020 · Paper I · Q9(b) (4 marks) · Oct 2023 · Paper I · Q10(a) (5 marks) · May 2024 · Paper I · Q6(a) (6 marks) · Oct 2024 · Paper I · Q5(b) (4 marks)		
57. Grenz zone	2×	173
Asked in: Oct 2023 · Paper II · Q5(a) (5 marks) · Dec 2025 · Paper I · Q8(a) (5 marks)		
58. Dermal deposition disorders (histopathology)	1×	175
Asked in: Dec 2022 · Paper I · Q7(a) (4 marks)		
59. Pseudoepitheliomatous & psoriasiform hyperplasia	2×	177
Asked in: Dec 2022 · Paper I · Q7(b) (3 marks) · Dec 2022 · Paper I · Q7(c) (3 marks)		
60. Cutaneous tissue reaction patterns (overview)	1×	179
Asked in: Oct 2024 · Paper I · Q5(a) (6 marks)		
61. Spongiotic reaction pattern	1×	183
Asked in: Dec 2025 · Paper I · Q9(a) (5 marks)		
62. Special stains (incl. PAS) in dermatopathology	3×	185
Asked in: Jun 2021 · Paper I · Q7(b) (5 marks) · Oct 2023 · Paper I · Q7(b) (5 marks) · Dec 2025 · Paper I · Q9(b) (5 marks)		
63. Immunohistochemistry in dermatology	3×	188
Asked in: Dec 2021 · Paper I · Q7(b) (4 marks) · Oct 2023 · Paper I · Q5(b) (5 marks) · May 2024 · Paper IV · Q5(b) (4 marks)		
64. Immunofluorescence: techniques & uses	1×	191
Asked in: Jun 2021 · Paper IV · Q9 (10 marks)		
65. Electron microscopy in skin diseases	1×	199
Asked in: Apr 2023 · Paper I · Q9(b) (5 marks)		
66. Skin biopsy: indications, techniques & fixatives	5×	201
Asked in: Jun 2021 · Paper IV · Q3(a) (4 marks) · Jun 2021 · Paper IV · Q3(b) (6 marks) · Dec 2022 · Paper I · Q6(a) (3 marks) · May 2024 · Paper IV · Q4(d) (3 marks) · Jun 2025 · Paper I · Q2 (10 marks)		
67. Wood's lamp	3×	206
Asked in: Jun 2021 · Paper I · Q4(b) (5 marks) · Dec 2022 · Paper I · Q6(c) (3 marks) · May 2024 · Paper IV · Q5(c) (3 marks)		
68. Confocal microscopy (reflectance / ex-vivo)	2×	209
Asked in: Jun 2020 · Paper I · Q3(b) (5 marks) · May 2024 · Paper IV · Q1(a) (3 marks)		
69. Tzanck smear & cytodiagnosis	2×	210
Asked in: Jun 2021 · Paper I · Q7(a) (5 marks) · Oct 2023 · Paper I · Q5(a) (5 marks)		
70. Dark field microscopy	1×	212
Asked in: Jun 2020 · Paper I · Q3(a) (5 marks)		
71. High-frequency ultrasound	1×	214
Asked in: Jun 2021 · Paper IV · Q10(b) (5 marks)		
72. Intradermal tests	1×	216
Asked in: Dec 2022 · Paper I · Q6(b) (4 marks)		
73. Trichoscopy in hair shaft disorders	2×	217
Asked in: Jun 2020 · Paper I · Q2(b) (5 marks) · Dec 2020 · Paper I · Q10(a) (4 marks)		
74. Dermoscopy of lichen planus	2×	220
Asked in: Dec 2020 · Paper IV · Q10(a) (3 marks) · Apr 2023 · Paper I · Q10(b) (6 marks)		

75. Dermoscopy of pigmentary disorders (incl. exogenous ochronosis)	2×	222
Asked in: Dec 2021 · Paper I · Q9(c) (3 marks) · Jun 2025 · Paper II · Q2(a) (6 marks)		
76. Dermoscopy of tumours (BCC, melanocytic lesions, vascular structures)	3×	224
Asked in: Dec 2021 · Paper I · Q9(a) (4 marks) · Dec 2022 · Paper I · Q2(a) (5 marks) · Dec 2022 · Paper I · Q2(b) (5 marks)		
77. Trichoscopy of scarring alopecias (LPP, DLE)	2×	228
Asked in: Oct 2023 · Paper II · Q7(a) (5 marks) · May 2024 · Paper I · Q9(b) (4 marks)		
78. Dermoscopy of psoriasis	1×	229
Asked in: Dec 2020 · Paper IV · Q10(c) (3 marks)		
79. Dermoscopy of vitiligo	1×	230
Asked in: Dec 2020 · Paper IV · Q10(b) (4 marks)		
80. Trichoscopy of alopecia areata	1×	232
Asked in: Jun 2021 · Paper I · Q5(b) (4 marks)		
81. Trichoscopy of tinea capitis	1×	233
Asked in: Dec 2021 · Paper I · Q9(b) (3 marks)		
82. Onychoscopy (dermoscopy of onychomycosis)	1×	234
Asked in: May 2024 · Paper IV · Q7(c) (3 marks)		
83. Ultraviolet-fluorescence dermoscopy	1×	235
Asked in: Jun 2025 · Paper IV · Q10(a) (4 marks)		
84. Calamine lotion & gentian violet	2×	237
Asked in: Dec 2020 · Paper IV · Q3(b) (5 marks) · Jun 2021 · Paper IV · Q1(b) (3 marks)		
85. Topical drug delivery: absorption, vehicles, enhancers & novel systems	4×	238
Asked in: Jun 2022 · Paper I · Q7 (10 marks) · Jun 2025 · Paper I · Q7(a) (4 marks) · Jun 2025 · Paper I · Q7(b) (3 marks) · Jun 2025 · Paper I · Q7(c) (3 marks)		
86. Topical amphotericin B	1×	242
Asked in: Dec 2021 · Paper IV · Q7(b) (3 marks)		
87. Nail lacquers	1×	242
Asked in: Apr 2023 · Paper IV · Q6(a) (4 marks)		
88. Topical vehicles & fingertip unit	2×	244
Asked in: Apr 2023 · Paper I · Q8(a) (6 marks) · Apr 2023 · Paper I · Q8(b) (4 marks)		
89. Iontophoresis	1×	247
Asked in: Jun 2025 · Paper I · Q10(a) (5 marks)		
90. Systemic corticosteroids: regimens & indications	2×	249
Asked in: Dec 2020 · Paper IV · Q6(a) (5 marks) · Jun 2025 · Paper I · Q5(a) (5 marks)		
91. Systemic steroid adverse effects (osteoporosis)	1×	250
Asked in: Apr 2023 · Paper IV · Q3(a) (6 marks)		
92. Topical corticosteroids: classification	1×	252
Asked in: Jun 2025 · Paper I · Q5(b) (5 marks)		
93. Dexamethasone–cyclophosphamide pulse (DCP) therapy	1×	254
Asked in: Dec 2025 · Paper II · Q1 (10 marks)		
94. Topical steroid misuse & topical steroid-dependent face	1×	257
Asked in: Dec 2025 · Paper IV · Q10(a) (5 marks)		
95. Methotrexate (use & monitoring)	2×	259
Asked in: Dec 2020 · Paper II · Q9(b) (6 marks) · Dec 2022 · Paper IV · Q10(a) (5 marks)		
96. Azathioprine	1×	262
Asked in: Dec 2020 · Paper IV · Q6(b) (5 marks)		
97. Therapeutic paradox in dermatology	1×	264
Asked in: Dec 2020 · Paper IV · Q7(b) (5 marks)		
98. Antioxidants in dermatology	1×	266
Asked in: Jun 2021 · Paper IV · Q8 (10 marks)		

99. Leflunomide	1×	269
Asked in: Dec 2021 · Paper IV · Q10(a) (3 marks)		
100. Newer antihistamines	1×	270
Asked in: Dec 2021 · Paper IV · Q5(a) (4 marks)		
101. Antimalarials in dermatology	1×	272
Asked in: Apr 2023 · Paper I · Q4(b) (5 marks)		
102. Ivermectin	1×	273
Asked in: Oct 2023 · Paper IV · Q4 (10 marks)		
103. Pentoxifylline	1×	276
Asked in: Oct 2023 · Paper IV · Q10(b) (5 marks)		
104. Lenalidomide	1×	277
Asked in: May 2024 · Paper IV · Q2(a) (3 marks)		
105. Opioid modulators: naltrexone & buprenorphine	2×	278
Asked in: May 2024 · Paper IV · Q2(b) (4 marks) · May 2024 · Paper IV · Q2(c) (3 marks)		
106. Intravenous immunoglobulin (IVIG)	1×	280
Asked in: Jun 2025 · Paper I · Q10(b) (5 marks)		
107. Retinoids: classification, topical retinoids & prescribing guidelines	2×	281
Asked in: Dec 2021 · Paper IV · Q7(a) (4 marks) · Oct 2024 · Paper IV · Q10 (10 marks)		
108. Retinoids: monitoring & teratogenicity	2×	285
Asked in: Dec 2022 · Paper IV · Q10(b) (5 marks) · Apr 2023 · Paper IV · Q3(b) (4 marks)		
109. IL-17 & IL-23 inhibitors (secukinumab, tildrakizumab)	2×	287
Asked in: Dec 2021 · Paper IV · Q10(b) (4 marks) · Oct 2023 · Paper IV · Q7(b) (3 marks)		
110. Rituximab: uses & work-up	2×	288
Asked in: Jun 2022 · Paper IV · Q2(b) (4 marks) · Apr 2023 · Paper II · Q3(b) (5 marks)		
111. Anakinra (IL-1 blockade)	1×	290
Asked in: Jun 2025 · Paper IV · Q7(c) (3 marks)		
112. CAR T-cell therapy	1×	291
Asked in: Jun 2025 · Paper IV · Q8(a) (5 marks)		
113. Nemozoliumab (anti-IL-31)	1×	292
Asked in: Dec 2025 · Paper IV · Q2(a) (5 marks)		
114. JAK inhibitors: mechanism, uses & adverse effects (AA, vitiligo, AD, psoriasis)	6×	294
Asked in: Dec 2020 · Paper IV · Q3(a) (5 marks) · Jun 2022 · Paper IV · Q2(a) (3 marks) · Dec 2022 · Paper IV · Q8(b) (3 marks) · May 2024 · Paper IV · Q3 (10 marks) · Jun 2025 · Paper IV · Q1 (10 marks) · Dec 2025 · Paper IV · Q2(b) (5 marks)		
115. Apremilast & PDE4 inhibitors	3×	298
Asked in: Dec 2021 · Paper IV · Q5(b) (3 marks) · Dec 2022 · Paper IV · Q8(a) (3 marks) · Dec 2025 · Paper IV · Q10(b) (5 marks)		
116. Bruton's tyrosine kinase (BTK) inhibitors	1×	301
Asked in: Jun 2025 · Paper IV · Q9(c) (4 marks)		
117. Ozenoxacin	2×	303
Asked in: Jun 2021 · Paper IV · Q1(a) (3 marks) · Jun 2025 · Paper IV · Q7(b) (4 marks)		
118. Trifarotene	2×	305
Asked in: Dec 2021 · Paper IV · Q10(c) (3 marks) · Oct 2023 · Paper IV · Q2(c) (3 marks)		
119. Antifibrotics: nintedanib & topical pirfenidone	2×	306
Asked in: Oct 2023 · Paper IV · Q7(a) (4 marks) · Dec 2025 · Paper IV · Q3(a) (3 marks)		
120. Oral minoxidil	2×	308
Asked in: Oct 2023 · Paper IV · Q7(c) (3 marks) · Dec 2025 · Paper IV · Q1(b) (5 marks)		
121. Berdazimer gel	1×	310
Asked in: Jun 2025 · Paper IV · Q7(a) (3 marks)		
122. mTOR inhibitors	1×	310
Asked in: Dec 2025 · Paper IV · Q5(b) (5 marks)		

123. Phototherapy: principles, dosing & NB-UVB Asked in: Dec 2022 · Paper II · Q6(c) (4 marks) · Dec 2025 · Paper I · Q7(b) (4 marks)	2×	312
124. Extracorporeal photopheresis Asked in: Oct 2023 · Paper I · Q6 (10 marks)	1×	314
125. PASI & disease severity scoring indices Asked in: Jun 2020 · Paper II · Q2(b) (3 marks) · Dec 2020 · Paper IV · Q8(a) (7 marks) · Dec 2020 · Paper IV · Q8(b) (3 marks)	3×	317
126. Quality-of-life indices (DLQI etc.) Asked in: Jun 2020 · Paper II · Q2(a) (7 marks) · Dec 2020 · Paper IV · Q7(a) (5 marks)	2×	321
127. Case-control vs cohort study, sample size & sampling methods Asked in: Jun 2020 · Paper I · Q6 (10 marks) · Dec 2020 · Paper I · Q3 (10 marks)	2×	323
128. Impact factor & Indian dermatology journals Asked in: Jun 2021 · Paper IV · Q10(a) (5 marks) · Dec 2021 · Paper IV · Q4(c) (3 marks)	2×	327
129. Systematic review, meta-analysis, levels of evidence & EBM Asked in: Dec 2021 · Paper IV · Q4(a) (4 marks) · Dec 2021 · Paper IV · Q4(b) (3 marks) · Jun 2022 · Paper IV · Q9(a) (2 marks)	3×	329
130. Basic & advanced cardiac life support (BLS/ACLS) Asked in: Dec 2022 · Paper I · Q10(a) (4 marks)	1×	332

C · High-yield list (asked 3× or more)

Nail apparatus: diagram, development, parts & functions	6×	49
Wound healing: principles/stages, abnormal healing & newer therapies	6×	113
Itch: neurophysiology, mediators & anti-itch agents	6×	119
JAK inhibitors: mechanism, uses & adverse effects (AA, vitiligo, AD, psoriasis)	6×	294
Interface dermatitis & lichenoid tissue reaction	5×	169
Skin biopsy: indications, techniques & fixatives	5×	201
Dermo-epidermal junction / basement membrane zone: labelled diagram	4×	15
Hair cycle (diagram) & factors affecting it	4×	41
Sweat glands (eccrine & apocrine): structure & function	4×	56
Cutaneous stem cells: properties, types & role	4×	105
Skin microbiome & normal flora (site-specific)	4×	143
Cutaneous mosaicism	4×	148
Topical drug delivery: absorption, vehicles, enhancers & novel systems	4×	238
Desmosomes, cell junctions & desmosomal diseases	3×	17
Melanogenesis & its regulation	3×	30
Hyperhidrosis: primary & management	3×	52
Skin ageing: intrinsic & extrinsic	3×	71
Antimicrobial peptides	3×	82
Toll-like receptors & innate immunity	3×	86
Regulatory T cells & resident memory T cells	3×	89
Langerhans cells, dendritic cells & antigen-presenting cells	3×	94
Stem cell-based therapies	3×	109
Effects of UV radiation on skin	3×	135
Solar / UV spectrum, sources & measurement	3×	137
Special stains (incl. PAS) in dermatopathology	3×	185
Immunohistochemistry in dermatology	3×	188

Wood's lamp	3×	206
Dermoscopy of tumours (BCC, melanocytic lesions, vascular structures)	3×	224
Apremilast & PDE4 inhibitors	3×	298
PASI & disease severity scoring indices	3×	317
Systematic review, meta-analysis, levels of evidence & EBM	3×	329

PART 1

Structure and Function of the Skin and Appendages

1. Keratinization: keratins & cornified cell envelope

ASKED 2* · 10-mark scope

Asked in: Jun 2021 · Paper I · Q6 (10 marks) · Jun 2022 · Paper I · Q1 (10 marks)

Q: Discuss the process of formation of the cornified cell envelope (CE). [10]

Q: Keratinization in skin: a) Structure of keratin. [3] b) Function of keratin. [2] c) Keratin gene mutation disorders in skin. [5] [10]

Formation of the cornified cell envelope (CE)

- **Corneocyte:** the outermost cell of the stratum corneum. It has lost its nucleus and cytoplasmic organelles and is flattened.
- **Cornified envelope:** a highly insoluble structure inside the plasma membrane. It forms by cross-linking of the soluble precursor involucrin and other proteins.
- **Enzyme:** an epidermal transglutaminase, synthesised in the high stratum spinosum, catalyses the cross-linking.
- **Epidermal differentiation complex (EDC):** about 25 genes in a c.2 Mb region on the long arm of chromosome 1.
 - The EDC is derived from at least three families of related genes.
 - EDC proteins have roles in structural integrity, signal transduction and cell cycle progression.
 - They may be disrupted in inflammatory or neoplastic disorders.
- **Main envelope proteins:** involucrin is made in the lower suprabasal cells and loricrin in the granular layer. Others are small proline-rich proteins (SPR1, SPR2), elafin, hornerin, envoplakin and keratolinin.
- **Thickness:** the protein envelope forms a layer about 15 nm thick on the cytoplasmic surface.
- **Lamellar granules (Odland bodies):** 100–300 nm. They are numerous in the uppermost spinous cells and move to the cell periphery in the granular layer.

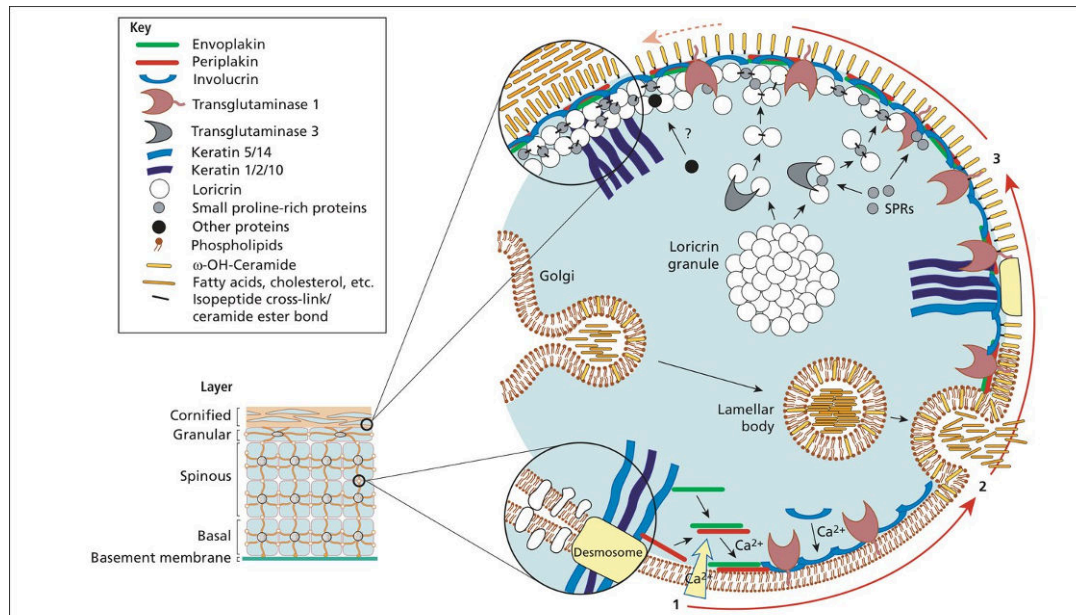


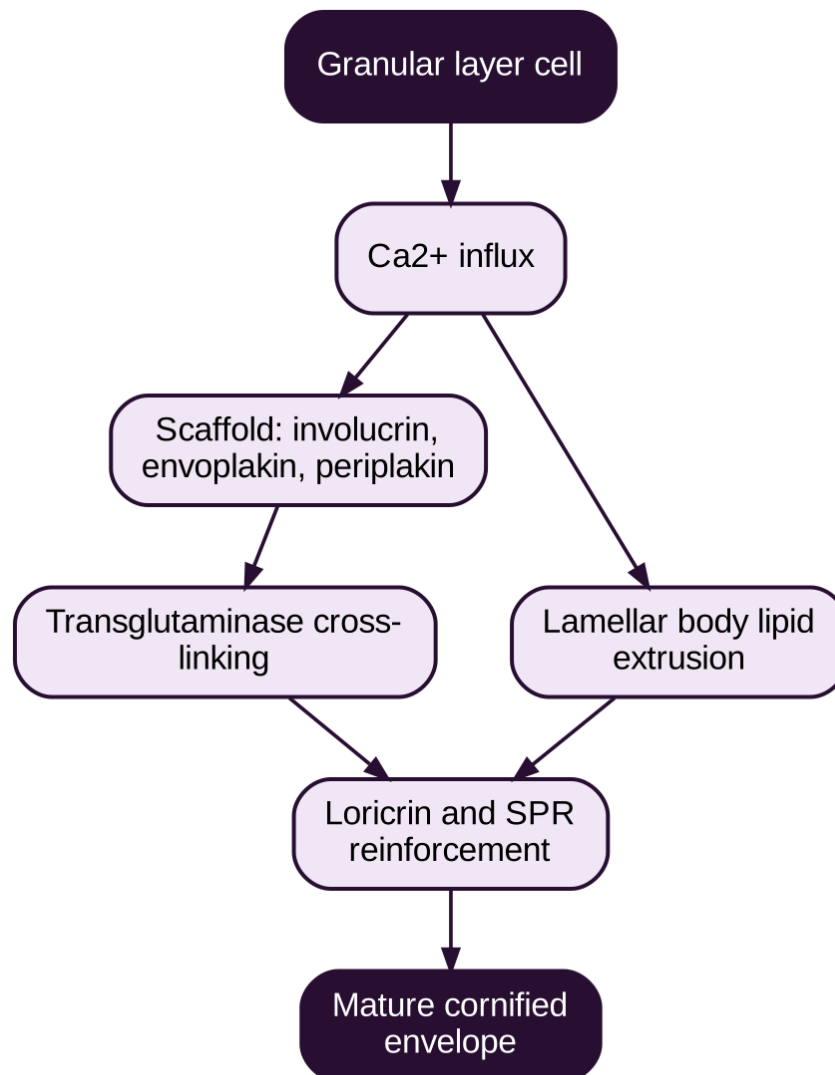
Figure 2.9 — Rook's Textbook of Dermatology 10e V1, p. 2.7. Assembly of the epidermal cornified cell envelope. In response to increasing intracellular calcium, an internal scaffold of desmosomal proteins is laid down along the plasma membrane.

Steps

1. **Intracellular Ca²⁺ rises.** An internal scaffold of desmosomal proteins is laid down along the plasma membrane.
2. **Transglutaminases** are calcium-dependent. They are activated by calcium influx and cross-link involucrin, loricrin and proline-rich proteins.
3. **Lamellar bodies** discharge ceramides, other fatty acids, cholesterol and cholesterol esters into the intercellular space. They form a lipid membrane.

4. **ω -hydroxyceramides** are bound to involucrin by transglutaminase-1. This connects the lipid envelope to the protein envelope.
5. The developing envelope is **reinforced** by loricrin, small proline-rich proteins, other desmosomal remnants and attached keratin filaments.
6. The result is a durable, flexible envelope. It gives mechanical and barrier function.
7. Under **filaggrin**, keratin filaments align into disulphide cross-linked macrofibres. Filaggrin is then broken down into hygroscopic amino acids, the **natural moisturising factor**.
8. **Desquamation**: lamellated intercellular lipid is degraded and the residual desmosomal links are lost.
9. In palmoplantar skin an extra electron-lucent **stratum lucidum** lies between the granulosum and corneum. Its cells are still nucleated ("transitional" cells).

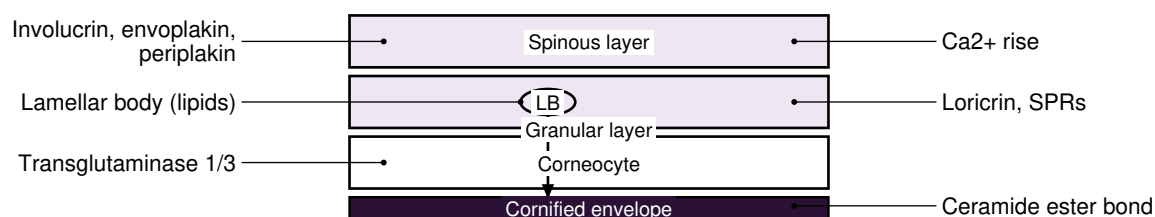
Flowchart 1: Cornified envelope assembly



Clinical correlation

- **Transglutaminase-1** defect: autosomal recessive congenital ichthyosis (lamellar ichthyosis/CIE). Diagnosis is by sequencing or in situ TG1 activity in cryostat sections.
- **Lipoxygenase E3/12B** (hepoxilin pathway) defects impair lamellar body secretion and intercellular lipid formation.
- **CERS3** mutations cause loss of very long acyl chains (C26–C34) and impair barrier formation.
- **Loricrin** frameshift mutations (LOR) give an abnormal arginine-rich C-terminus. The mutant protein enters the nucleus and interferes with cornification. This causes loricrin keratoderma.
- **Filaggrin** loss-of-function mutations cause ichthyosis vulgaris. They are a major risk factor for atopic eczema, asthma and systemic allergies.

- About two-thirds of ichthyosis vulgaris patients have two FLG mutations and a clear-cut phenotype.
- FLG mutations reduce natural moisturising factor and impair barrier formation.



Schematic (exam diagram): Cornified cell envelope assembly (granular layer to stratum corneum). Based on ROOK V1, Figure 2.9, pr.2.7.

Structure of keratin

- Keratins are **intermediate filaments**, the epithelial member of six types. The others are vimentin, GFAP, neurofilaments, desmin and peripherin.
- Intermediate filaments are 7–10 nm, between actin microfilaments (about 7 nm) and microtubules (20–25 nm).
- Each polypeptide has a classic **α -helical rod** with heptad repeats. It has four helical zones joined by interhelical linkers, with non-helical amino- and carboxy-terminals.
- There are **54 functional keratin genes** in two compact clusters, plus many pseudogenes.
- They fall into **type I (acidic)** and **type II (basic)** families.
- Partner acidic–basic pairs are co-expressed in a cell- and tissue-specific manner.
- An acidic–basic pair forms a **heterodimer**. Heterodimers assemble into protofibrils and protofilaments by an antiparallel stagger.
- Hair and nails express modified "hard" keratins rich in **cysteine**, which forms numerous cross-links.
- Retinoids, growth factors and hormones may regulate keratin gene expression.

Table 1: Keratin pairs by site

Site	Keratins
Simple epithelia	K8/K18
Basal keratinocytes	K5/K14
Suprabasal epidermis	K1/K10
Palmoplantar epidermis	K9
Superficial interfollicular epidermis	K2
Hair follicle bulge (stem cells)	K15
Hyperproliferation (wound, psoriasis)	K6/K16/K17

Function of keratin

- Forms the **cytoskeleton**, extending from the nucleus to the cell membrane.
- Attaches to **desmosomes** (cell–cell) and **hemidesmosomes** (cell–matrix). This gives mechanical strength and resilience.
- Filaggrin aggregates keratin filaments into **macrofibres**, which flatten the corneocyte.
- Has direct roles in **cell signalling, the stress response and apoptosis**.
- Provides structural strength to hair and nails through cysteine cross-links.
- Marks **differentiation**: K5/K14 in the basal layer and K1/K10 suprabasally.
- Also regulates cell size, growth, proliferation, migration and wound healing.

Keratin gene mutation disorders in skin

- At least **21 of the 54 keratins** are linked to monogenic disorders.
- The first keratin disorder described was **epidermolysis bullosa simplex** (K5 or K14).

- About half of the keratin genes are expressed in the hair follicle. Only a few are linked to disease.
- **Mechanism:** mutant keratins form cytoplasmic aggregates (clumps) or perinuclear shells. This causes epidermolytic hyperkeratosis (EHK) on histology.
- Aggregates are induced by trauma, heat, fever and infection, which modulate severity.
- Retinoids reduce aggregate formation in heat-stressed keratinocytes with a KRT10 mutation.
- Keratinopathic ichthyoses are traditionally autosomal dominant. They are ultra-rare, with a prevalence of 1 in 350 000 in Denmark.

Table 2: Keratin gene disorders

Disorder	Gene	Key feature
Epidermolysis bullosa simplex	KRT5, KRT14	Blistering after trauma
Epidermolytic ichthyosis	KRT1/KRT10	Erosions and erythroderma at birth, then hyperkeratosis; blisters, infections
Superficial epidermolytic ichthyosis	KRT2	Brown "moulting" scale
Ichthyosis Curth–Macklin	KRT1/KRT10	Spiky PPK, binuclear cells
CRIE (ichthyosis en confetti)	KRT10/KRT1	Reticular pattern, pronounced redness
Epidermolytic PPK	KRT9 (rarely KRT1)	Diffuse PPK, sharp red edge, sparing the dorsum
Pachyonychia congenita	KRT6/16/17	Painful focal keratoderma, wedge-shaped nails, cysts
Monilethrix, woolly hair, pseudofolliculitis barbae	Hair keratins	Hair disorders

Points on individual disorders

- **Epidermolytic ichthyosis:** KRT1 mutation gives epidermolytic PPK. KRT10 mutation spares the palms and soles.
- **EPPK:** histology shows epidermolytic change in suprabasal keratinocytes with eosinophilic inclusions. Prevalence is 4.4/100 000 in Northern Ireland.
- **Pachyonychia congenita:** oral leukokeratosis, natal teeth, steatocystomas (especially KRT17) and laryngeal involvement may occur.
 - Botulinum toxin restores function and reduces pain. Statins act through KRT6A promoter dependence on STAT1.
- **Diagnosis:** electron microscopy shows tonofilament aggregates. Mutation analysis confirms.

PEARLS

- Cornified envelope = involucrin scaffold + transglutaminase cross-linking + lipid envelope + loricrin and SPR reinforcement.
- Filaggrin loss (FLG) causes ichthyosis vulgaris and predisposes to atopic disease. Loricrin mutation causes loricrin keratoderma. TGM1 mutation causes ARCI.
- Keratin pairs: K5/K14 basal, K1/K10 suprabasal, K9 palmoplantar.
- First keratinopathy described: EBS (K5/K14).
- Histology of KRT1/KRT10 disease: epidermolytic hyperkeratosis.
- KRT10 mutation spares palms and soles. KRT1 mutation involves them.

Sources

- ROOK V1 — Ch 2 Structure and Function of the Skin — Keratinocytes — pp. 2.6–2.8
- ROOK V2 — Ch 63 Inherited Disorders of Cornification — pp. 63.4, 63.11, 63.13, 63.50, 63.52, 63.56
- IADVL V1 — Ch 14 Disorders of Keratinization — pp. 429–431

2. Dermo-epidermal junction / basement membrane zone: labelled diagram

ASKED 4* · 5-mark scope

Asked in: Jun 2020 · Paper I · Q1(a) (5 marks) · Dec 2020 · Paper I · Q1(a) (5 marks) · Oct 2023 · Paper I · Q9(a) (5 marks) · May 2024 · Paper I · Q7(a) (2 marks)

Q: Draw a labelled diagram of dermo-epidermal junction. [5]

Q: Draw a labelled diagram of dermo-epidermal junction. [5]

Q: Draw a labeled diagram of basement membrane zone. [5]

Q: Draw a labelled diagram of dermo-epidermal junction. [2]

Labelled diagram of dermo-epidermal junction

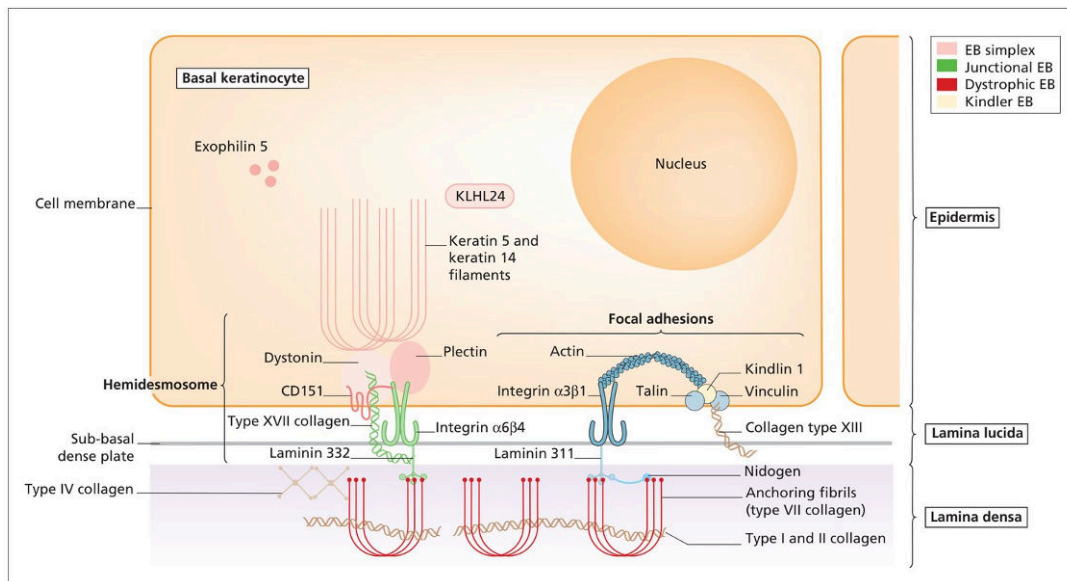
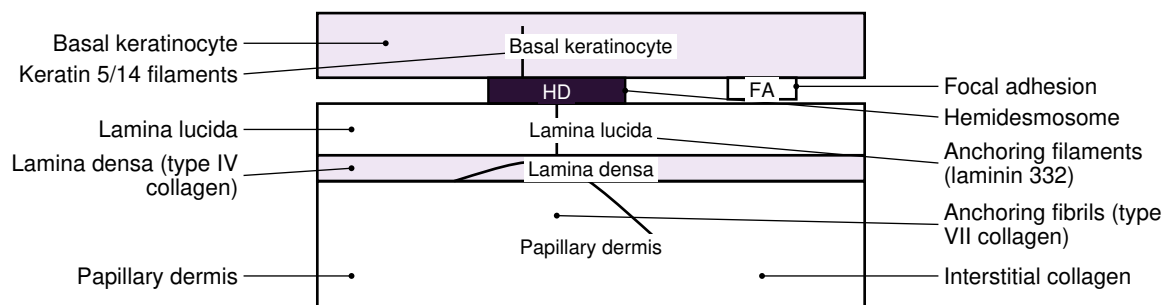


Figure 2.30 — Rook's Textbook of Dermatology 10e V1, p. 2.22. Structural components at or close to the dermal-epidermal junction that have physiological or disease relevance to maintaining adhesion at the derma



Schematic (exam diagram): Dermo-epidermal junction (hemidesmosome-anchoring filament-anchoring fibril complex). Based on ROOK V1, Figure 2.30 and Figure 2.31, pr.2.22; text pr.2.21, pr.2.26.

- **Dermal-epidermal junction (BMZ)** is the interface between the lower epidermis and the top layer of the dermis.
- It is **less than 200 µm** across and has over 30 interacting macromolecules (collagens, laminins, integrins).
- Draw it from top to bottom: basal keratinocyte → hemidesmosome → **lamina lucida** → **lamina densa** → sublamina densa with anchoring fibrils in the papillary dermis.
- The lamina lucida is the less electron-dense upper layer. It abuts the plasma membrane of basal keratinocytes.
- The lamina densa is electron-dense. Its lower part interacts with the upper-dermal matrix.
- A contiguous attachment network runs from the basal cell interior to the upper papillary dermis. It consists of **hemidesmosomes, anchoring filaments and anchoring fibrils**.
- The **hemidesmosome** has an inner plaque, an outer plaque and a sub-basal dense plate in the upper lamina lucida. Keratin intermediate filaments attach to it.

- **Anchoring filaments** cross the lamina lucida and concentrate under the hemidesmosomes. They merge with the lamina densa.
- **Anchoring fibrils** are U-shaped. They extend from the lower lamina densa into the dermis and entrap interstitial collagen fibres.
- **Focal adhesions** anchor actin microfilaments. They are not visible by transmission electron microscopy.

Labelled diagram of basement membrane zone

Use the same diagram. Add the molecular labels below and the Table 1 correlation.

- The BMZ stains positive with **PAS**.
- Transmission electron microscopy shows two layers of different optical density: lamina lucida and lamina densa.
- BMZ components are synthesised by **basal keratinocytes and dermal fibroblasts**.
- The major component is **type IV collagen**, chain composition $[\alpha 1(\text{IV})]_2\alpha 2(\text{IV})$ in the cutaneous BMZ.
- The hemidesmosome inner plaque contains **plectin** and the **230 kDa BP antigen**.
- The **180 kDa BP antigen (type XVII collagen)** is transmembrane and interacts with **$\alpha 6\beta 4$ integrin**.
- **Laminin 332** ($\alpha 3, \beta 3, \gamma 2$ chains) is probably the major anchoring filament component. It binds $\alpha 6\beta 4$ integrin and type VII collagen.
- Focal adhesions contain **talin and vinculin**. These connect $\alpha 3\beta 1$ integrin to actomyosin.
- **Type VII collagen** is the major component of anchoring fibrils. Its gene is **COL7A1**.

Table 1: BMZ components and the diseases from their defects

Component	Location	Key molecules	Disease when defective
Hemidesmosome	Basal cell → upper lamina lucida	K5/K14, plectin, BP230, BP180, $\alpha 6\beta 4$ integrin	EB; ITGA6/ITGB4 mutations: junctional EB with pyloric atresia
Anchoring filaments	Lamina lucida	Laminin 332	Severe junctional EB; split in the lamina lucida
Focal adhesion	Basal cell–BMZ	Kindlin-1, talin, vinculin	Kindler EB
Anchoring fibrils	Lamina densa → dermis	Type VII collagen	Dystrophic EB; total absence causes severe recessive dystrophic EB

PEARLS

- BMZ = lamina lucida + lamina densa + sublamina densa fibrous zone, held together by hemidesmosomes, anchoring filaments and anchoring fibrils.
- PAS-positive on histology.
- Draw the hemidesmosome → anchoring filament → anchoring fibril chain and label the key molecule at each level (plectin/BP230/BP180, laminin 332, type VII collagen).
- Exam trap: BP180 (type XVII collagen) is transmembrane and sits with $\alpha 6\beta 4$ integrin. BP230 lies in the inner plaque.
- Type IV collagen is the main BMZ collagen. Type VII collagen forms anchoring fibrils.

Sources

- ROOK V1 — Ch 2 Structure and Function of the Skin — §Dermal–epidermal basement membrane; Hemidesmosomes; Anchoring fibrils — pp. 2.2, 2.21–2.22, 2.25–2.27

3. Desmosomes, cell junctions & desmosomal diseases

ASKED 3* · 10-mark scope

Asked in: Jun 2021 · Paper I · Q3(a) (5 marks) · Dec 2022 · Paper I · Q1 (10 marks) · Apr 2023 · Paper I · Q7(a) (5 marks)

Q: Desmosomes and their clinical importance. [5]

Q: a) Desmosomes. [4] b) Other Cell Junctions in epidermis. [3] c) Skin diseases associated with desmosomal proteins. [3] [10]

Q: Desmosomes: structure, function and role in dermatological disorders. [5]

Desmosomes: structure

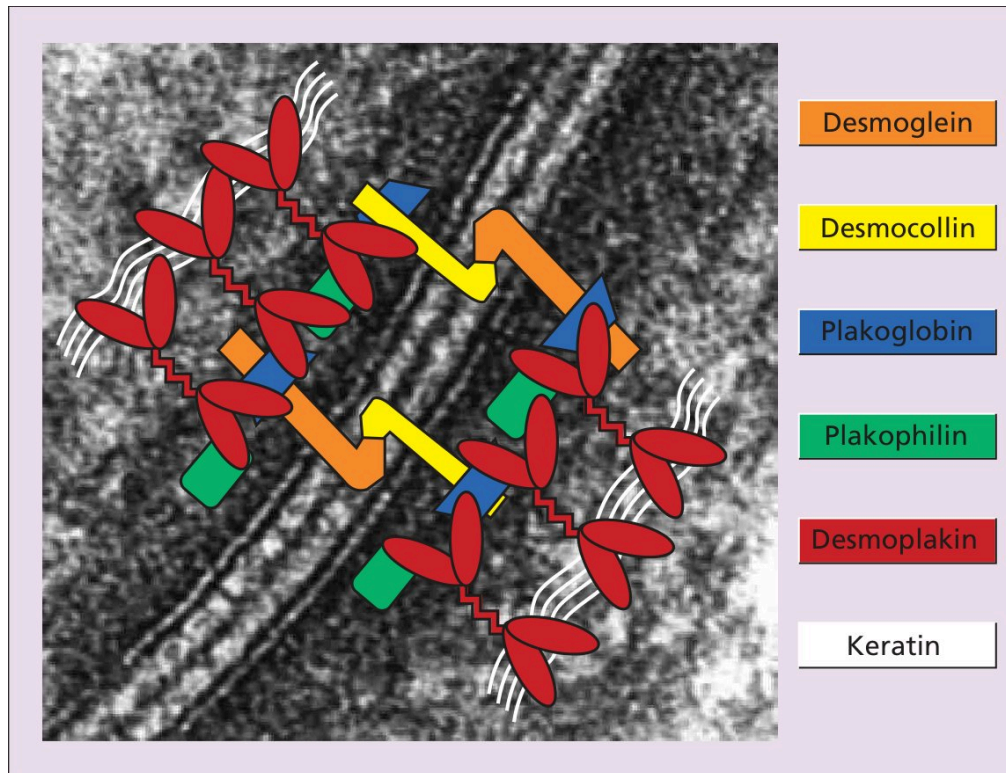
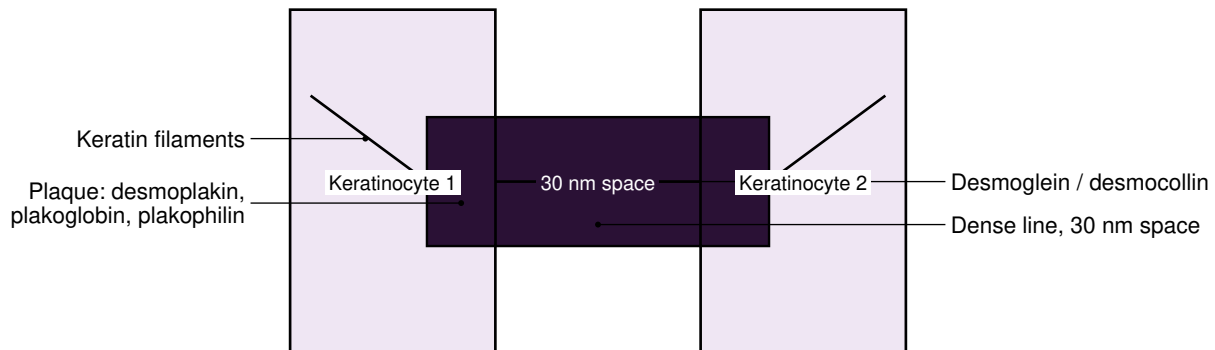


Figure 2.26 — Rook's Textbook of Dermatology 10e V1, p. 2.19. Macromolecular composition of desmosomes linking adjacent

- **Definition:** the major adhesion complex of the epidermis. It anchors keratin intermediate filaments to the cell membrane and bridges adjacent keratinocytes, so cells can withstand trauma.
- **Other sites:** myocardium, meninges, GI mucosa, bladder and cortex of lymph nodes.
- **Ultrastructure:** the membranes of two adjacent cells form a symmetrical junction. The central intercellular space is **30 nm** and contains a dense line.
- **Cytoplasmic plaque:** electron-dense plaques run parallel to the junction. They show three bands: a dense band next to the membrane, a less dense band, then a fibrillar area.
- **Components:** products of three gene superfamilies:
 - desmosomal cadherins
 - armadillo family of nuclear and junctional proteins
 - plakins
- **Transmembrane cadherins:** mostly heterophilic desmoglein–desmocollin associations. There are four epidermal desmogleins (**Dsg1–4**) and three desmocollins (**Dsc1–3**).
- **Differentiation-specific expression:**
 - Dsg1 and Dsc1 are preferentially superficial.
 - Dsg3 and Dsc3 are greater in basal keratinocytes.
- **Plaque proteins:** desmoplakin, plakoglobin and the armadillo protein plakophilin 1 attach the cadherins to the keratin network.
 - Plakophilin 1 is an important stabiliser of adhesion in differentiated keratinocytes.
 - Envoplakin and periplakin are other site-specific plakin envelope proteins.

- **Calcium-binding glycoproteins:** the desmogleins and desmocollins are calcium-binding members of the cadherin superfamily.
- **Histological correlate:** the spines of spinous cells correspond to abundant desmosomes. The spinous layer is 8–10 cells thick, and its K1/K10 keratin bundles insert peripherally into desmosomes.
- **Development:** desmosomes start to appear when intermediate-layer cells mature into the spinous layer at 12–16 weeks.



Schematic (exam diagram): Desmosome between two keratinocytes. Based on ROOK V1, Figure 2.26, pr.2.18-2.19.

Desmosomes: function

- **Adhesion:** the main adhesion complex bridging adjacent keratinocytes.
- **Mechanical stability:** anchoring keratin filaments to the membrane gives resistance to trauma, and the junction acts as a network of stability.
- **Layer-specific adhesion:** graded Dsg and Dsc expression gives different adhesive properties in superficial and basal layers.
 - Dsg1 and Dsg3 act cooperatively, which explains the site of blistering in pemphigus.
- **Corneocyte shedding:** corneocytes are joined by desmosomes, which eventually break apart so the cells are shed.
- **Mechanical versus physiological coupling:** desmosomes give mechanical coupling, whereas gap junctions are the sites of physiological communication.
- **Autoantigens:** desmosomal proteins also serve as autoantigens in immunobullous diseases.

Other cell junctions in the epidermis

Table 1: Epidermal cell junctions

Junction	Structure / proteins	Cytoskeleton link	Function
Desmosome	Dsg, Dsc, desmoplakin, plakoglobin, plakophilin	Keratin	Strong adhesion
Adherens junction	Nectin–afadin and cadherin–catenin complexes. Membranes ~20 nm apart, 0.2–0.5 µm diameter	Actin	Epithelial assembly, adhesion, barrier formation, cell motility
Gap junction	Connexons of six connexin subunits. 13 human connexins	–	Passage of metabolites <1000 Da and ions, which coordinates cells
Tight junction	Claudins, JAMs, occludin, ZO-1/2/3	Actin via ZO proteins	Paracellular permeability and barrier

- **Adherens junction details:**
 - Nectins link to actin via afadin (AF-6) and may be important in initial junction formation.
 - Cadherins form a complex with α -, β - and p120 catenin.
 - β -catenin signalling can activate Wnt pathways.
- **Gap junction details:**
 - Connexins fall into α , β and γ groups.
 - Six subunits assemble into a connexon in the Golgi, then move to the plasma membrane.
 - Stability is regulated by protein kinase C, Src kinase, calcium, calmodulin, cAMP and local pH.

- The junction is essential for cell synchronisation, differentiation, growth and metabolic coordination of the avascular epidermis.
- **Tight junction details:**
 - They are located between the first and second granular cell layers.
 - About 24 claudin subtypes exist. Seven are expressed in human epidermis, mainly claudins 1 and 4.
- **Basal-surface junctions:**
 - Hemidesmosomes link keratin to anchoring filaments in the lamina lucida. They contain plectin, BP230, type XVII collagen (BP180) and $\alpha 6 \beta 4$ integrin.
 - Focal adhesions contain talin and vinculin, which connect $\alpha 3 \beta 1$ integrin to actomyosin.

Skin diseases associated with desmosomal proteins

Pemphigus (autoimmune)

- **Pemphigus vulgaris (PV):** mainly anti-Dsg3 antibodies, with generally marked mucosal blistering.
- **Pemphigus foliaceus (PF):** anti-Dsg1 alone, so skin blisters without mucosal involvement.
- **Desmoglein compensation hypothesis:**
 - Skin has high Dsg1 throughout the epidermis, but Dsg3 only in the basal and immediate suprabasal layers. Anti-Dsg1 therefore blisters skin.
 - Oral epithelium has Dsg3 in all layers and little Dsg1. Anti-Dsg3 therefore causes severe oral blistering.
- **Paraneoplastic pemphigus:** multiple targets, including Dsg1, Dsg3, envoplakin, desmoplakin I/II, periplakin, epiplakin and BP230. It occurs with haematological malignancy.
- **IgA pemphigus:** intercellular IgA with Dsg1 and Dsg3, sometimes desmocolins.
- **Epitope spreading:** antibodies to multiple desmosomal proteins may develop, as in paraneoplastic pemphigus.
- **Non-desmoglein targets:** desmocolins, plakoglobin, E-cadherin, BP180 and acetylcholine receptors.
- **Pathogenicity of IgG:** passive transfer of purified IgG reproduces the blister. IgG also dissociates keratinocyte monolayers in vitro.
- **Indian point:** PV onset is relatively early in North India (mean c.40 years).

Non-autoimmune

- **SSSS and bullous impetigo:** bacterial toxin cleaves the extracellular domain of Dsg1. The toxin is a protease.

Inherited (genodermatoses)

Table 2: Genodermatoses due to defects in desmosomal components

Component	Disorder
Desmoglein 1	Type I striate PPK; SAM syndrome
Desmoglein 4	AR hypotrichosis; AR monilethrix
Desmocollin 2	PPK, woolly hair, cardiomyopathy
Desmocollin 3	Hypotrichosis and recurrent skin vesicles
Desmoplakin	Type II striate PPK; Carvajal syndrome; skin fragility/woolly hair; lethal acantholytic EB
Plakoglobin	Naxos disease; lethal congenital EB
Plakophilin 1	Ectodermal dysplasia–skin fragility syndrome
Corneodesmosin	Peeling skin syndrome type B; hypotrichosis simplex

- These disorders may also show hair abnormalities and cardiomyopathy, because the proteins are expressed in cardiac myocytes and hair follicles.

Other junction diseases

- **Plakoglobin:** Naxos disease was the first human mutation reported in an adherens junction component.
- **P-cadherin (CDH3):** hypotrichosis with juvenile macular dystrophy, and EEM syndrome.
- **Nectin-1 and -4:** ectodermal dysplasia syndromes (nectinopathies).

74. Dermoscopy of lichen planus

ASKED 2* · 6-mark scope

Asked in: Dec 2020 · Paper IV · Q10(a) (3 marks) · Apr 2023 · Paper I · Q10(b) (6 marks)

Q: Dermoscopy findings in lichen planus. [3]

Q: Write characteristic Dermoscopic findings in lichen planus of skin, nail and hair. [6]

Dermoscopy findings in lichen planus

- **Principle:** dermoscopy is a supportive tool. **Histology** remains the most useful test to confirm lichen planus (LP). The device helps when Wickham striae are seen better with it.
- **Hallmark:** a network of whitish striae (**Wickham striae**) with **red globules at the periphery**.



Figure 37.24 — Rook's Textbook of Dermatology 10e V2, p. 37.13. Dermoscopy image of lichen planus with typical Wickham striae. Courtesy

- **Pattern of the striae:** whitish structures in reticular, circular, white-dot or leaf-venation arrangements.
- **Borders:** linear vessels or red globules are usually visible.
- **Histological correlate:** a focal increase in thickness of the granular layer and infiltrate corresponds to Wickham striae.
- **Clinical background:** the primary lesion is a firm, shiny, polygonal, 1–3 mm violaceous papule with a white lacy tracery.
- **Resolved lesions:** greyish-brown pigmentation from melanin deposited in the superficial dermis. The book's dermoscopy figure shows brown dots and globules at the periphery.
- **Hypertrophic LP:** radially arranged white streaks, comedo-like openings and brown-black globules. Reddish vascular globules are also reported.
- **Mucosal LP (mucoscopy of the tongue):** a tricolored pattern. Gray-white areas (Wickham striae) are prominent, with glossy red erosions and violaceous to brown clods.
- **Standard IDS framework for inflammatory dermatoses:** five parameters are assessed:
 - vessels
 - scales
 - follicular findings
 - other structures
 - specific clues [w]

Dermoscopic findings in lichen planus of skin, nail and hair

Table 1: Dermoscopic findings by site in lichen planus

Site	Characteristic findings	Note
Skin (classic papule)	Whitish Wickham striae in reticular, circular, dotted or leaf-venation pattern	Red globules or linear vessels at the periphery
Skin (hypertrophic)	Radial white streaks, comedo-like openings, brown-black globules, reddish vascular globules	Wickham striae appear as whitish reticular areas
Skin (resolved)	Brown dots and globules at the periphery	Melanin in the superficial dermis
Nail (onychoscopy)	Trachyonychia in 40.51% and pitting in 34.18%	Also chromonychia, fragmentation of the nail body and splinter haemorrhage
Hair, early lichen planopilaris (LPP)	Whitish-gray perifollicular scales (peripilar casts) with perifollicular erythema and arboriform vessels [w2]	Reflects active perifollicular inflammation [w2]
Hair, late or scarring LPP	Irregular cicatricial alopecic areas, absent follicular openings and follicular plugging [w2]	Loss of ostia reflects fibrosis and is a poor prognostic sign [w3]

Skin

- Wickham striae are the diagnostic clue. They are most often seen on the volar wrists, lumbar region and ankles.
- Dermoscopy is useful when the striae are not obvious to the naked eye.

Nail

- Clinical correlates in nail LP:
 - Nail involvement occurs in up to 10% of cases.
 - The commonest change is exaggeration of the longitudinal lines, with linear depressions from thinning of the plate.
- Onychoscopy therefore shows trachyonychia and pitting most often.

Hair (lichen planopilaris)

- Clinical correlate: perifollicular scaling with scarring alopecia.
- Biopsy confirms LPP and excludes keratosis pilaris, Darier disease, follicular mucinosis and lupus erythematosus.
- Dermoscopic findings vary with the stage and activity of the disease. [w2]
- Peripilar cylindrical casts with perifollicular erythema and arborizing vessels suggest **early** LPP. [w3]
- Tubular casts are seen significantly more often in LPP than in frontal fibrosing alopecia (FFA). [w3]
- FFA shows the lonely hair sign and vellus hairs more often than LPP. [w3]
- The activity zone (hyperkeratosis, erythema) lies at the periphery of the patch, and the centre shows absent ostia and white scarring areas. [w2]

PEARLS

- LP dermoscopy hallmark: **Wickham striae** (white reticular, leaf-venation, circular or dotted) with peripheral red globules or linear vessels.
- Histological correlate of Wickham striae: focal thickening of the granular layer (wedge-shaped hypergranulosis).
- Hypertrophic LP: radial white streaks, comedo-like openings and brown-black globules.
- Nail LP: trachyonychia and pitting are the commonest onychoscopic signs.
- Hair LP (LPP): peripilar casts with perifollicular erythema indicate early disease. Absent follicular openings indicate scarring and a poor prognosis.
- Dermoscopy supports the diagnosis. Biopsy confirms it.

Sources

- ROOK V2 — Ch 37 Lichen Planus and Lichenoid Disorders — §Lichen planus (Presentation; Lichen planopilaris; Complications; Investigations) — pp. 37.3, 37.6, 37.11, 37.13
- IADVL V1 — Ch 32 Lichen Planus and Lichenoid Disorders — §Diagnosis; Dermoscopy in LP — p. 1210

- [w1] International Dermoscopy Society — Standardization of dermoscopic terminology and basic dermoscopic parameters to evaluate in general dermatology (non-neoplastic dermatoses) (Errichetti et al.) — Br J Dermatol 2020;182:454–467 — <https://pubmed.ncbi.nlm.nih.gov/31077336/>
- [w2] PMC — Dermoscopic findings in different clinical variants of lichen planus. Is dermoscopy useful? — <https://pmc.ncbi.nlm.nih.gov/articles/PMC4667604/>
- [w3] PMC — Trichoscopic Features of Lichen Planopilaris versus Frontal Fibrosing Alopecia: A Systematic Review — <https://pmc.ncbi.nlm.nih.gov/articles/PMC11928120/>

75. Dermoscopy of pigmentary disorders (incl. exogenous ochronosis)

ASKED 2* · 6-mark scope

Asked in: Dec 2021 · Paper I · Q9(c) (3 marks) · Jun 2025 · Paper II · Q2(a) (6 marks)

Q: Describe the dermoscopic features of: Exogenous ochronosis. [3]

Q: Role of dermoscopy in disorders of hyperpigmentation. [6]

Dermoscopic features of exogenous ochronosis

- **Definition:** exogenous ochronosis is discolouration of the skin after topical use, mainly of hydroquinone, and sometimes of phenol or resorcinol.
- **Clinical correlate:** grey-brown or blue-black macules appear on skin in contact with hydroquinone. The face, neck, back and extensor limbs are usual sites. Cartilage, sclerae and conjunctivae are spared.
- **Dermoscopy:** amorphous **blue-grey areas** that obliterate the follicular openings.
 - Irregular **grey-brown structures** are also seen. They are globular, annular or arciform.
 - These correspond to ochronotic pigment deposited in the dermis.
- **Reflectance confocal microscopy (emerging tool):** hyporefractile oval to **banana-shaped spaces** in the dermis. They correspond to the banana-shaped bodies seen on histology.
- **Histological correlate:** comma- or banana-shaped ochronotic collagen bundles. Ochre pigment lies in the papillary dermis.

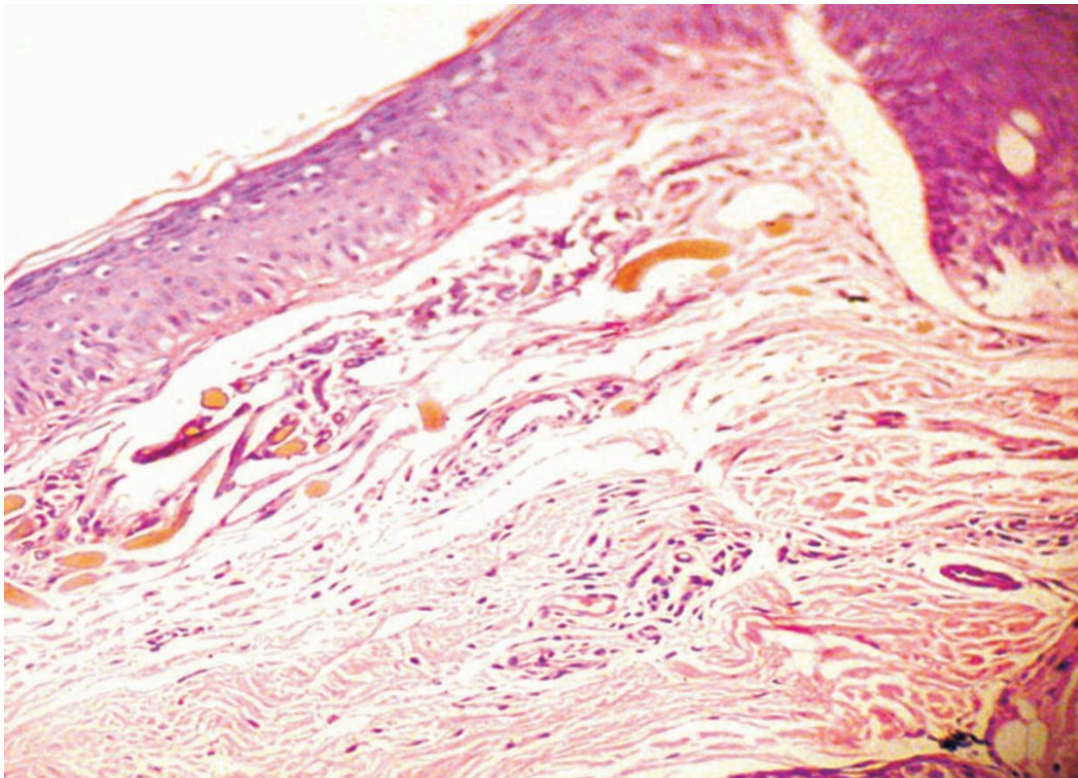


Fig. 42.18 — IADVL Textbook of Dermatology 5e V2, p. 1505. Histopathology of exogenous ochronosis demon-

- **Diagnostic value:**

- Early lesions may be mistaken for melasma. Hydroquinone is then continued and worsens the condition.
- Histopathology remains the gold standard. Dermoscopy is a non-invasive pointer.
- A history of prolonged hydroquinone use confirms exogenous ochronosis.

Role of dermoscopy in disorders of hyperpigmentation

- **Diagnosis in facial melanosis is mainly clinical.** The group includes melasma, erythema dyschromicum perstans, lichen planus pigmentosus, Riehl melanosis, post-inflammatory hyperpigmentation and exogenous ochronosis.
- **Wood's light:** it shows the depth of melanin. Epidermal melasma is light brown with enhanced contrast. Dermal melasma is grey-blue with less contrast, and it responds less to topical treatment.
- **Dermoscopy:** it is a non-invasive adjunct to this clinical and Wood's light assessment. It shows pigment pattern, vessels, scale and follicular findings in vivo. This guides biopsy and avoids wrong treatment, as in ochronosis mistaken for melasma.
- **Standardised terminology:**
 - The International Dermoscopy Society (IDS) has issued separate consensus vocabularies for skin neoplasms and for non-neoplastic dermatoses [w1].
 - **Non-neoplastic dermatoses** are inflammatory, infiltrative and infectious. The consensus defines five basic parameters for them (Table 1) [w1].
 - **Consensus size:** 31 subitems reached agreement [w1].
 - **Pigmented and neoplastic lesions:** the 2016 third consensus accepted both the metaphoric and the descriptive (5 basic terms) terminologies. It asked for definitions of metaphoric terms, avoidance of synonyms and no new metaphoric terms [w1].
 - **Skin of colour:** the IDS tumour criteria may also be used in dark phototypes. A few items, such as "clods, pink and small", are exceptions [w1].
- **Specific uses in hyperpigmentation:**
 - **Exogenous ochronosis:** blue-grey amorphous areas obliterating follicles, plus globular, annular or arciform grey-brown structures.
 - **Periorbital hyperpigmentation:** the eyelid stretch test separates true pigmentation from shadow effect.
 - **Differential diagnosis:** many other conditions share the same macular grey-brown pigmentation clinically, such as erythema dyschromicum perstans, lichen planus pigmentosus and Riehl melanosis.
 - **Hair and nails:** trichoscopy should be done at least once in every patient treated for hair loss [w1].
- **Limitation:** histopathology remains the gold standard where dermoscopy is not conclusive.

Table 1: IDS basic dermoscopic parameters for non-neoplastic dermatoses [w1]

No.	Parameter	What is described
1	Vessels	Morphology and distribution
2	Scales	Colour and distribution
3	Follicular findings	Follicular openings and perifollicular change
4	Other structures	Colour and morphology
5	Specific clues	Disease-specific signs

PEARLS

- Dermoscopy of exogenous ochronosis: **blue-grey amorphous areas** obliterating follicular openings, with globular, annular or arciform grey-brown structures.
- Histology shows **banana-shaped** ochre collagen bundles. Reflectance confocal microscopy shows banana-shaped hyporefractile spaces.
- Exogenous ochronosis can arise with hydroquinone at 2% or less. It spares cartilage and sclerae.
- Melasma looks similar early on, so a blue-grey dermoscopic pattern in a hydroquinone user should prompt a biopsy.
- IDS non-neoplastic dermatoses consensus: five parameters (vessels, scales, follicular findings, other structures, specific clues).

Sources

- ROOK V3 — Ch 86 Acquired Pigmentary Disorders — §Acquired hypermelanosis; §Non-melanin pigmentation — pp. 86.11, 86.50–86.51
- IADVL V2 — Ch 42 Hyperpigmentary Disorders — pp. 1497, 1503–1505
- [w] International Dermoscopy Society — Standardization of dermoscopic terminology and basic dermoscopic parameters to evaluate in general dermatology (non-neoplastic dermatoses), Errichetti et al., Br J Dermatol 2020;182:454–467; with the IDS third consensus on pigmented and neoplastic lesions (Kittler et al., J Am Acad Dermatol 2016;74:1093–1106) and the European trichoscopy consensus (JEADV 2021) — <https://pubmed.ncbi.nlm.nih.gov/31077336/>

76. Dermoscopy of tumours (BCC, melanocytic lesions, vascular structures)

ASKED 3* · 10-mark scope

Asked in: Dec 2021 · Paper I · Q9(a) (4 marks) · Dec 2022 · Paper I · Q2(a) (5 marks) · Dec 2022 · Paper I · Q2(b) (5 marks)

Q: Describe the dermoscopic features of: Basal cell carcinoma. [4]

Q: Dermoscopy: Vascular structures. [5]

Q: Dermoscopy: Benign melanocytic lesion versus Melanoma. [5]

Dermoscopic features of basal cell carcinoma

- Dermoscopy with non-polarised or cross-polarised light is an indispensable adjunct. It helps diagnose BCC and separate it from other tumours and inflammatory conditions.
- Adding dermoscopy to naked-eye examination raises sensitivity in expert hands from 66.9% to 85%. Specificity rises from 97.2% to 98.2%. The greatest benefit is in pigmented BCC.
- **Vascular structures:** arborising vessels and short fine telangiectasia.
 - Arborising vessels are the most noteworthy structure.
- **Pigment structures:** maple-leaf-like areas, spoke-wheel areas, multiple blue-grey globules, in-focus dots and concentric structures.
- **Other structures:** ulceration, multiple small erosions, shiny white-red structureless areas and white streaks.
- Metaphoric and descriptive equivalents:
 - Blue-gray ovoid nests = blue-gray globules.
 - Maple-leaf structures = pigmented peripheral radial lines with a common base.
 - Spoke-wheel structures = pigmented radial lines converging at a central dot or clod.
- Nodular BCC shows white and grey-brown structureless areas, blue-grey globules and telangiectatic vessels.
- Superficial BCC shows spoke-wheel areas, concentric structures, and arborising, comma and telangiectatic vessels.
- Criteria exist for each subtype. The greatest use is to differentiate BCC from benign naevi, Bowen disease, SCC and melanoma.
- Intradermal naevus (IDN) versus BCC:
 - IDN wobble on contact dermoscopy with horizontal pressure; BCC tends not to.
 - IDN have limited arborisation, sometimes a single 'Y'-shaped vessel.
 - BCC vessels are sharper in focus and brighter red.
 - IDN show no shiny white blotches or strands on polarised dermoscopy.

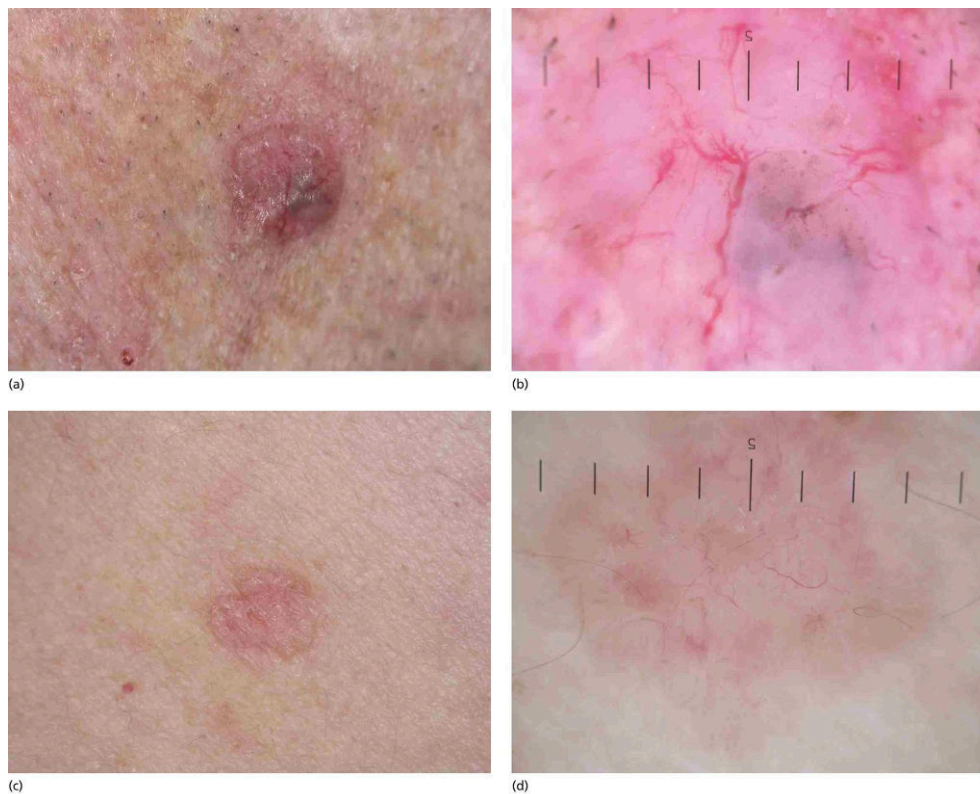


Figure 140.9 — Rook's Textbook of Dermatology 10e V4, p. 140.11. Dermoscopic images of basal cell carcinoma (BCC). (a) Nodular BCC. (b) Corresponding dermoscopic image showing white and grey-brown structureless

Dermoscopy: vascular structures

- Polarised dermoscopy shows deeper structures such as blood vessels, collagen and dermal pigment to about 100 μm . Surface glare is minimised.
- Assess vessels by (i) morphology, (ii) arrangement and (iii) other features such as halo and background colour.
- Use non-contact polarised dermoscopy to see vessels best.
- With non-polarised dermoscopy, avoid blanching of vessels from pressure. Use a gel interface with minimal pressure.
- Vessels are **monomorphic** when one morphology predominates. They are **polymorphic** when different morphologies are present.
- Basic morphologies: straight, curved, spiral, looped, serpentine, dotted, globular, coiled, branching and comma.
- Vessel pattern is a diagnostic clue:
 - Uniform-sized dotted vessels, regularly distributed, suggest psoriasis.
 - Polymorphic vascular pattern suggests malignancy (non-specific).

Table 1: Vessel morphology and associated lesions

Vessel (metaphoric term)	Description	Associated lesions
Comma	Short, curved	Dermal naevi, seborrhoeic keratosis, BCC
Hairpin	Linear loop	Viral wart, keratoacanthoma
Arborising	Branching, progressively tapering	BCC
Cherry blossom	Branching with expanded tips	Poroma
Glomerular	Coiled	Bowen's disease, psoriasis, clear cell acanthoma
Corkscrew	Helical	Squamous cell carcinoma

- In melanocytic tumours, vessels other than comma vessels in IDN should raise concern for melanoma.
- Atypical vessels:
 - Comma vessels in lesions that are not clinically IDN.
 - Dotted, linear irregular or corkscrew vessels.

- Milky-red globules and milky-red areas.
- The most common melanoma vessel pattern is polymorphous, with two or more morphologies in one lesion.
- Corkscrew vessels are usually seen in nodular or desmoplastic melanoma and in metastases.
- Dotted vessels over a milky-red background suggest melanoma or Spitz naevus.
- Terminology: the International Dermoscopy Society (IDS) consensus for non-neoplastic dermatoses lists vessels (morphology and distribution) as the first of five basic parameters. The others are scales, follicular findings, other structures and specific clues [w].

Dermoscopy: benign melanocytic lesion versus melanoma

- Naevi tend to show symmetrical, organised patterns. Melanomas tend to show asymmetrical, disorganised patterns with at least one melanoma-specific structure.
- Benign patterns include homogeneous brown, homogeneous tan/pink, and the patterns of IDN and Spitz naevi.
- Spitz naevi can overlap with melanoma. An excision biopsy is recommended for symmetric spitzoid lesions in patients older than 12 years.

Table 2: Benign melanocytic lesion versus melanoma

Feature	Benign naevus	Melanoma
Overall pattern	Organised, symmetric	Disorganised, asymmetric, chaotic
Network	Regular	Atypical network: lines of varying colour and thickness, holes of varying diameter
Streaks	Typical streaks (Spitz) evenly around the perimeter	Irregular, focal peripheral streaks
Dots/globules	Few regular dots/globules	Varying size, shape and colour, disorganised
Blue colour	Homogeneous steel-blue over the entire lesion (blue naevus)	Blue-white veil, not covering the whole lesion, multiple hues
Vessels	Comma vessels in IDN	Polymorphous, dotted, corkscrew, milky-red areas
Regression	Absent	Scar-like areas and peppering, often more than 50% of the surface

- Other melanoma-specific structures:
 - Negative network.
 - Angulated lines.
 - Shiny white lines, seen only with polarised light.
 - Atypical blotch.
 - Peripheral tan structureless areas, covering more than 10% of the lesion.
- Newly acquired homogeneous brown lesions in adults need caution. If non-palpable, monitor them digitally and biopsy if they change.
- A homogeneous tan/pink naevus is expected in skin types I–II. In darker skin, a new one should raise concern for amelanotic melanoma.
- Use the comparative approach: an outlier ("ugly duckling") lesion raises suspicion.
- Melanoma on special sites:
 - Face and sun-damaged skin: annular-granular pattern, asymmetric follicular openings, rhomboidal structures, circle within a circle.
 - Acral skin: parallel ridge pattern. In darker skin, ridge pigmentation can be normal, so this cannot be relied on.
- Digital dermoscopy follow-up detects featureless melanomas by change.

Table 145.2 Melanoma-specific structures		
Dermoscopic structure	Definition	Schematic illustration
Atypical network	Increased variability in the width of the network lines, their colour and distribution. The hole sizes also have increased variability. The network can appear broken up (non-contiguous), appearing as branched streaks, and the network may end abruptly at the periphery	
Angulated lines	Brown to bluish grey dots and/or lines arranged in an angulated linear pattern	
Negative network	Serpiginous interconnecting hypopigmented lines, which surround irregularly shaped pigmented structures that resemble elongated curvilinear globules. It can be seen diffusely throughout the lesion or focally and asymmetrically located within the lesion	
Streaks, including pseudopods and radial streaming	Radial projections located at the periphery of the lesion, extending from the tumour toward the surrounding normal skin. The presence of irregular, asymmetrical and focally distributed streaks are highly suggestive of melanoma. Pseudopods are finger-like projections with small knobs at their tips, whereas radial streaming are the same structures without the knobs. Because both structures represent confluent junctional nests of melanocytes, they are now both categorised under the term streaks	
Shiny white lines	Shiny white linear streaks that are often orientated parallel or orthogonal to each other	
Atypical dots or globules	Dots are small, round structures, which may be black, brown and/or blue-grey in colour. In melanoma, dots vary in size, colour and distribution, tending to be located towards the periphery of the lesion, and are not associated with the pigmented network lines. Globules consist of 3–5 or more clustered, well-demarcated, round to oval structures that may be brown, black, blue and/or white in colour and that are larger than dots. In melanoma they are usually multiple and of differing sizes, shapes and colours. They are often asymmetrically and/or focally distributed within the lesion	
Atypical blotch, including off-centre blotch or multiple asymmetrically located blotches	Dark-brown to black, usually homogeneous areas with varying hues of pigment that obscure visualisation of any other structures. In melanoma, blotches are asymmetrically and/or focally located towards the periphery of the lesion or can present as multiple blotches. Eccentric peripheral hyperpigmentation is often found in melanoma	
Regression structures (white scar-like depigmentation and/or blue-grey granularity or peppering)	Consist of granularity (also known as peppering) and scar-like areas. When both are present together, it gives the appearance of a blue-white veil over a macular area. In melanoma, regression structures tend to be asymmetrically located and often involve more than 50% of the lesion's surface area	
Blue-white veil	Confluent blue pigmentation of varying hues with an overlying white 'ground glass' haze, which tend to be asymmetrically located and seen over a raised area of the lesion.	

Table 145.2 — Rook's Textbook of Dermatology 10e V4, p. 145.8. Melanoma-specific structures.

Table 145.2 (continued)

Dermoscopic structure	Definition	Schematic illustration
Atypical vascular structures	<i>Dotted vessels</i> over milky-red background suggests melanoma or Spitz naevus <i>Serpentine vessels</i> : linear and irregular <i>Polymorphous vessels</i> : two or more vessel morphologies within the same lesion <i>Corkscrew vessels</i> : usually seen in nodular or desmoplastic melanoma, and melanoma metastases	
Peripheral tan structureless areas	Structureless light brown area/s located at the periphery of the lesion and encompassing more than 10% of a lesion's surface area	

Table 145.2 (continued, 2/2)

PEARLS

- BCC hallmark: arborising vessels, with spoke-wheel, maple-leaf and blue-grey globule pigment structures.
- Polarised dermoscopy shows vessels and shiny white lines. Non-polarised dermoscopy shows surface scale and keratin.
- IDN wobble on contact dermoscopy and BCC does not.
- The most common melanoma vessel pattern is polymorphous. Corkscrew vessels occur in nodular or desmoplastic melanoma and in metastases.
- Naevus is organised and symmetric. Melanoma is disorganised with at least one specific structure.
- Always interpret dermoscopy in the clinical context and the skin phototype.

Sources

- ROOK V4 — Ch 140 Basal Cell Carcinoma — pp. 140.10–140.11
- ROOK V4 — Ch 145 Dermoscopy of Melanoma and Naevi — pp. 145.6–145.11, 145.15
- IADVL V1 — Ch 7 Basics of Dermoscopy — pp. 189, 191–193
- [W1] International Dermoscopy Society — Standardization of dermoscopic terminology and basic dermoscopic parameters to evaluate in general dermatology (non-neoplastic dermatoses) (Errichetti et al.) — Br J Dermatol 2020;182:454–467 — <https://pubmed.ncbi.nlm.nih.gov/31077336/>

114. JAK inhibitors: mechanism, uses & adverse effects (AA, vitiligo, AD, psoriasis)

ASKED 6* · 10-mark scope

Asked in: Dec 2020 · Paper IV · Q3(a) (5 marks) · Jun 2022 · Paper IV · Q2(a) (3 marks) · Dec 2022 · Paper IV · Q8(b) (3 marks) · May 2024 · Paper IV · Q3 (10 marks) · Jun 2025 · Paper IV · Q1 (10 marks) · Dec 2025 · Paper IV · Q2(b) (5 marks)

Q: Mechanism of action of tofacitinib and its uses in Dermatology. [5]

Q: JAK inhibitors in alopecia areata. [3]

Q: Janus Kinase inhibitors. [3]

Q: Discuss the use of JAK inhibitors in: a) Alopecia areata. [4] b) Vitiligo. [3] c) Atopic dermatitis. [3] [10]

Q: JAK inhibitors in psoriasis: a) Indications. [3] b) Mechanism of action. [3] c) Adverse effects. [4] [10]

Q: Upadacitinib. [5]

Janus kinase inhibitors (JAKi)

- **Definition:** small-molecule inhibitors of intracellular JAK activity. JAKs are intracellular tyrosine kinases that transduce cytokine signals via the JAK/STAT pathway.
- **JAK family:** four members, **JAK1, JAK2, JAK3 and TYK2**.
- **Licensed for dermatological indications:** abrocitinib and upadacitinib (more specific to JAK1), and baricitinib (JAK1 and JAK2).
- **First-generation JAKi:** tofacitinib bound all three JAKs. Newer drugs are relatively selective because non-selective JAKi caused serious systemic adverse events in other fields.
- **Onset in AD:** itch improves within a week and clinical improvement is seen within 4 weeks.

Table 1: Oral JAKi doses (once daily)

Drug	Lower dose	Upper dose
Abrocitinib	100 mg	200 mg
Baricitinib	2 mg	4 mg
Upadacitinib	15 mg	30 mg

- Table 1 is from.
- The lower dose is used if creatinine clearance is 30–60 mL/min and in those over 65 years. Treatment is not recommended if clearance is <30 mL/min.

Mechanism of action

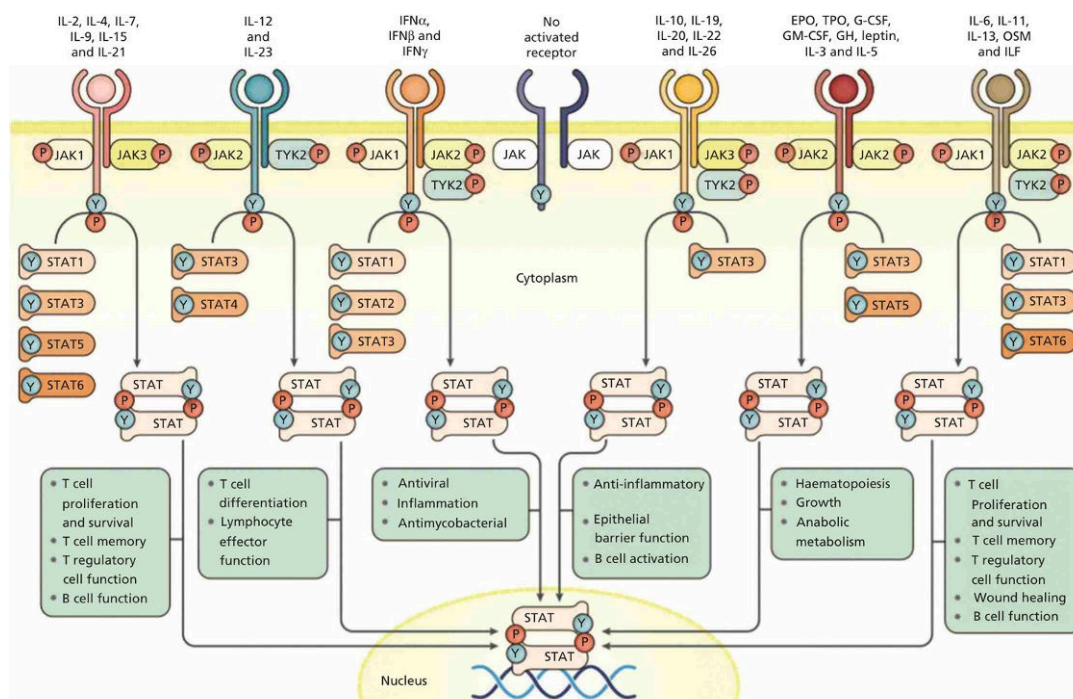
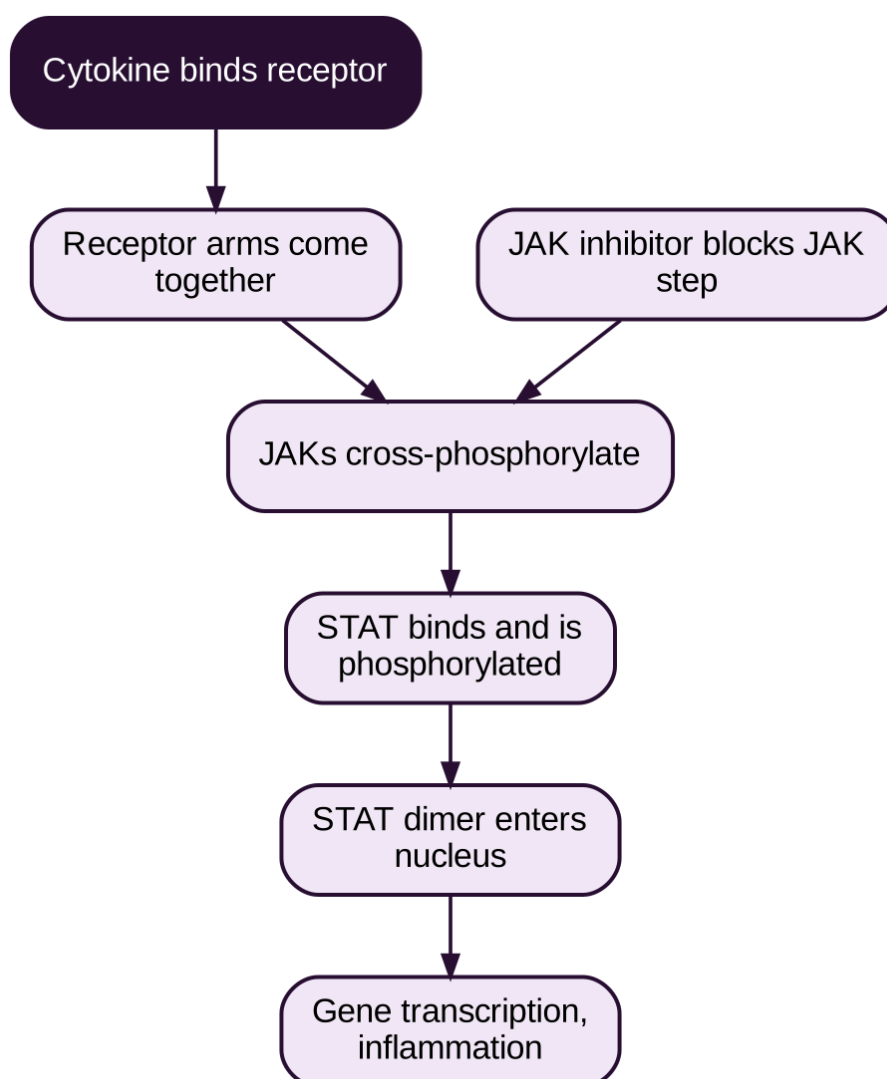


Figure 19.3 — Rook's Textbook of Dermatology 10e V1, p. 19.23. Extracellular signals involving the JAK/STAT pathway and their respective downstream functions. JAKs (Janus kinases) are intracellular tyrosine kinases

Flowchart 1: JAK/STAT signalling and its blockade



- Each cytokine receptor uses a specific JAK pair, as homodimers or heterodimers (e.g. JAK1/JAK2, JAK1/TYK2). The JAKs phosphorylate each other and the receptor tyrosines, which allows STAT binding and gene transcription.
- **Tofacitinib** selectively inhibits JAK1- and JAK3-dependent STAT activation. It blocks signalling by IFN- γ , IL-2, IL-4, IL-7, IL-15 and IL-21.
- **Atopic dermatitis:** IL-4, IL-13 and IL-31 all need JAK1, so JAK1 inhibition modulates them.
- **Psoriasis:** IL-23 signals through JAK1 and TYK2 (and IL-12 through TYK2), so TYK2 inhibition can modify this signal. Evidence for TYK2 inhibition is emerging.
- **Alopecia areata:**
 - IL-15 and IFN- γ activate CD8⁺ NKG2D⁺ T cells through JAK-STAT.
 - JAKi block this signalling in the T cells and the follicular epithelium, and hair regrows.
 - JAKi also disrupt Th17 and Th1/Th2 differentiation, and activate hair follicle stem cells.

Uses of tofacitinib in dermatology

- **Alopecia areata (oral):** 5 mg twice daily for up to 6 months.
- **Alopecia areata (topical):** 2% solution twice daily for 4 months was highly effective in eyelash AA. Efficacy is lower than with oral tofacitinib.
- **Atopic dermatitis (India):** topical tofacitinib is conditionally recommended for adults when conventional treatment is ineffective, inaccessible or causes adverse effects [w].
- **Outside dermatology:** psoriatic arthritis, rheumatoid arthritis and ulcerative colitis.

JAK inhibitors in alopecia areata

MANAGEMENT BASIS

IADVL — STAND AD consensus (atopic dermatitis; no AA-specific guideline in hand) — IADVL Textbook 5e, ch. 53; international — Rook's 10e, ch. 87 [w]

- **Baricitinib** (JAK1/2) is the first agent authorised, for severe AA in the USA in 2022.
 - Two phase III RCTs enrolled 1200 patients with SALT ≥ 50 .
 - At 36 weeks on 4 mg daily, 36–39% reached SALT ≤ 20 and 23% reached SALT ≤ 10 .
- **Tofacitinib:** in adults with $\geq 40\%$ scalp involvement, SALT fell by 81.9% in extensive patchy AA and by 59.0% in AT/AU. In adolescents, median SALT reduction was 93%.
- **Ruxolitinib** (JAK1/2): oral 20 mg twice daily for 6 months has been reported effective.
- **Side effects in AA** appear minor, with upper respiratory infection most common.
- **Relapse:** continued treatment is needed to maintain response in most patients. Long-term safety remains uncertain.
- **Place in treatment:** JAKi are off-label options for $>50\%$ scalp involvement in chronic disease.

JAK inhibitors in vitiligo

- Trials show promising effect of JAK inhibition in vitiligo.
- JAK inhibitors are not yet licensed for vitiligo in the pages and guidelines available here.
- Ahead of any JAKi use, screening and the contraindications under "Adverse effects" below apply.

JAK inhibitors in atopic dermatitis

MANAGEMENT BASIS

IADVL SIG Paediatric Dermatology — STAND AD consensus — [w]; EuroGuiDerm — Atopic eczema living update — 2025 [w2]

- **Licensed oral drugs:** abrocitinib, baricitinib and upadacitinib for moderate-to-severe AD in adults.
- **Efficacy:**
 - Baricitinib was superior to placebo for EASI75 (risk difference 0.16) at 16 weeks.

- Abrocitinib was as effective as dupilumab in a phase 3 study.
- **Evidence gap:** comparisons with other systemic agents are limited, and cardiovascular and venous embolic concerns may argue against use in at-risk populations.

Table 2: India vs international positioning in AD

India: IADVL STAND AD [W1]	International: EuroGuiDerm 2025 [W2]
Cyclosporine first-line; JAKi conditional options	JAKi listed with biologics as approved systemic options
JAKi not yet recommended in children	Baricitinib from age 2, abrocitinib from age 12

- **EMA/EuroGuiDerm restriction:** oral JAKi only in patients ≥65 years or with CV or cancer risk if no suitable alternative [W3].

JAK inhibitors in psoriasis: indications

- **Psoriasis:** no JAKi is licensed for skin psoriasis in Rook. TYK2-targeting agents show emerging evidence.
- **Psoriatic arthritis:** tofacitinib and upadacitinib are used.
- Biologics remain the Indian systemic route [W4].

JAK inhibitors: adverse effects

- **Common:** infections (herpes simplex, herpes zoster), nausea, vomiting, abdominal pain, acne, headache and raised creatine kinase.
- **Uncommon:** hyperlipidaemia and venous thrombosis (PE, DVT).
- **Drug-specific:**
 - Thrombocytopenia: abrocitinib.
 - Thrombocytosis: baricitinib.
 - Anaemia: upadacitinib.
 - Neutropenia, diverticulitis and transaminitis: baricitinib and upadacitinib.
- **Class warning:** the FDA warns of increased risk of serious heart events, cancer, blood clots and death. This followed a tofacitinib safety trial in rheumatoid arthritis.
- **Zoster:** tofacitinib carries a higher zoster risk, so zoster vaccination before starting is advised where possible [W5].
- **Contraindications:** hypersensitivity, active TB or serious infection, severe hepatic impairment, pregnancy and breastfeeding (abrocitinib and upadacitinib). Baricitinib is contraindicated in hypersensitivity and pregnancy.
- **Do not start if:** platelets $<150 \times 10^3/\text{mm}^3$, neutrophils $<1 \times 10^3/\text{mm}^3$, haemoglobin $<8 \text{ g/dL}$ or lymphocytes $<0.5 \times 10^3/\text{mm}^3$.
- **Vaccines:** avoid live vaccines within 2 weeks of starting, during treatment and for 6 months after stopping.
- **Pre-treatment screening:** FBC with differential, renal and liver function, lipids, and latent TB, hepatitis B and C and HIV.
- **Monitoring:**
 - Abrocitinib needs 4-weekly blood counts and lipids. Baricitinib needs 12-weekly lipids.
 - Rook suggests blood count, renal and hepatic function at 4 weeks, 4 months, then 4–6 monthly for all three.

Upadacitinib

- **Mechanism:** oral selective **JAK1** inhibitor.
- **Pharmacokinetics:**
 - Bioavailability is 60–79%, with peak levels in 1–4 h.
 - It is metabolised mainly by CYP3A4 and excreted in urine and faeces.
 - Half-life is 8–14 h.
- **Dose:** 15 or 30 mg once daily as tablets.
 - Use the lower dose if creatinine clearance is 30–60 mL/min or age is >65 years.
- **Indications:**



CLINICA
PUBLICATION

STRATUM CORNEUM

EPIDERMIS

DERMIS

HYPODERMIS

DNB Dermatology, Venereology & Leprosy

Solved Previous-Year Questions

VOLUME 2

Clinical Dermatology

- Part 7 · Papulosquamous and Eczematous Disorders
- Part 8 · Urticaria, Drug Reactions and Mastocytosis
- Part 9 · Bullous and Connective Tissue Diseases
- Part 10 · Vasculitis, Neutrophilic, Granulomatous and Vascular Disorders
- Part 11 · Pigmentary, Hair, Nail and Adnexal Disorders
- Part 12 · Infections and Infestations
- Part 13 · Genetic, Metabolic and Nutritional Disorders; Skin in Systemic Disease
- Part 14 · Paediatric, Pregnancy, Erythroderma and Photodermatoses
- Part 15 · Cutaneous Oncology
- Part 16 · Psychodermatology, Environmental and Miscellaneous Dermatoses

REVIEWED BY

Dr Soumik Dey
DNB (Dermatology)

Dr Sirsa Ghosh
DNB (Dermatology)

Based on Rook's Textbook of Dermatology (10th ed.), IADVL Textbook of Dermatology (5th ed.),
IAL Textbook of Leprosy (3rd ed.) and current Indian & international guidelines

UPDATED TILL OCTOBER 2026

Order at www.clinicapublication.com

Index

HOW TO USE

Each answer groups every paper and year in which the question was asked (badge = how often) and answers exactly the parts asked, sized to the marks. The amber Management-basis strip names the guideline used (Indian programme or IADVL first, then international). [W#] tags mark guideline/web facts, listed in the Web-Sourced Facts Register. Book figures are reproduced with their book, volume and page; diagram questions also carry a schematic you can draw in the exam. Each answer ends with its Sources (book, chapter and pages).

A • Parts

Part 1 — Structure and Function of the Skin and Appendages	Volume 1	—
Part 2 — Immunology, Physiology, Photobiology and Genetics	Volume 1	—
Part 3 — Dermatopathology and Diagnostic Techniques	Volume 1	—
Part 4 — Topical and Systemic Therapeutics	Volume 1	—
Part 5 — Biologics, Small Molecules, Newer Drugs and Phototherapy	Volume 1	—
Part 6 — Severity Scores, Research Methods and Emergency Care	Volume 1	—
Part 7 — Papulosquamous and Eczematous Disorders	21 answers	14
Part 8 — Urticaria, Drug Reactions and Mastocytosis	16 answers	59
Part 9 — Bullous and Connective Tissue Diseases	22 answers	101
Part 10 — Vasculitis, Neutrophilic, Granulomatous and Vascular Disorders	24 answers	156
Part 11 — Pigmentary, Hair, Nail and Adnexal Disorders	32 answers	219
Part 12 — Infections and Infestations	30 answers	297
Part 13 — Genetic, Metabolic and Nutritional Disorders; Skin in Systemic Disease	26 answers	372
Part 14 — Paediatric, Pregnancy, Erythroderma and Photodermatoses	8 answers	446
Part 15 — Cutaneous Oncology	18 answers	469
Part 16 — Psychodermatology, Environmental and Miscellaneous Dermatoses	13 answers	509
Part 17 — Genital Anatomy and Sexually Transmitted Infections	Volume 3	—
Part 18 — Syndromic Management, HIV and STI Programmes	Volume 3	—
Part 19 — Leprosy: Basic Science, Classification and Diagnosis	Volume 3	—
Part 20 — Leprosy: Reactions, Neuritis, Treatment, Disability and Programme	Volume 3	—
Part 21 — Dermatosurgery and Lasers	Volume 3	—
Part 22 — Aesthetic Dermatology	Volume 3	—
Part 23 — Recent Advances: AI, Tele dermatology and COVID-19	Volume 3	—

B • This volume, question by question

131. Psoriatic arthritis: classification (CASPAR) & management	3×	14
Asked in: Dec 2021 · Paper II · Q8(c) (3 marks) · Jun 2022 · Paper II · Q8(a) (4 marks) · Apr 2023 · Paper II · Q1(b) (6 marks)		
132. Biologics in psoriasis	2×	18
Asked in: Dec 2020 · Paper II · Q9(a) (4 marks) · Oct 2024 · Paper IV · Q2(b) (5 marks)		
133. Erythrodermic psoriasis (incl. children)	2×	20
Asked in: Dec 2020 · Paper II · Q6(b) (6 marks) · Dec 2021 · Paper II · Q8(a) (4 marks)		
134. Childhood psoriasis	1×	22
Asked in: Dec 2021 · Paper II · Q8(b) (3 marks)		
135. Severity scoring in psoriasis (nail, pustular, PsA)	1×	23
Asked in: Dec 2022 · Paper II · Q2 (10 marks)		
136. Pustular psoriasis	1×	26
Asked in: Apr 2023 · Paper II · Q1(a) (4 marks)		

137. Psoriasis: molecular etiopathogenesis	1×	28
Asked in: Oct 2024 · Paper IV · Q2(a) (5 marks)		
138. Keratosis lichenoides chronica	1×	31
Asked in: Dec 2025 · Paper II · Q2(b) (4 marks)		
139. Lichenoid drug eruptions	1×	32
Asked in: Dec 2025 · Paper II · Q2(a) (6 marks)		
140. Dupilumab & biologics in atopic dermatitis	3×	34
Asked in: Jun 2020 · Paper IV · Q8(b) (4 marks) · Oct 2023 · Paper II · Q3(a) (5 marks) · May 2024 · Paper II · Q4(b) (2 marks)		
141. Atopic dermatitis: diagnostic criteria & severity scoring	2×	37
Asked in: Jun 2022 · Paper II · Q2(a) (4 marks) · Oct 2024 · Paper IV · Q3(a) (5 marks)		
142. Atopic dermatitis: management (severe/childhood, pruritus)	2×	39
Asked in: Jun 2022 · Paper II · Q2(b) (6 marks) · Oct 2024 · Paper IV · Q3(b) (5 marks)		
143. Atopic dermatitis: aetiology	1×	41
Asked in: Dec 2022 · Paper II · Q9(b) (2 marks)		
144. Patch test & photopatch test	4×	43
Asked in: Dec 2020 · Paper I · Q10(b) (3 marks) · Jun 2021 · Paper II · Q1(b) (5 marks) · Dec 2022 · Paper II · Q6(a) (3 marks) · Jun 2025 · Paper I · Q8(b) (5 marks)		
145. Systemic contact dermatitis	2×	45
Asked in: Jun 2021 · Paper II · Q1(a) (5 marks) · Oct 2023 · Paper II · Q3(b) (5 marks)		
146. Occupational & footwear contact dermatitis	2×	47
Asked in: Jun 2022 · Paper II · Q9(a) (5 marks) · Apr 2023 · Paper II · Q8(a) (5 marks)		
147. Allergic contact dermatitis: pathophysiology	1×	48
Asked in: Jun 2020 · Paper I · Q5(a) (5 marks)		
148. Non-eczematous forms of allergic contact dermatitis	1×	50
Asked in: Jun 2020 · Paper I · Q5(b) (5 marks)		
149. Allergic vs irritant contact dermatitis	1×	52
Asked in: Jun 2025 · Paper I · Q8(a) (5 marks)		
150. Red face	1×	54
Asked in: Jun 2022 · Paper II · Q10(a) (3 marks)		
151. Perioral, periorbital & scrotal dermatitis	1×	55
Asked in: Jun 2025 · Paper II · Q3 (10 marks)		
152. Angioedema without wheals: hereditary & drug-induced	3×	59
Asked in: Jun 2020 · Paper II · Q6 (10 marks) · Jun 2022 · Paper II · Q8(b) (3 marks) · Jun 2025 · Paper II · Q9(a) (4 marks)		
153. Chronic spontaneous urticaria: investigations & management	3×	63
Asked in: Jun 2021 · Paper II · Q3(b) (5 marks) · Oct 2024 · Paper II · Q3(c) (4 marks) · Dec 2025 · Paper II · Q3(a) (4 marks)		
154. Physical / inducible urticarias (incl. cholinergic)	3×	66
Asked in: Jun 2021 · Paper II · Q3(a) (5 marks) · Oct 2024 · Paper II · Q3(b) (3 marks) · Dec 2025 · Paper II · Q3(c) (3 marks)		
155. Urticaria: classification	1×	69
Asked in: Oct 2024 · Paper II · Q3(a) (3 marks)		
156. Autologous serum therapy	1×	70
Asked in: Jun 2025 · Paper IV · Q8(b) (5 marks)		
157. Urticaria in pregnancy	1×	72
Asked in: Dec 2025 · Paper II · Q3(b) (3 marks)		
158. Mastocytosis: classification & management	3×	73
Asked in: Jun 2021 · Paper II · Q10 (10 marks) · Jun 2022 · Paper II · Q8(c) (3 marks) · Oct 2024 · Paper I · Q10(b) (3 marks)		
159. Telangiectasia macularis eruptiva perstans	1×	78
Asked in: Oct 2024 · Paper I · Q10(c) (4 marks)		

160. DRESS: diagnosis (RegiSCAR) & management	6×	80
Asked in: Jun 2020 · Paper IV · Q6(b) (5 marks) · Jun 2021 · Paper II · Q2(b) (7 marks) · Dec 2021 · Paper II · Q10(c) (3 marks) · Oct 2023 · Paper III · Q8(b) (5 marks) · Oct 2024 · Paper II · Q6(a) (5 marks) · Oct 2024 · Paper II · Q6(b) (5 marks)		
161. SJS/TEN: management & SCORTEN	3×	86
Asked in: Dec 2020 · Paper II · Q4(b) (5 marks) · Dec 2021 · Paper II · Q10(a) (4 marks) · Apr 2023 · Paper IV · Q7(b) (6 marks)		
162. Mechanisms of drug reactions & role of HLA	2×	90
Asked in: Jun 2020 · Paper IV · Q6(a) (5 marks) · Dec 2022 · Paper I · Q8(a) (5 marks)		
163. SCARs: definition & classification	2×	93
Asked in: Jun 2021 · Paper II · Q2(a) (3 marks) · Apr 2023 · Paper IV · Q7(a) (4 marks)		
164. Drug-induced autoimmune bullous diseases	2×	95
Asked in: May 2024 · Paper IV · Q9(b) (4 marks) · Jun 2025 · Paper II · Q9(b) (6 marks)		
165. Drug provocation test	1×	97
Asked in: Dec 2020 · Paper II · Q4(a) (5 marks)		
166. SDRIFE	1×	98
Asked in: Dec 2021 · Paper II · Q10(b) (3 marks)		
167. Cutaneous adverse effects of targeted anticancer therapy	1×	99
Asked in: May 2024 · Paper II · Q5(b) (5 marks)		
168. Pemphigus & AIBDs: target antigens & immunofluorescence	4×	101
Asked in: Jun 2022 · Paper IV · Q5(c) (3 marks) · Dec 2022 · Paper II · Q8(b) (3 marks) · Apr 2023 · Paper II · Q3(a) (5 marks) · Oct 2024 · Paper I · Q6(b) (4 marks)		
169. Bullous pemphigoid & its variants	3×	104
Asked in: Jun 2020 · Paper IV · Q9(c) (3 marks) · Dec 2021 · Paper II · Q1 (10 marks) · Oct 2023 · Paper IV · Q8(a) (5 marks)		
170. Paraneoplastic pemphigus	2×	108
Asked in: Oct 2023 · Paper II · Q1(b) (5 marks) · May 2024 · Paper IV · Q9(c) (3 marks)		
171. Subcorneal split disorders: differential diagnosis	1×	110
Asked in: Dec 2020 · Paper I · Q1(b) (5 marks)		
172. Dermatitis herpetiformis	1×	112
Asked in: Dec 2022 · Paper II · Q8(c) (3 marks)		
173. Epidermolysis bullosa acquisita	1×	112
Asked in: May 2024 · Paper I · Q7(b) (3 marks)		
174. Epidermolysis bullosa: pathogenesis & classification	2×	113
Asked in: Jun 2020 · Paper I · Q1(b) (5 marks) · Jun 2022 · Paper II · Q4(a) (3 marks)		
175. Immunofluorescence mapping in EB	1×	116
Asked in: Jun 2022 · Paper II · Q4(b) (3 marks)		
176. Kindler syndrome	1×	117
Asked in: Jun 2022 · Paper II · Q4(c) (4 marks)		
177. Long-term complications of EB	1×	119
Asked in: Dec 2022 · Paper II · Q8(a) (4 marks)		
178. Epidermolysis bullosa pruriginosa	1×	121
Asked in: May 2024 · Paper II · Q2(b) (5 marks)		
179. Autoantibodies & ANA in connective tissue diseases	5×	123
Asked in: Dec 2021 · Paper I · Q3(a) (4 marks) · Dec 2021 · Paper I · Q3(c) (3 marks) · Dec 2022 · Paper II · Q10(b) (3 marks) · Oct 2023 · Paper I · Q7(a) (5 marks) · Oct 2024 · Paper IV · Q5 (10 marks)		
180. Antiphospholipid syndrome / antibody	3×	128
Asked in: Jun 2020 · Paper IV · Q5(a) (5 marks) · Jun 2021 · Paper II · Q7(a) (5 marks) · Dec 2021 · Paper I · Q3(b) (3 marks)		
181. Dermatomyositis (incl. amyopathic DM)	3×	131
Asked in: Dec 2020 · Paper II · Q2(a) (4 marks) · Dec 2020 · Paper II · Q2(b) (6 marks) · Dec 2022 · Paper II · Q10(a) (3 marks)		

182. Systemic lupus erythematosus: diagnosis, criteria & treatment	2×	134
Asked in: Jun 2022 · Paper II · Q3 (10 marks) · May 2024 · Paper II · Q10(c) (3 marks)		
183. Calcinosis cutis	2×	138
Asked in: May 2024 · Paper IV · Q8(a) (3 marks) · Dec 2025 · Paper II · Q10(b) (5 marks)		
184. Raynaud's phenomenon & nailfold capillaroscopy	2×	140
Asked in: May 2024 · Paper IV · Q8(b) (3 marks) · Oct 2024 · Paper I · Q7(a) (6 marks)		
185. Systemic sclerosis: diagnosis, investigations, mRSS & management	3×	143
Asked in: Jun 2022 · Paper II · Q10(b) (4 marks) · Dec 2022 · Paper II · Q9(a) (2 marks) · Apr 2023 · Paper II · Q4 (10 marks)		
186. Eosinophilic fasciitis	1×	149
Asked in: Dec 2021 · Paper II · Q9(c) (3 marks)		
187. Scleredema	1×	151
Asked in: Dec 2021 · Paper II · Q9(b) (3 marks)		
188. Scleromyxedema	1×	152
Asked in: Jun 2022 · Paper II · Q6(c) (4 marks)		
189. Histopathology of scleroderma & scleromyxedema	1×	153
Asked in: Oct 2023 · Paper II · Q1(a) (5 marks)		
190. ANCA-associated vasculitis (incl. polyangiitis)	3×	156
Asked in: Jun 2021 · Paper IV · Q6 (10 marks) · Dec 2021 · Paper II · Q7(c) (3 marks) · Oct 2023 · Paper IV · Q5 (10 marks)		
191. Classification of vasculitis (Chapel Hill) & approach	3×	159
Asked in: Dec 2020 · Paper II · Q5(a) (5 marks) · Oct 2024 · Paper II · Q1(a) (3 marks) · Oct 2024 · Paper II · Q1(b) (7 marks)		
192. IgA vasculitis (Henoch–Schönlein purpura)	2×	164
Asked in: Dec 2021 · Paper II · Q7(b) (4 marks) · May 2024 · Paper II · Q4(a) (4 marks)		
193. Kawasaki disease	1×	166
Asked in: Dec 2021 · Paper II · Q2(c) (4 marks)		
194. Urticarial vasculitis	1×	167
Asked in: Dec 2021 · Paper II · Q7(a) (3 marks)		
195. Pigmented purpuric dermatosis (lichen aureus)	1×	169
Asked in: Jun 2025 · Paper II · Q10(c) (3 marks)		
196. Vasculitis vs vasculopathy; livedoid vasculopathy	2×	170
Asked in: Jun 2025 · Paper II · Q10(a) (3 marks) · Jun 2025 · Paper II · Q10(b) (4 marks)		
197. Autoinflammatory disorders of skin	3×	172
Asked in: Dec 2021 · Paper II · Q2(a) (3 marks) · May 2024 · Paper II · Q6(a) (5 marks) · May 2024 · Paper II · Q6(b) (3 marks)		
198. Schnitzler syndrome	1×	176
Asked in: Dec 2021 · Paper II · Q2(b) (3 marks)		
199. Sweet syndrome & pyoderma gangrenosum: variants & associations	1×	177
Asked in: Jun 2025 · Paper II · Q8 (10 marks)		
200. Behçet's disease: mucocutaneous manifestations	1×	182
Asked in: Dec 2025 · Paper II · Q4 (10 marks)		
201. Neutrophilic eccrine hidradenitis	1×	185
Asked in: Dec 2025 · Paper II · Q5(b) (5 marks)		
202. SAPHO syndrome	1×	187
Asked in: Dec 2025 · Paper II · Q5(a) (5 marks)		
203. Palisading granulomas	3×	188
Asked in: Dec 2020 · Paper I · Q4(b) (4 marks) · Dec 2020 · Paper II · Q1(a) (4 marks) · Dec 2025 · Paper I · Q6 (10 marks)		
204. Orofacial granulomatosis	2×	192
Asked in: Oct 2023 · Paper II · Q6(a) (5 marks) · Dec 2025 · Paper II · Q7 (10 marks)		
205. Sarcoidosis	2×	197
Asked in: Oct 2023 · Paper II · Q4(a) (5 marks) · May 2024 · Paper II · Q10(a) (3 marks)		

206. Granuloma annulare	1×	199
Asked in: Dec 2020 · Paper II · Q1(b) (6 marks)		
207. Histopathology of non-infectious granulomas	1×	203
Asked in: Apr 2023 · Paper I · Q10(a) (4 marks)		
208. Lipodermatosclerosis	2×	205
Asked in: Jun 2022 · Paper II · Q6(b) (3 marks) · Oct 2024 · Paper II · Q9(b) (5 marks)		
209. Lupus panniculitis (lupus profundus)	2×	208
Asked in: Oct 2023 · Paper II · Q7(b) (5 marks) · Dec 2025 · Paper II · Q9(b) (5 marks)		
210. Panniculitis: classification	1×	210
Asked in: Oct 2024 · Paper II · Q9(a) (5 marks)		
211. Chronic leg ulcer: investigation	1×	212
Asked in: Dec 2020 · Paper II · Q5(b) (5 marks)		
212. Chronic venous disease: evaluation, CEAP, sclerotherapy & compression	1×	213
Asked in: Dec 2022 · Paper IV · Q3 (10 marks)		
213. Anatomy & physiology of lower-limb venous system	1×	217
Asked in: Apr 2023 · Paper I · Q2(a) (5 marks)		
214. Vitiligo surgery (NCES, NCMT, acrofacial)	4×	219
Asked in: Dec 2021 · Paper IV · Q6(b) (3 marks) · Jun 2022 · Paper IV · Q4(a) (4 marks) · Dec 2022 · Paper IV · Q1(a) (5 marks) · May 2024 · Paper II · Q7(a) (3 marks)		
215. Vitiligo: psychosocial impact & counselling	3×	222
Asked in: Dec 2020 · Paper II · Q10(a) (6 marks) · Dec 2020 · Paper II · Q10(b) (4 marks) · Dec 2021 · Paper IV · Q8(c) (3 marks)		
216. Phototherapy in vitiligo (NB-UVB, home phototherapy)	2×	226
Asked in: Jun 2022 · Paper II · Q7(a) (3 marks) · Apr 2023 · Paper II · Q9(a) (6 marks)		
217. Vitiligo: newer & topical biologic treatments	1×	230
Asked in: Jun 2022 · Paper IV · Q2(c) (3 marks)		
218. Vitiligo: aetiopathogenesis	1×	230
Asked in: Dec 2022 · Paper II · Q9(d) (2 marks)		
219. Hypopigmented lesions in infancy: differential diagnosis	1×	231
Asked in: May 2024 · Paper I · Q8(b) (4 marks)		
220. Melasma: aetiology, management & recent advances	5×	233
Asked in: Jun 2020 · Paper IV · Q5(b) (5 marks) · Dec 2020 · Paper II · Q3(b) (6 marks) · Jun 2022 · Paper IV · Q8(b) (3 marks) · Dec 2022 · Paper II · Q9(c) (2 marks) · May 2024 · Paper II · Q7(b) (3 marks)		
221. Facial hyperpigmentation: differential diagnosis	1×	236
Asked in: Dec 2020 · Paper II · Q3(a) (4 marks)		
222. Frictional melanosis & maturational hyperpigmentation	2×	239
Asked in: Jun 2025 · Paper II · Q2(b) (2 marks) · Jun 2025 · Paper II · Q2(c) (2 marks)		
223. Skin lightening agents	1×	241
Asked in: Dec 2025 · Paper IV · Q8(a) (5 marks)		
224. Primary cicatricial alopecias: classification, LPP & FFA	3×	242
Asked in: Jun 2020 · Paper II · Q9(a) (5 marks) · Jun 2020 · Paper II · Q9(b) (5 marks) · Dec 2025 · Paper II · Q9(a) (5 marks)		
225. Hair evaluation: hair pull test, trichogram	2×	245
Asked in: Dec 2020 · Paper I · Q10(c) (3 marks) · Jun 2022 · Paper IV · Q3(c) (4 marks)		
226. Androgenetic alopecia & female pattern hair loss	2×	246
Asked in: Dec 2022 · Paper IV · Q5(b) (4 marks) · Apr 2023 · Paper IV · Q6(b) (6 marks)		
227. Scalp biopsy in alopecias	2×	249
Asked in: Apr 2023 · Paper I · Q1(b) (5 marks) · Oct 2024 · Paper I · Q4(b) (5 marks)		
228. Alopecia areata	1×	250
Asked in: Jun 2020 · Paper II · Q3(c) (2 marks)		

229. Telogen effluvium (post-COVID) & diffuse hair loss Asked in: Dec 2021 · Paper II · Q6 (10 marks)	1×	251
230. Hair shaft disorders: enumeration Asked in: May 2024 · Paper I · Q9(c) (4 marks)	1×	255
231. Non-scarring alopecias: classification Asked in: May 2024 · Paper I · Q9(a) (2 marks)	1×	257
232. Nail psoriasis & nail lichen planus Asked in: Jun 2021 · Paper I · Q5(a) (6 marks) · Jun 2025 · Paper I · Q4(c) (4 marks)	2×	257
233. Ingrown toenail (onychocryptosis): surgical management Asked in: Jun 2022 · Paper IV · Q9(c) (5 marks) · Oct 2024 · Paper IV · Q7(a) (5 marks)	2×	261
234. Nail signs: matrix changes & Beau's lines Asked in: Dec 2021 · Paper I · Q2(b) (4 marks) · Dec 2021 · Paper I · Q2(c) (3 marks)	2×	263
235. Injection therapy in nail disorders Asked in: Apr 2023 · Paper II · Q5(b) (6 marks)	1×	266
236. Onycholysis Asked in: Apr 2023 · Paper II · Q5(a) (4 marks)	1×	268
237. Drug-induced nail changes Asked in: Dec 2025 · Paper IV · Q9(b) (5 marks)	1×	270
238. Hormonal acne, hormonal therapy & PCOS Asked in: Jun 2020 · Paper IV · Q2(b) (5 marks) · Dec 2022 · Paper II · Q7(b) (5 marks) · Jun 2025 · Paper II · Q4(b) (5 marks)	3×	272
239. Acne-related syndromes Asked in: Jun 2020 · Paper IV · Q2(a) (5 marks)	1×	275
240. Newer topical therapies for acne Asked in: Jun 2021 · Paper IV · Q1(c) (4 marks)	1×	277
241. Rosacea: pathogenesis & histopathology Asked in: Dec 2020 · Paper I · Q4(c) (2 marks) · Jun 2021 · Paper II · Q5(a) (5 marks) · May 2024 · Paper II · Q2(a) (5 marks)	3×	279
242. Rosacea: subtypes & treatment Asked in: Jun 2021 · Paper II · Q5(b) (5 marks) · Oct 2023 · Paper II · Q9 (10 marks) · Dec 2025 · Paper II · Q10(a) (5 marks)	3×	282
243. Hidradenitis suppurativa: pathogenesis, staging, complications & management Asked in: Dec 2020 · Paper IV · Q1(a) (5 marks) · Jun 2022 · Paper II · Q5 (10 marks) · Dec 2022 · Paper IV · Q2 (10 marks) · Apr 2023 · Paper II · Q7 (10 marks) · Jun 2025 · Paper II · Q6 (10 marks)	5×	287
244. Follicular occlusion tetrad Asked in: May 2024 · Paper II · Q6(c) (2 marks)	1×	292
245. Hirsutism & hyperandrogenism: causes, evaluation & treatment Asked in: Jun 2021 · Paper II · Q9(a) (3 marks) · Jun 2021 · Paper II · Q9(b) (7 marks) · Dec 2022 · Paper II · Q7(a) (5 marks) · Apr 2023 · Paper II · Q2 (10 marks) · Dec 2025 · Paper II · Q6(a) (6 marks)	5×	292
246. Staphylococcal infections, SSSS & MRSA Asked in: Jun 2021 · Paper IV · Q5(b) (5 marks) · Dec 2021 · Paper I · Q5(b) (3 marks) · Dec 2022 · Paper IV · Q4(b) (5 marks)	3×	297
247. Bacillary angiomatosis Asked in: Jun 2022 · Paper II · Q10(c) (3 marks)	1×	301
248. Lyme disease & erythema chronicum migrans Asked in: Oct 2024 · Paper II · Q2(a) (2 marks) · Oct 2024 · Paper II · Q2(b) (3 marks)	2×	302
249. Gram-negative folliculitis Asked in: Dec 2025 · Paper II · Q6(b) (4 marks)	1×	305
250. Tuberculids (incl. papulonecrotic tuberculid) Asked in: Dec 2021 · Paper II · Q3(a) (4 marks) · Oct 2023 · Paper II · Q8(b) (5 marks) · May 2024 · Paper II · Q1(a) (6 marks) · Dec 2025 · Paper II · Q8(b) (5 marks)	4×	306

251. Atypical mycobacteria (swimming pool granuloma, NTM)	2×	311
Asked in: Jun 2021 · Paper IV · Q5(a) (5 marks) · Oct 2023 · Paper II · Q10(a) (6 marks)		
252. MDR / XDR tuberculosis	2×	313
Asked in: Dec 2021 · Paper II · Q3(b) (3 marks) · Jun 2025 · Paper II · Q5(a) (4 marks)		
253. Cutaneous tuberculosis: <i>M. tuberculosis</i> & pathogenesis of the spectrum	2×	314
Asked in: Oct 2024 · Paper I · Q9(a) (4 marks) · Oct 2024 · Paper I · Q9(b) (6 marks)		
254. Mycetoma: classification, histopathology, diagnosis & management	3×	318
Asked in: Dec 2021 · Paper II · Q4(c) (4 marks) · Oct 2023 · Paper II · Q2(a) (5 marks) · May 2024 · Paper II · Q8 (10 marks)		
255. Recalcitrant dermatophytosis in India (incl. <i>T. indotineae</i>)	3×	322
Asked in: Dec 2021 · Paper II · Q4(a) (3 marks) · Jun 2022 · Paper II · Q9(b) (5 marks) · Dec 2025 · Paper IV · Q7(b) (5 marks)		
256. Antifungal drug resistance: mechanisms & testing	3×	325
Asked in: Dec 2022 · Paper I · Q8(b) (5 marks) · Apr 2023 · Paper I · Q7(b) (5 marks) · Oct 2024 · Paper I · Q6(a) (6 marks)		
257. Antifungal drugs (topical & systemic azoles, naftifine)	3×	328
Asked in: Dec 2022 · Paper IV · Q8(c) (4 marks) · Apr 2023 · Paper II · Q8(b) (5 marks) · Oct 2023 · Paper IV · Q2(b) (3 marks)		
258. Tinea capitis in children: treatment	1×	331
Asked in: Dec 2021 · Paper II · Q4(b) (3 marks)		
259. MALDI-TOF MS in dermatophytes	1×	332
Asked in: Dec 2022 · Paper I · Q3(c) (2 marks)		
260. Sporotrichoid lesions: differential diagnosis	1×	332
Asked in: Dec 2022 · Paper II · Q5(a) (5 marks)		
261. Onychomycosis & non-dermatophyte moulds	1×	334
Asked in: Apr 2023 · Paper I · Q6(b) (5 marks)		
262. Warts: periungual & recalcitrant warts	3×	337
Asked in: Jun 2020 · Paper II · Q3(a) (4 marks) · Apr 2023 · Paper I · Q2(b) (5 marks) · May 2024 · Paper IV · Q10(a) (3 marks)		
263. Chikungunya	2×	340
Asked in: Jun 2020 · Paper IV · Q3(b) (3 marks) · Apr 2023 · Paper IV · Q8(b) (5 marks)		
264. Molluscum contagiosum	2×	341
Asked in: Jun 2020 · Paper II · Q3(b) (4 marks) · Oct 2024 · Paper III · Q5(a) (4 marks)		
265. Herpes zoster: post-herpetic neuralgia & zoster vaccine	2×	344
Asked in: Dec 2020 · Paper IV · Q4(a) (5 marks) · Oct 2023 · Paper IV · Q2(a) (4 marks)		
266. Poxvirus infections (incl. mpox)	2×	346
Asked in: Apr 2023 · Paper I · Q6(a) (5 marks) · May 2024 · Paper II · Q1(b) (4 marks)		
267. Dengue	1×	347
Asked in: Jun 2020 · Paper IV · Q3(c) (3 marks)		
268. Eczema herpeticum	1×	349
Asked in: Jun 2020 · Paper II · Q7 (10 marks)		
269. Cutaneous manifestations of hepatitis B, hepatitis C & EBV	1×	352
Asked in: Dec 2022 · Paper II · Q4 (10 marks)		
270. Cutaneous leishmaniasis: classification, diagnosis & treatment	2×	355
Asked in: Jun 2020 · Paper II · Q1 (10 marks) · Jun 2025 · Paper II · Q5(b) (6 marks)		
271. Scabies (incl. nodular scabies)	2×	359
Asked in: Jun 2020 · Paper IV · Q9(b) (3 marks) · Jun 2022 · Paper II · Q1 (10 marks)		
272. Pediculosis: head lice & phthiriasis pubis	2×	362
Asked in: Dec 2020 · Paper III · Q2(a) (5 marks) · Apr 2023 · Paper II · Q10(c) (3 marks)		
273. Post kala-azar dermal leishmaniasis (PKDL)	2×	364
Asked in: Dec 2021 · Paper II · Q3(c) (3 marks) · Oct 2023 · Paper II · Q2(b) (5 marks)		

274. Cutaneous larva migrans & myiasis	2×	367
Asked in: Apr 2023 · Paper II · Q10(a) (4 marks) · Apr 2023 · Paper II · Q10(b) (3 marks)		
275. Demodex & skin	1×	370
Asked in: Dec 2025 · Paper IV · Q1(a) (5 marks)		
276. Ichthyoses: classification, diagnostic clues & syndromic ichthyosis	4×	372
Asked in: Jun 2022 · Paper IV · Q7(b) (5 marks) · Dec 2022 · Paper II · Q3(a) (5 marks) · Dec 2022 · Paper II · Q3(b) (2 marks) · Oct 2023 · Paper II · Q4(b) (5 marks)		
277. Collodion baby	2×	376
Asked in: Dec 2021 · Paper II · Q5(c) (3 marks) · Dec 2022 · Paper II · Q3(c) (3 marks)		
278. Xeroderma pigmentosum	2×	377
Asked in: Dec 2021 · Paper I · Q10(b) (4 marks) · May 2024 · Paper II · Q9(b) (5 marks)		
279. Genodermatoses with photosensitivity	1×	381
Asked in: Dec 2020 · Paper I · Q9(b) (6 marks)		
280. Silvery hair syndromes	1×	384
Asked in: Dec 2021 · Paper I · Q4(c) (3 marks)		
281. Tuberous sclerosis complex	1×	385
Asked in: Apr 2023 · Paper II · Q6(b) (5 marks)		
282. Ehlers–Danlos syndrome	1×	386
Asked in: Oct 2024 · Paper II · Q7 (10 marks)		
283. Premature ageing syndromes	1×	393
Asked in: Jun 2025 · Paper I · Q6(b) (5 marks)		
284. Heme biosynthesis pathway & classification of porphyrias	4×	395
Asked in: Jun 2021 · Paper II · Q6(a) (3 marks) · Dec 2022 · Paper I · Q9(a) (3 marks) · May 2024 · Paper II · Q3(b) (6 marks) · Oct 2024 · Paper I · Q3(a) (3 marks)		
285. Cutaneous porphyrias (incl. PCT): clinical & histopathology	3×	399
Asked in: Dec 2022 · Paper I · Q9(b) (3 marks) · Oct 2024 · Paper I · Q3(b) (3 marks) · Oct 2024 · Paper I · Q3(c) (4 marks)		
286. Erythropoietic protoporphyria	1×	402
Asked in: Jun 2021 · Paper II · Q6(b) (7 marks)		
287. Cutaneous amyloidosis	5×	406
Asked in: Jun 2020 · Paper IV · Q9(a) (4 marks) · Dec 2020 · Paper II · Q7(a) (5 marks) · Dec 2021 · Paper IV · Q2(c) (3 marks) · Jun 2022 · Paper II · Q6(a) (3 marks) · Dec 2022 · Paper II · Q10(c) (4 marks)		
288. Xanthomas & xanthelasma palpebrarum	3×	409
Asked in: Dec 2020 · Paper II · Q7(b) (5 marks) · Dec 2021 · Paper IV · Q1(b) (4 marks) · Dec 2021 · Paper IV · Q1(c) (2 marks)		
289. Alkaptonuria	1×	412
Asked in: Dec 2021 · Paper IV · Q2(b) (3 marks)		
290. Zinc deficiency	2×	413
Asked in: Jun 2020 · Paper IV · Q4(b) (5 marks) · Dec 2022 · Paper I · Q10(c) (3 marks)		
291. Vitamin deficiencies & skin (water-soluble, vitamin A)	2×	415
Asked in: Jun 2021 · Paper II · Q8(a) (4 marks) · Dec 2022 · Paper I · Q10(b) (3 marks)		
292. Pellagra	1×	417
Asked in: Jun 2021 · Paper II · Q8(b) (6 marks)		
293. Metabolic syndrome, obesity & acanthosis nigricans	4×	421
Asked in: Dec 2021 · Paper IV · Q1(a) (4 marks) · Dec 2022 · Paper II · Q9(e) (2 marks) · Oct 2023 · Paper IV · Q8(b) (5 marks) · Dec 2025 · Paper IV · Q5(a) (5 marks)		
294. Paraneoplastic dermatoses & cutaneous markers of internal malignancy	3×	423
Asked in: Jun 2021 · Paper IV · Q4 (10 marks) · May 2024 · Paper IV · Q8(c) (4 marks) · Oct 2024 · Paper IV · Q1(a) (6 marks)		
295. Hypereosinophilic syndrome	1×	428
Asked in: Jun 2020 · Paper II · Q8(b) (5 marks)		

296. Cutaneous manifestations of endocrine (adrenal) disorders Asked in: Jun 2021 · Paper IV · Q7 (10 marks)	1×	430
297. Graft-versus-host disease Asked in: Dec 2021 · Paper II · Q9(a) (4 marks)	1×	433
298. Kikuchi–Fujimoto disease Asked in: Oct 2023 · Paper II · Q10(b) (4 marks)	1×	435
299. Sacroiliitis in dermatology Asked in: Oct 2023 · Paper IV · Q3 (10 marks)	1×	437
300. Cutaneous manifestations of cardiac disease Asked in: Oct 2024 · Paper IV · Q1(b) (4 marks)	1×	442
301. Rheumatoid arthritis & accelerated rheumatoid nodulosis Asked in: Oct 2024 · Paper II · Q8 (10 marks)	1×	443
302. Neonatal vesiculopustular & transient neonatal dermatoses Asked in: Dec 2021 · Paper II · Q5(b) (4 marks) · Dec 2022 · Paper II · Q5(b) (5 marks) · Jun 2025 · Paper II · Q1(b) (5 marks)	3×	446
303. Physiological skin changes in the newborn Asked in: Oct 2023 · Paper II · Q8(a) (5 marks)	1×	451
304. Specific dermatoses of pregnancy Asked in: Jun 2020 · Paper IV · Q4(a) (5 marks) · Dec 2020 · Paper II · Q8 (10 marks) · Jun 2021 · Paper II · Q4(a) (3 marks) · May 2024 · Paper II · Q7(c) (4 marks)	4×	453
305. Impetigo herpetiformis (pustular psoriasis of pregnancy) Asked in: Jun 2020 · Paper II · Q4(a) (5 marks) · Jun 2022 · Paper IV · Q7(a) (5 marks)	2×	457
306. Pemphigoid gestationis Asked in: Jun 2020 · Paper II · Q4(b) (5 marks) · Jun 2021 · Paper II · Q4(b) (7 marks)	2×	459
307. Physiological skin changes in pregnancy Asked in: Dec 2022 · Paper I · Q9(c) (4 marks)	1×	461
308. Erythroderma in children Asked in: Dec 2020 · Paper II · Q6(a) (4 marks) · Apr 2023 · Paper IV · Q5 (10 marks)	2×	463
309. Idiopathic & photoaggravated photodermatoses Asked in: Dec 2022 · Paper II · Q6(b) (3 marks) · May 2024 · Paper II · Q10(b) (4 marks)	2×	467
310. Basal cell carcinoma: pathogenesis, hedgehog pathway, topical agents & vismodegib Asked in: Dec 2021 · Paper IV · Q3 (10 marks) · Oct 2023 · Paper IV · Q9(b) (5 marks)	2×	469
311. Non-melanoma skin cancer: risk factors, chemicals & genetic syndromes Asked in: Dec 2022 · Paper II · Q1 (10 marks) · Oct 2024 · Paper IV · Q4(a) (3 marks)	2×	474
312. Bowen's disease & actinic keratosis Asked in: May 2024 · Paper I · Q6(b) (4 marks) · Oct 2024 · Paper II · Q5(c) (5 marks)	2×	478
313. Extramammary Paget disease Asked in: Apr 2023 · Paper II · Q9(b) (4 marks)	1×	480
314. Seborrhoeic keratosis: histopathology Asked in: Apr 2023 · Paper I · Q5(a) (5 marks)	1×	482
315. Painful skin tumours Asked in: Jun 2022 · Paper IV · Q9(b) (3 marks)	1×	484
316. Cutaneous metastases Asked in: Apr 2023 · Paper IV · Q1(a) (5 marks)	1×	485
317. Targeted therapy & immunotherapy in skin cancers Asked in: Oct 2024 · Paper IV · Q4(b) (7 marks)	1×	487
318. CTCL: WHO-EORTC classification & variants Asked in: Jun 2020 · Paper II · Q10(c) (5 marks) · Jun 2022 · Paper IV · Q5(a) (4 marks)	2×	490
319. Pseudolymphoma Asked in: Jun 2020 · Paper II · Q10(a) (2 marks) · Jun 2020 · Paper II · Q10(b) (3 marks)	2×	492

320. CTCL: immunohistochemistry	1×	494
Asked in: Jun 2022 · Paper IV · Q5(b) (3 marks)		
321. Mycosis fungoides (incl. hypopigmented MF)	1×	496
Asked in: Apr 2023 · Paper IV · Q1(b) (5 marks)		
322. Langerhans cell histiocytosis & classification of histiocytoses	2×	497
Asked in: Dec 2021 · Paper IV · Q2(a) (4 marks) · May 2024 · Paper II · Q3(a) (4 marks)		
323. Immunohistochemical markers of histiocytoses	1×	499
Asked in: Apr 2023 · Paper I · Q5(b) (5 marks)		
324. Infantile haemangioma: management	4×	501
Asked in: Dec 2020 · Paper IV · Q1(b) (5 marks) · Jun 2022 · Paper IV · Q6(a) (3 marks) · May 2024 · Paper II · Q9(a) (5 marks) · Oct 2024 · Paper IV · Q8(b) (4 marks)		
325. Angiolymphoid hyperplasia with eosinophilia	1×	503
Asked in: Jun 2020 · Paper II · Q8(a) (5 marks)		
326. Pyogenic granuloma	1×	505
Asked in: Jun 2021 · Paper II · Q7(b) (5 marks)		
327. Vascular malformations: classification	1×	507
Asked in: Oct 2024 · Paper IV · Q8(a) (6 marks)		
328. Trichotillomania	3×	509
Asked in: Dec 2021 · Paper IV · Q8(a) (4 marks) · Jun 2022 · Paper II · Q7(b) (3 marks) · Oct 2024 · Paper II · Q4(b) (6 marks)		
329. Body dysmorphic disorder	2×	511
Asked in: Dec 2021 · Paper IV · Q8(b) (3 marks) · Jun 2022 · Paper II · Q7(c) (4 marks)		
330. Classification of psychodermatological disorders	1×	513
Asked in: Oct 2024 · Paper II · Q4(a) (4 marks)		
331. Delusional parasitosis	1×	515
Asked in: Dec 2025 · Paper II · Q8(a) (5 marks)		
332. Air pollution & skin	4×	518
Asked in: Jun 2020 · Paper IV · Q7(a) (4 marks) · Oct 2023 · Paper IV · Q9(a) (5 marks) · May 2024 · Paper I · Q10(c) (3 marks) · Dec 2025 · Paper IV · Q4 (10 marks)		
333. Chronic arsenic toxicity	2×	521
Asked in: Apr 2023 · Paper II · Q6(a) (5 marks) · Oct 2024 · Paper II · Q10 (10 marks)		
334. Dermatoses at high altitude & in desert areas	2×	525
Asked in: Jun 2020 · Paper IV · Q7(b) (3 marks) · Jun 2020 · Paper IV · Q7(c) (3 marks)		
335. IV drug abuse & nicotine: effects on skin	2×	528
Asked in: Jun 2025 · Paper II · Q7(a) (5 marks) · Jun 2025 · Paper II · Q7(b) (5 marks)		
336. Perforating disorders (incl. reactive perforating collagenosis)	2×	533
Asked in: Dec 2021 · Paper I · Q7(a) (6 marks) · May 2024 · Paper II · Q5(a) (5 marks)		
337. Diet & dermatology (incl. acne)	2×	536
Asked in: Jun 2022 · Paper IV · Q8(c) (3 marks) · Jun 2025 · Paper II · Q4(a) (5 marks)		
338. Annular & figurate erythemas	2×	538
Asked in: May 2024 · Paper II · Q4(c) (4 marks) · Oct 2024 · Paper II · Q2(c) (5 marks)		
339. Porokeratosis	1×	541
Asked in: Jun 2020 · Paper II · Q5 (10 marks)		
340. Gender & dermatology	1×	544
Asked in: Jun 2025 · Paper II · Q1(a) (5 marks)		

C · High-yield list (asked 3× or more)

DRESS: diagnosis (RegiSCAR) & management	6×	80
Autoantibodies & ANA in connective tissue diseases	5×	123
Melasma: aetiology, management & recent advances	5×	233

Hidradenitis suppurativa: pathogenesis, staging, complications & management	5×	287
Hirsutism & hyperandrogenism: causes, evaluation & treatment	5×	292
Cutaneous amyloidosis	5×	406
Patch test & photopatch test	4×	43
Pemphigus & AIBDs: target antigens & immunofluorescence	4×	101
Vitiligo surgery (NCES, NCMT, acrofacial)	4×	219
Tuberculids (incl. papulonecrotic tuberculid)	4×	306
Ichthyoses: classification, diagnostic clues & syndromic ichthyosis	4×	372
Heme biosynthesis pathway & classification of porphyrias	4×	395
Metabolic syndrome, obesity & acanthosis nigricans	4×	421
Specific dermatoses of pregnancy	4×	453
Infantile haemangioma: management	4×	501
Air pollution & skin	4×	518
Psoriatic arthritis: classification (CASPAR) & management	3×	14
Dupilumab & biologics in atopic dermatitis	3×	34
Angioedema without wheals: hereditary & drug-induced	3×	59
Chronic spontaneous urticaria: investigations & management	3×	63
Physical / inducible urticarias (incl. cholinergic)	3×	66
Mastocytosis: classification & management	3×	73
SJS/TEN: management & SCORTEN	3×	86
Bullous pemphigoid & its variants	3×	104
Antiphospholipid syndrome / antibody	3×	128
Dermatomyositis (incl. amyopathic DM)	3×	131
Systemic sclerosis: diagnosis, investigations, mRSS & management	3×	143
ANCA-associated vasculitis (incl. polyangiitis)	3×	156
Classification of vasculitis (Chapel Hill) & approach	3×	159
Autoinflammatory disorders of skin	3×	172
Palisading granulomas	3×	188
Vitiligo: psychosocial impact & counselling	3×	222
Primary cicatricial alopecias: classification, LPP & FFA	3×	242
Hormonal acne, hormonal therapy & PCOS	3×	272
Rosacea: pathogenesis & histopathology	3×	279
Rosacea: subtypes & treatment	3×	282
Staphylococcal infections, SSSS & MRSA	3×	297
Mycetoma: classification, histopathology, diagnosis & management	3×	318
Recalcitrant dermatophytosis in India (incl. T. indotineae)	3×	322
Antifungal drug resistance: mechanisms & testing	3×	325
Antifungal drugs (topical & systemic azoles, naftifine)	3×	328
Warts: periungual & recalcitrant warts	3×	337
Cutaneous porphyrias (incl. PCT): clinical & histopathology	3×	399
Xanthomas & xanthelasma palpebrarum	3×	409
Paraneoplastic dermatoses & cutaneous markers of internal malignancy	3×	423
Neonatal vesiculopustular & transient neonatal dermatoses	3×	446
Trichotillomania	3×	509

PART 7

Papulosquamous and Eczematous Disorders

131. Psoriatic arthritis: classification (CASPAR) & management

ASKED 3* · 6-mark scope

Asked in: Dec 2021 · Paper II · Q8(c) (3 marks) · Jun 2022 · Paper II · Q8(a) (4 marks) · Apr 2023 · Paper II · Q1(b) (6 marks)

Q: Classification of psoriatic arthritis and CASPAR criteria. [3]

Q: Psoriatic arthritis - management. [4]

Q: Management of psoriatic arthritis. [6]

Classification of psoriatic arthritis

- **Definition:** seronegative inflammatory arthritis, seen in up to 40% of patients with moderate-to-severe psoriasis.
- It is heterogeneous and traditionally classified by **Moll and Wright**. Subtypes overlap considerably, and several alternative schemes exist.
- **Asymmetric oligoarthritis** is the commonest pattern, followed by symmetric polyarthritis.
- **Distal interphalangeal (DIP)** predominance distinguishes it from rheumatoid arthritis.
- **Arthritis mutilans** is a rare, severe, deforming arthritis of the hands and feet. It is associated with enthesitis, dactylitis and more severe skin disease.
- Moll and Wright has five subgroups, which often overlap.

Table 1: Moll and Wright patterns of psoriatic arthritis

Pattern	Key features	Frequency (Indian text)
Asymmetric mono-/oligoarthritis	Few joints, asymmetrical; dactylitis ("sausage digit")	About 70%
Predominant DIP arthritis	Distinguishes it from rheumatoid arthritis; nails of the affected digits usually involved	About 16%
Symmetrical rheumatoid-like polyarthritis	PIP joints affected; rheumatoid factor usually negative	About 15%
Arthritis mutilans	Osteolysis, digital shortening and telescoping	About 5%
Axial (spondylitis/sacroiliitis)	Spine and sacroiliac joints involved	Spondylitis reported in 50%

- A simplified three-group scheme is also used: asymmetrical arthritis, symmetrical polyarthritis and predominant spondylitis.
- Dactylitis (sausage fingers), enthesitis (heel pain, plantar fasciitis) and nail changes (up to 80%) support the diagnosis.
- Early morning back stiffness may be the only feature of sacroiliitis or cervical spondylitis.

CASPAR criteria

Table 35.8 Classification of Psoriatic Arthritis (CASPAR) criteria for psoriatic arthritis. To be characterised as having psoriatic arthritis, a patient with inflammatory articular disease (joint, spine or enthesal) must have three or more points from five categories. Each category scores a maximum of 1 point, except category 1, which scores 2 points for current psoriasis.

Psoriasis	Current psoriasis	2
	Personal history of psoriasis	1
	Psoriasis in a first- or second-degree relative	1
Typical psoriatic nail involvement		1
A negative test for rheumatoid factor		1
Dactylitis	Current dactylitis	1
	History of dactylitis	1
Radiological evidence of juxta-articular new bone formation		1

Table 35.8 — Rook's Textbook of Dermatology 10e V2, p. 35.41. Classification of Psoriatic Arthritis (CASPAR) criteria for psoriatic arthritis.

- CASPAR (Classification Criteria for Psoriatic Arthritis) has **99% sensitivity and 91% specificity**.
- **Prerequisite:** inflammatory articular disease (joint, spine or entheses).
- **Diagnosis:** **≥3 points** from five categories.
- Each category scores a maximum of 1 point, except current psoriasis, which scores 2.

Table 2: CASPAR criteria and scores

Feature	Points
Current psoriasis	2
Personal history of psoriasis	1
Psoriasis in a first- or second-degree relative	1
Typical psoriatic nail involvement	1
Negative rheumatoid factor	1
Current dactylitis	1
History of dactylitis	1
Radiological juxta-articular new bone formation	1

- In the nail category, the Indian text lists onycholysis, pitting and hyperkeratosis.
- Rheumatoid factor testing should preferably use ELISA or nephelometry, not latex.
- The new-bone-formation criterion is assessed on plain radiographs of the hands or feet.
- Screening tools such as PASE, PEST and ToPAS help dermatologists decide on rheumatology referral.

Psoriatic arthritis – management

MANAGEMENT BASIS

Rook's 10e, ch. 35, which reproduces EULAR 2019 recommendations (Ann Rheum Dis 2020) as Figure 35.40.

General principles

- Managed by a **rheumatologist**. Combined dermatology–rheumatology clinics help in complex cases.
- Early treatment prevents irreversible joint damage.
- Options are narrower than in psoriasis and depend on the pattern of joint disease.
- Prefer a **single agent** effective for both skin and joints.

- Ask about joint symptoms at every psoriasis consultation.

Stepwise treatment

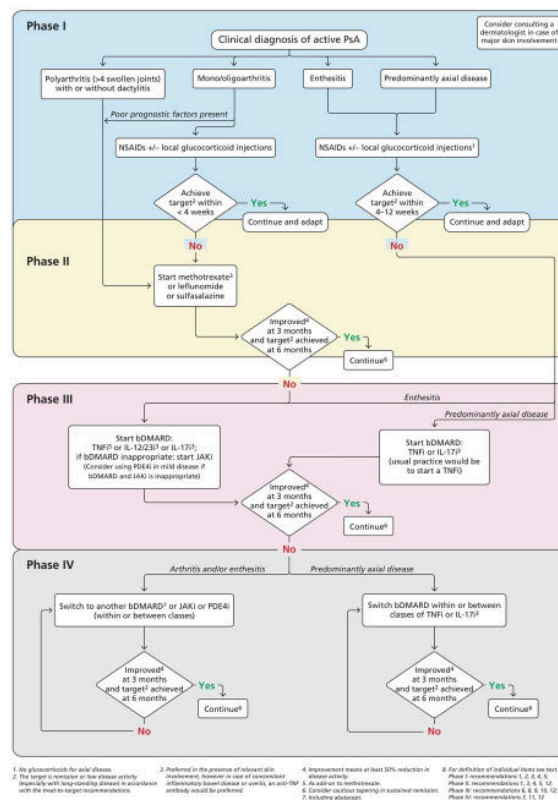
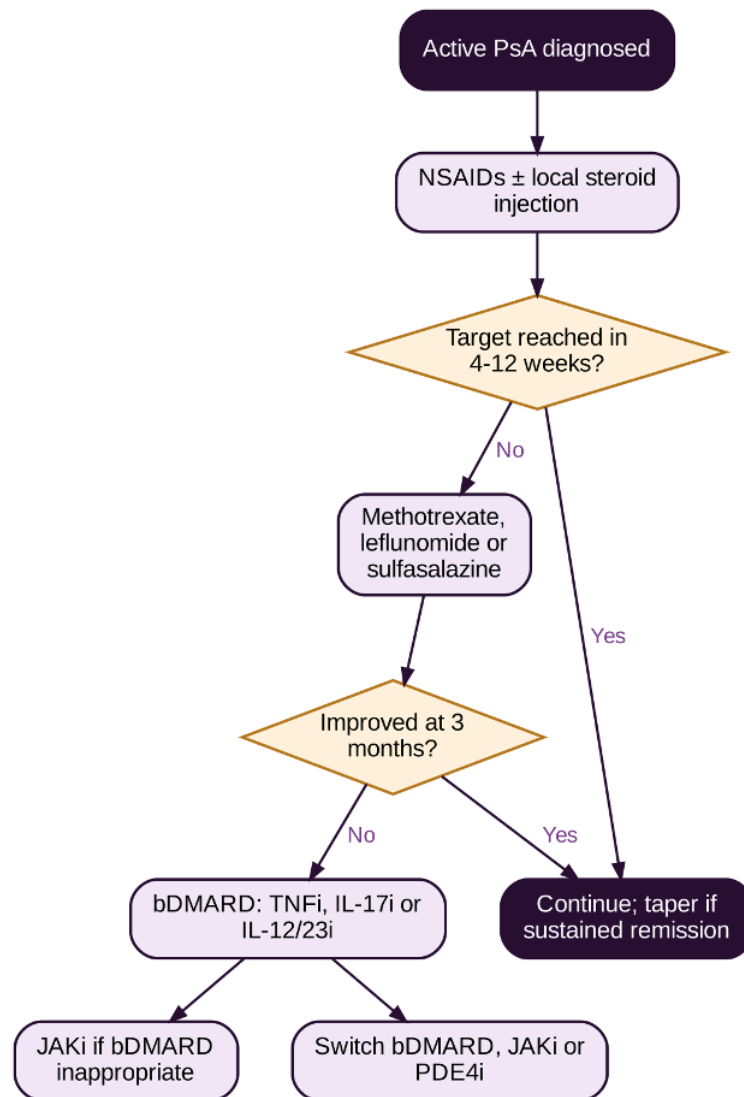


Figure 35.40 — Rook's Textbook of Dermatology 10e V2, p. 35.44. EULAR guidelines for

Flowchart 1: Stepwise management of psoriatic arthritis (EULAR 2019)



- Methotrexate is preferred when skin involvement is relevant. If there is concomitant inflammatory bowel disease or uveitis, an anti-TNF antibody is preferred.
- A TNFi is added to methotrexate when needed.
- **Predominantly axial disease:** NSAIDs, then a bDMARD (TNFi or IL-17i), with a TNFi usual. **No glucocorticoids for axial disease.**
- **Improvement** means $\geq 50\%$ reduction in disease activity. The **target** is remission or low disease activity.
- Consider cautious tapering in sustained remission.

Drugs

- **NSAIDs** are the mainstay for mild disease. Use the lowest dose for short-term control.
- **Oral corticosteroids** are used for acute flares. They can destabilise skin disease or precipitate GPP, so taper the dose slowly.
- **Intra-articular steroids** give symptomatic relief, with little evidence that they prevent bone remodelling or fibrosis.
- **Physiotherapy** may be beneficial.
- **Conventional agents:** methotrexate, sulphasalazine, leflunomide. **Apremilast** is also used.
- **Biologics:** TNFi, ustekinumab, IL-17 inhibitors and IL-23p19 inhibitors.
- Secukinumab was non-different from adalimumab 40 mg every 2 weeks on ACR-20 at 52 weeks (67% vs 62%).

Table 3: Biologics with licensed or trial doses in psoriatic arthritis

Drug	Dose	Note
Secukinumab	150 mg every 4 weeks (licensed for PsA)	Psoriasis dose is 300 mg every 4 weeks
Ixekizumab	80 mg SC every 2 or 4 weeks	Non-inferior to adalimumab
Bimekizumab	160 mg every 4 weeks	Effective after TNFi failure
Ustekinumab	45 or 90 mg at weeks 0, 4, 16, then every 12 weeks	Not effective in axial disease
Guselkumab	100 mg at weeks 0 and 4, then every 4 or 8 weeks	Effective in axial disease in biologic-naïve patients
Risankizumab	150 mg at weeks 0, 4 and 12	Licensed for PsA
Abatacept	125 mg SC weekly	Not effective for psoriasis

Response assessment

- **ACR-20, 50 and 70** denote 20%, 50% and 70% improvement in joint counts and global, pain, disability and acute-phase measures. ACR measures are required by licensing agencies.
- **PsARC, DAS** (uses ESR) and **BASDAI** (axial disease) are also used.
- Biologic treatment of psoriasis, especially with TNFi, may reduce the incidence of PsA.

PEARLS

- PsA is a seronegative inflammatory arthritis, occurring in up to 40% of moderate-to-severe psoriasis.
- Hallmarks are DIP involvement, dactylitis, nail disease and enthesitis.
- CASPAR needs inflammatory articular disease plus ≥ 3 points; current psoriasis alone scores 2.
- Asymmetric oligoarthritis is the commonest pattern.
- Ladder: NSAID, then csDMARD, then bDMARD (TNFi, IL-17i or IL-12/23i), then switch.
- Trap: abatacept does not work for skin psoriasis, and ustekinumab does not work in axial disease.
- Trap: oral steroids can destabilise skin disease or precipitate GPP, so taper them slowly.

Sources

- ROOK V2 — Ch 35 Psoriasis and Related Disorders — §Psoriatic arthritis — pp. 35.41–35.44, 35.46
- IADVL V1 — Ch 31 Psoriasis — §Psoriatic arthritis — pp. 1144–1146

132. Biologics in psoriasis

ASKED 2* · 5-mark scope

Asked in: Dec 2020 · Paper II · Q9(a) (4 marks) · Oct 2024 · Paper IV · Q2(b) (5 marks)

Q: Enumerate various biologics for the treatment of psoriasis. [4]

Q: Role of newer biologicals in management of psoriasis. [5]

Biologics available for psoriasis

Table 1: Biologics licensed for plaque psoriasis, by target

Target	Agents	Maintenance interval
TNF- α	Etanercept, infliximab, adalimumab, certolizumab pegol	Etanercept: 25 mg twice weekly or 50 mg once weekly. Adalimumab: 80 mg at week 0, then 40 mg every other week from week 1
IL-12/23 (p40)	Ustekinumab	45 mg if <100 kg, 90 mg if >100 kg, at weeks 0 and 4, then 12-weekly
IL-23 (p19)	Guselkumab, risankizumab, tildrakizumab	Guselkumab 8-weekly. Risankizumab and tildrakizumab 12-weekly
IL-17	Bimekizumab (IL-17A and IL-17F), brodalumab (IL-17 receptor), ixekizumab and secukinumab (IL-17A)	Brodalumab 2-weekly. Ixekizumab and secukinumab 4-weekly. Bimekizumab 8-weekly

- Secukinumab is licensed at 300 mg every 4 weeks for psoriasis.

- Mirikizumab is effective in psoriasis but is being developed for regulatory approval only in inflammatory bowel disease.
- Biologics used in India under the Indian consensus: etanercept, infliximab, adalimumab biosimilar and secukinumab [w1].

Role of newer biologicals

- **Place in therapy:** Newer IL-17 and IL-23 blockers are more effective than older agents in head-to-head trials.
 - Etanercept is less effective than ustekinumab, secukinumab, ixekizumab and tildrakizumab. Its first-line use has declined.
 - Adalimumab is less effective than risankizumab, bimekizumab and guselkumab.
 - Biosimilar adalimumab remains a reasonable first-line biologic because it is cheaper.
- **IL-23 antagonists:**
 - They block IL-23, which drives Th17, $\gamma\delta$ T and ILC3 cells to produce IL-17. This explains the marked efficacy.
 - Dosing is 8- to 12-weekly. They are less immunogenic than TNF antagonists.
 - Adverse effects are mild (upper respiratory infection, headache, arthralgia). Serious adverse events are rare, estimated at <0.02%.
- **IL-17 antagonists:**
 - Bimekizumab has dual IL-17A/F specificity. Brodalumab blocks the receptor, so IL-17A, C, F and A/F signalling is all disrupted.
 - They are also less immunogenic than TNF antagonists.
 - Common adverse effects are nasopharyngitis and upper respiratory infections.
- **Guideline position:**
 - EuroGuiDerm now emphasises first-line biologics or targeted small molecules. Bimekizumab and deucravacitinib are newly incorporated [w2].
 - BAD still requires failure, intolerance or contraindication of methotrexate and ciclosporin first [w3].
 - BAD eligibility: DLQI or CDLQI >10 with BSA >10% or PASI $\geq$ 10. Severe disease at localised sites such as nail or face also qualifies [w3].
 - Indian dermatologists concurred that biologics are generally given as per international recommendations [w1].
- **Treatment targets:** PASI 75 is the minimum. PASI 90 or an absolute PASI <2 is desirable [w2].
- **Duration:** Do not simply stop therapy. Continue with monitoring, or taper or stop after 6 months of sustained remission [w1].
- **Contraindications (IL-23 and IL-17 classes):**
 - Active infection or untreated latent TB.
 - Recent malignancy, unless treated >5 years ago or cure is likely.
 - Live vaccines. For IL-23 antagonists, avoid within 4 weeks before the first dose and for 12–21 weeks after the last.
- **Pre-treatment screening:**
 - It is the same as for TNF antagonists.
 - Screen for latent TB with IGRA and a chest X-ray. Screen for hepatitis B and HIV.
- **Pregnancy:** All IL-23 antagonists have an Fc portion. They cross the placenta from 16 weeks and are excreted in breast milk.

PEARLS

- Biologic classes: TNF, IL-12/23, IL-23 (p19), IL-17.
- Bimekizumab is the only dual IL-17A/F inhibitor.
- IL-23 blockers have the longest maintenance intervals (8–12 weeks).
- Etanercept is the least effective biologic in head-to-head trials. It is no longer first-line.
- Screen for TB (IGRA plus chest X-ray) and hepatitis B and HIV before any biologic.
- BAD eligibility: failure of methotrexate and ciclosporin, plus PASI $\geq$ 10 or DLQI >10.

Sources

- ROOK V1 — Ch 19 Principles of Systemic Therapy — §Anti-inflammatory and immunomodulatory drugs — p. 19.35
- ROOK V2 — Ch 35 Psoriasis and Related Disorders — pp. 35.28, 35.46
- [w1] Indian expert panel (IDOG) — Systemic Management of Psoriasis Patients in Indian Scenario: An Expert Consensus — 2021 — <https://pmc.ncbi.nlm.nih.gov/articles/PMC8456263/>
- [w2] EuroGuiDerm / German S3 — S3 Guideline for the treatment of psoriasis vulgaris, adapted from EuroGuiDerm, Part 1 (JDDG 2026;24:122–139) — <https://pmc.ncbi.nlm.nih.gov/articles/PMC12800871/>
- [w3] BAD — Guidelines for biologic therapy for psoriasis — 2020 — <https://onlinelibrary.wiley.com/doi/10.1111/bjd.19039>

133. Erythrodermic psoriasis (incl. children)

ASKED 2* · 6-mark scope

Asked in: Dec 2020 · Paper II · Q6(b) (6 marks) · Dec 2021 · Paper II · Q8(a) (4 marks)

Q: Management of a child with psoriatic erythroderma. [6]

Q: Discuss lines of management of psoriatic erythroderma. [4]

Management of a child with psoriatic erythroderma

MANAGEMENT BASIS

Rook's 10e, ch. 39 and ch. 35; India — Indian expert panel — Systemic Management of Psoriasis in Indian Scenario: Expert Consensus — 2021 [w1]; International — EuroGuiDerm/German S3 psoriasis vulgaris guideline — 2026 [w2].

- **Admit** the child, because erythroderma is a skin emergency. Hospital care is frequently needed in acute and fulminant cases.
- Monitor **temperature, fluid and protein–electrolyte balance and circulation** closely.
 - A child's relatively large surface area makes heat and fluid loss and skin failure (sepsis, hypo/hyperthermia, hypoalbuminaemia, dehydration, high-output cardiac failure) easy to precipitate.
 - Correct hypothermia with emollients and warm blankets. Keep the ambient temperature adequate.
- **Look for and remove triggers.** These include systemic illness, irritating topicals, UV, antimalarials, and abrupt withdrawal of corticosteroids, ciclosporin, methotrexate or TNF inhibitors.
 - Withdraw non-essential drugs.
 - Treat infection with the appropriate antibiotics.
- **Topicals:** apply frequent **greasy emollients** to restore barrier function.
 - Other topicals need caution because systemic absorption is greatly increased.
 - Avoid tar and dithranol.
- **Avoid systemic corticosteroids**, which risk pustular transformation.
- **Systemic therapy** is usually needed if there is no response to basic measures.
 - **Methotrexate** has been used successfully in children with GPP.
 - Ciclosporin acts rapidly, but renal function must be watched.
 - **Biologics** for childhood psoriasis: etanercept, adalimumab, secukinumab, ixekizumab and ustekinumab are approved for moderate-to-severe childhood plaque psoriasis [w1].
 - Use a **weight-based** dose from the product label or a paediatric dermatologist's protocol.
- Give analgesia if needed. Avoid opioids, which can worsen pruritus.

Lines of management of psoriatic erythroderma



Figure 35.23 — Rook's Textbook of Dermatology 10e V2, p. 35.14. Erythrodermic psoriasis in an older man.

MANAGEMENT BASIS

Rook's 10e, ch. 39 (treatment ladder for erythroderma) and ch. 35; India — Indian expert panel — Systemic Management of Psoriasis in Indian Scenario — 2021 [w1]; International — EuroGuiDerm/German S3 — 2026 [w2].

Table 1: Treatment ladder for psoriatic erythroderma

Line	Measures
First	Hospital or day-unit admission; stop or switch implicated drugs; correct temperature and fluid balance; treat infection; frequent greasy emollients; analgesia
Second	Methotrexate, acitretin or biologics
Rapid control	Ciclosporin, with renal function monitored because of potential haemodynamic compromise

General and supportive measures (first line)

- The **chronic form** arises from gradual extension of plaques. Patients may be systemically well, and mild topicals are tolerated with a good prognosis.
- The **acute unstable form** may be febrile and ill, and has appreciable mortality if untreated.
- Aim to restore skin barrier function and prevent the complications of skin failure.
 - These are sepsis, thermoregulatory failure, hypoalbuminaemia, anaemia, dehydration and high-output cardiac failure.
- Most patients improve over 1–2 weeks with basic measures. The underlying diagnosis is established during this time.
- Hypoalbuminaemia, oedema and cardiac failure are treated as they arise.

Drugs to avoid

- **Systemic corticosteroids** should be avoided because of pustular transformation.
 - Abrupt steroid withdrawal and potent topical steroids can also precipitate GPP.
- Tar and dithranol are contraindicated.

Systemic therapy (second line)

- **Ciclosporin:** rapid improvement.
 - Where there is GPP overlap, high-dose ciclosporin gives rapid control and a lower dose maintains remission. The range is 2.5–5 mg/kg/day.

PEARLS

- TMEP is a rare adult cutaneous mastocytosis that falls within MPCM.
- Hallmark: persistent fixed red-brown macules with minimal visible telangiectasia, and a negative or weak Darier sign.
- Biopsy shows only mildly increased perivascular mast cells, so special stains are needed.
- Treatment is symptomatic and PUVA is the best option for skin lesions, but responses are poor.
- Exam trap: TMEP usually does not urticate on rubbing, unlike urticaria pigmentosa.

Sources

- ROOK V2 — Ch 46 Mastocytosis — pp. 46.1–46.5
- IADVL V3 — Ch 87 Mastocytosis — pp. 2687, 2695

160. DRESS: diagnosis (RegiSCAR) & management**ASKED 6*** · 10-mark scope

Asked in: Jun 2020 · Paper IV · Q6(b) (5 marks) · Jun 2021 · Paper II · Q2(b) (7 marks) · Dec 2021 · Paper II · Q10(c) (3 marks) · Oct 2023 · Paper III · Q8(b) (5 marks) · Oct 2024 · Paper II · Q6(a) (5 marks) · Oct 2024 · Paper II · Q6(b) (5 marks)

Q: Drug rash with eosinophilia and systemic symptoms. [5]

Q: Diagnostic criteria and treatment of Drug Reaction with Eosinophilia and Systemic Symptoms (DRESS). [7]

Q: DRESS (Drug rash with eosinophilia & systemic symptoms). [3]

Q: DRESS syndrome in HIV. [5]

Q: RegiSCAR (Severe Cutaneous Adverse Reaction) scoring system for DRESS (Drug Rash with Eosinophilia and Systemic Symptoms). [5]

Q: Clinical features, diagnosis and management of DRESS. [5]

Drug rash with eosinophilia and systemic symptoms: definition

- **DRESS** is an idiosyncratic, multisystem, delayed drug hypersensitivity. It is one of the SCAR syndromes, with a rash plus haematological and solid-organ disturbance.
- Synonyms: DIHS, anticonvulsant hypersensitivity syndrome, DIDMOHS. The acronym DRESS was proposed by Bocquet et al. in 1996.
- Incidence is **1 in 1000 to 1 in 10 000** among anticonvulsant users. Mean age of onset is 48 years.
- Mortality is **5–10%**, mostly from hepatic failure.
- Mechanisms:
 - drug-specific T-cell reaction (haptens, p-i concept)
 - sequential herpesvirus reactivation (HHV-6, EBV, then HHV-7, CMV)
 - HLA associations

Clinical features

- **Latency 2–6 weeks** after the culprit drug. It is shorter (<2 weeks) with antibiotics and contrast media, and longer with anticonvulsants, allopurinol and sulfasalazine.
- Prodrome of malaise and fever. Rash with **facial swelling** is usually the first sign.
- Common culprits:
 - allopurinol
 - carbamazepine, phenytoin, lamotrigine
 - vancomycin, minocycline
 - sulpha drugs, including dapsone
- Rash types (Walsh):
 - **urticated papular exanthem** (commonest)
 - morbilliform

- exfoliative erythroderma
- EM-like, with atypical targets and purpura. This type goes with severe liver dysfunction and higher mortality.
- **Head and neck oedema** is seen in most patients, best seen at the ears.
- Cheilitis is common. Frank mucosal involvement is rare and questions the diagnosis. Pustules are sparse.
- Lymphadenopathy at ≥ 2 sites is present in most patients.
- Blood:
 - eosinophilia (most consistent)
 - atypical lymphocytes
 - lymphocytosis, or lymphopenia/thrombocytopenia
- Organs:
 - **liver** (70–95%) is the commonest and the main cause of death
 - kidney (up to 10%; interstitial nephritis, especially with allopurinol)
 - myocarditis (ANEM, mortality >50%)
 - lung, CNS, gut
 - thyroid (monitor for 1 year)
 - pancreas
- Sequelae: autoimmune thyroiditis, type 1 diabetes, SLE, alopecia areata.
- A chronic phase occurs in a minority of patients.



Figure 118.7 — Rook's Textbook of Dermatology 10e V4, p. 118.7. Drug reaction with eosinophilia and systemic symptoms (DRESS). The

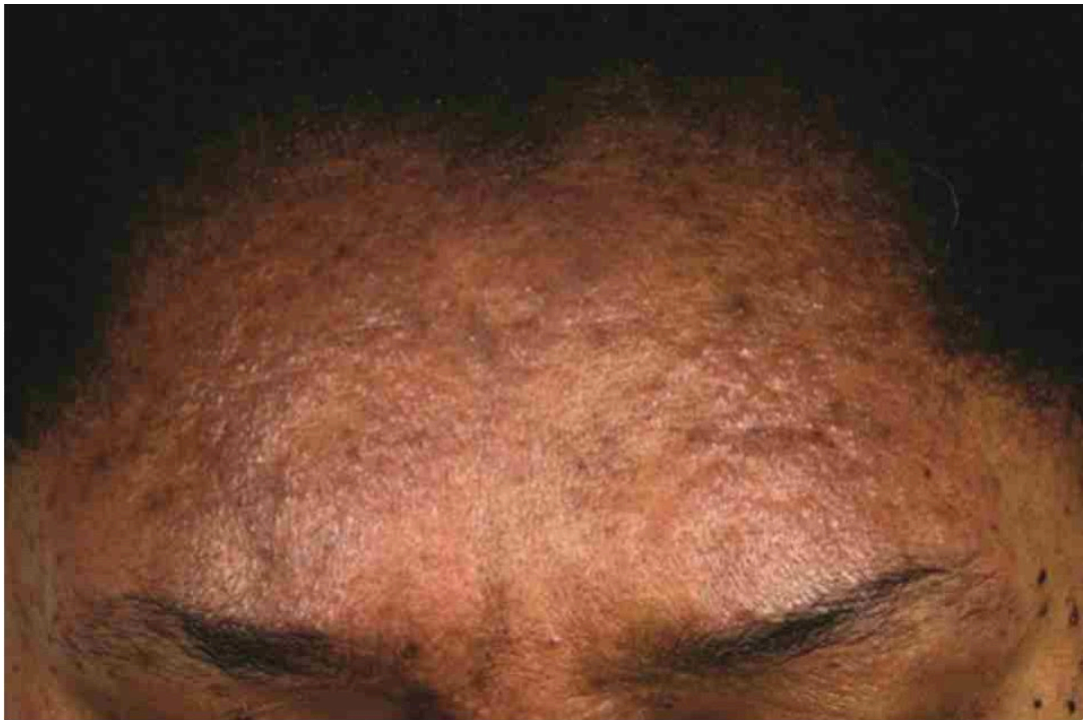


Figure 118.11 — Rook's Textbook of Dermatology 10e V4, p. 118.8. Drug reaction with eosinophilia and systemic symptoms (DRESS). Facial

RegiSCAR scoring system and diagnostic criteria

- This is the preferred scoring system. Patients may fulfil it only later in the illness, and "mini-DRESS" describes those who never fully do.
- Interpretation: <2 no case; 2–3 possible; 4–5 probable; ≥6 (>5) definite.

Clinical feature	Score
Extent of rash >50% body surface area	1 point
Rash suggestive of DRESS	1 point
Systemic involvement: lymphadenopathy ^a , eosinophilia ^b , atypical lymphocytosis ^b , organ involvement ^c	Maximum 6 points
Relevant negative serological tests ^d	1 point

<2 points: no case; 2–3 points: possible case; 4–5 points: probable case; >5 points: definite case.

^a ≥2 sites, ≥1 cm. A maximum of 1 point gained from lymphadenopathy.

^b Eosinophilia: 10–19% of total white cell count = 1 point; ≥20% = 2 points (if total leukocytes <4 × 10⁹/L, an eosinophil count of 0.7–1.5 × 10⁹/L will gain 1 point, an eosinophil count ≥1.5 × 10⁹/L will score 2 points). Atypical lymphocytosis will gain 1 point.

^c Liver: transaminases >2 × upper limit of normal (ULN) on two successive dates or bilirubin × 2 ULN on two successive days or aspartate aminotransferase, γ-glutamyltransferase and alkaline phosphatase >2 × ULN on one occasion. Renal: creatinine 1.5 × patient's baseline. Cardiac: echocardiographic evidence of pericarditis. Maximum of 2 points gained from internal organ involvement.

^d ≥3 of the following performed and negative: hepatitis A, B and C; *Mycoplasma*/chlamydia; antinuclear antibody; blood culture (performed ≤3 days after hospitalisation). A maximum of 1 point is gained for relevant negative serological tests.

Table 118.2 — Rook's Textbook of Dermatology 10e V4, p. 118.9. RegiSCAR drug reaction with eosinophilia and systemic symptoms

Table 1: RegiSCAR items and scores

Item	Score
Fever ≥ 38.5 °C	absent -1 [w1]
Lymph nodes (≥ 2 sites, ≥ 1 cm)	+1 (maximum 1 from nodes)
Eosinophils 700–1499/ μ L	+1 [w1]
Eosinophils ≥ 1500 / μ L	+2 [w1]
Atypical lymphocytes	+1 [w1]
Rash $>50\%$ BSA	+1
Rash suggestive of DRESS	+1; absent -1 [w1]
Biopsy suggestive of DRESS	not suggestive -1 [w1]
Organ involvement (liver, kidney, heart, etc.)	maximum 2
≥ 3 relevant negative serologies (HAV/HBV/HCV, Mycoplasma/Chlamydia, ANA, blood culture)	+1

- Liver involvement counts if transaminases are $>2\times$ ULN on two successive occasions.
- Japanese DIHS criteria differ:
 - they need a rash after >3 weeks of drug and persistence after withdrawal
 - they need fever ≥ 38 °C, ALT >100 U/L, and leukocyte abnormality
 - they need lymphadenopathy and **HHV-6 reactivation**
 - all seven present = typical DIHS; all except the last two (lymphadenopathy, HHV-6 reactivation) = atypical DIHS
- Severity score (Mizukawa 2019):
 - **<1** mild (topical therapy)
 - **1–3** moderate (systemic steroid ≤ 50 mg/day)
 - **≥ 4** severe (steroid ≥ 50 mg/day)

Investigations

- Full blood count with differential and a **blood film for atypical lymphocytes**.
- LFT, LDH, ferritin, coagulation screen, urea/creatinine, urinalysis.
- Hepatitis B and C, EBV, CMV, HHV-6 and HHV-7 titres.
- ECG, echocardiogram and troponin; chest X-ray; TSH and glucose.
- Skin biopsy:
 - spongiosis
 - superficial perivascular lymphocytic infiltrate
 - dermal eosinophils
 - sometimes lichenoid or EM-like change
- Lymphocyte transformation tests assign culpability with variable sensitivity and specificity.

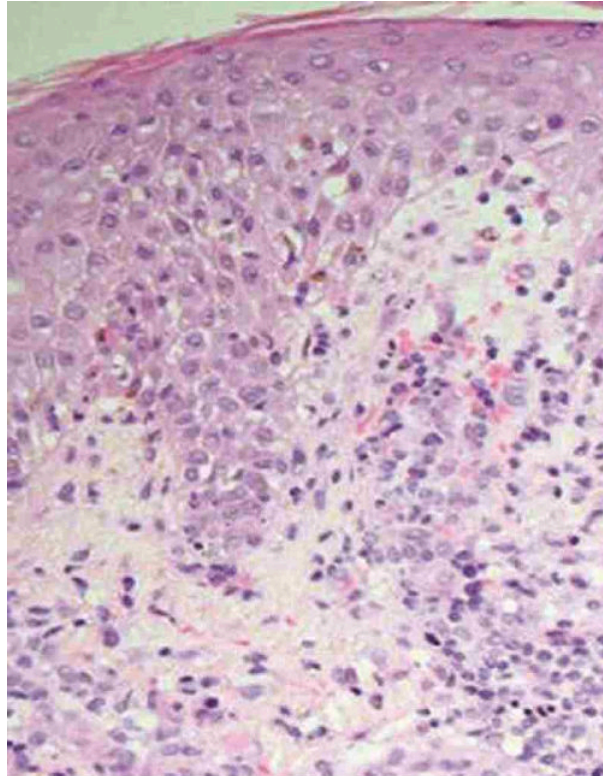


Figure 118.5 — Rook's Textbook of Dermatology 10e V4, p. 118.6. Drug reaction with eosinophilia and systemic symptoms (DRESS).

Differential diagnosis

- Sepsis or viral infection is the commonest mistake.
- SJS/TEN (latency 7–10 days) and AGEP (latency <5 days, flexural sheets of pustules) have a shorter latency than DRESS.
- Erythroderma-type DRESS mimics eczema, psoriasis or cutaneous lymphoma.
- Angioimmunoblastic lymphoma is a rare mimic.

DRESS in HIV

- HIV-positive patients may be at higher risk, partly because of the drugs used in their treatment.
- **Abacavir** hypersensitivity is linked to HLA-B*5701. Screen before starting.
- Other common precipitants listed in the Indian text: co-trimoxazole, dapsone and NSAIDs.
- Differentiate from opportunistic infection and from CMV or EBV reactivation (see investigations).
- Identify and withdraw the culprit drug, then manage by severity as below.

Management

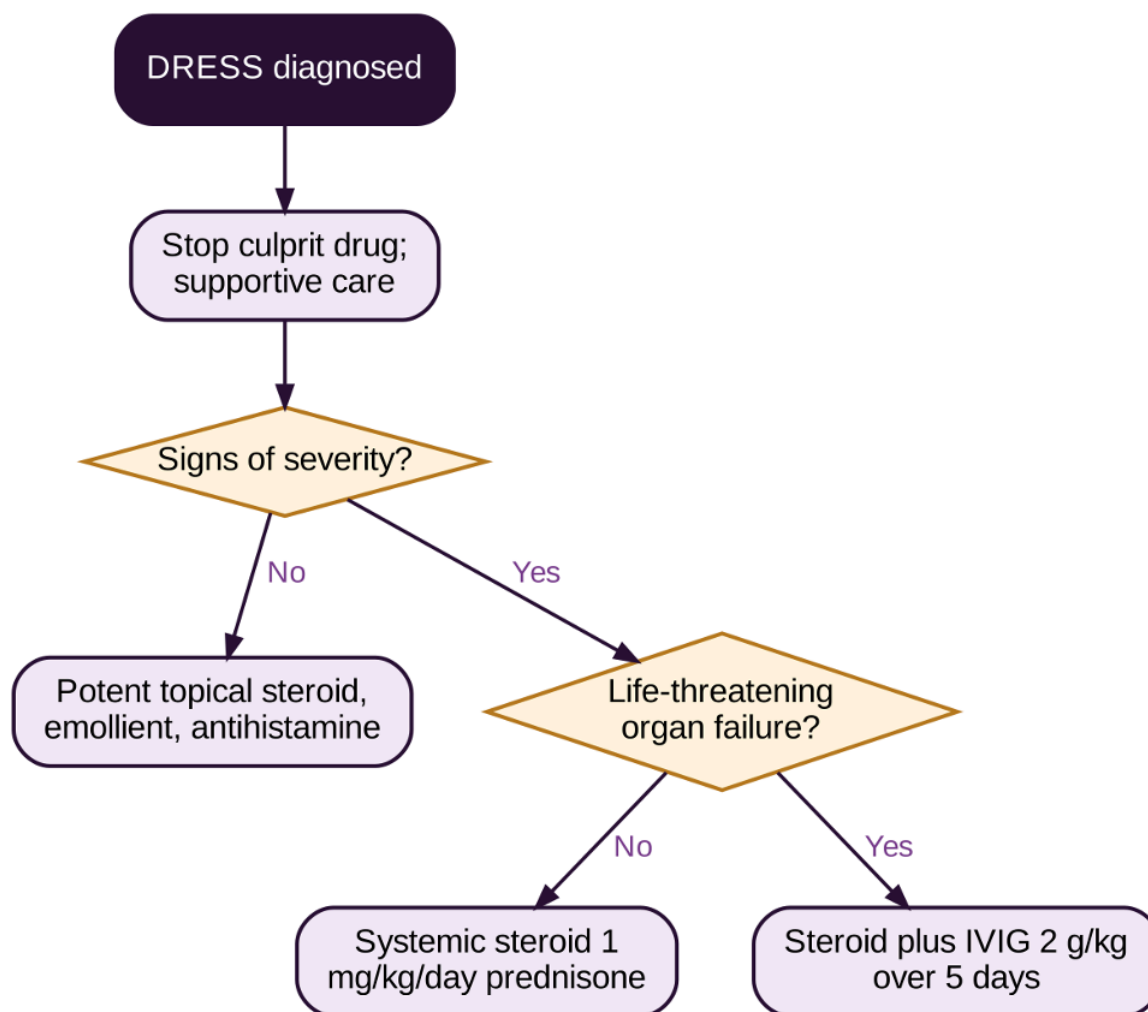
MANAGEMENT BASIS

Rook's 10e, ch. 118; IADVL Textbook 5e, ch. 101 (French Society consensus). No separate IADVL DRESS guideline is given. International: RegiSCAR validation scoring for DRESS [w].

- **Withdraw the culprit drug** and review all medicines to remove unnecessary ones.
- Supportive care in a unit suited to disease severity: IV fluids, thermoregulation, oxygen.
- Do not give empirical antibiotics unless infection is suspected.
- Emollients ± topical corticosteroid. Involve hepatology, cardiology, renal and respiratory teams for organ involvement.
- **Corticosteroids** are the mainstay, by topical, oral or IV route.
- Mild DRESS needs only drug withdrawal and topical steroids. Visceral involvement often needs high-dose systemic steroids [w].
- **Ciclosporin** is a steroid-sparing agent for refractory or chronic disease.

- Evidence rests on case series and has no definitive trials.

Flowchart 1: Management by severity (French Society consensus)



- Severity signs: transaminases $>5\times$ normal, renal or cardiac involvement, pneumonia, haemophagocytosis.
- Do not give immunoglobulin alone.
- With confirmed major viral reactivation, give systemic steroid plus ganciclovir and/or IVIG.
- Follow-up: thyroid function for 1 year, and glucose in the acute and convalescent phases.
- Avoid the culprit drug and related compounds for life, and counsel the patient. Consider family risk with HLA-linked drugs.

PEARLS

- DRESS is a delayed (2–6 weeks) drug reaction with fever, rash, facial oedema, lymphadenopathy, eosinophilia and organ involvement.
- Hallmarks are facial/ear oedema and atypical lymphocytes. Mucosal involvement is rare.
- The liver is the commonest organ involved and the main cause of death.
- RegiSCAR: ≥ 6 is definite, 4–5 probable, 2–3 possible.
- Withdraw the culprit and give steroids. Add IVIG for life-threatening disease and ciclosporin as a steroid-sparing agent.
- HLA links: abacavir with B*5701, allopurinol with B*5801, carbamazepine with B*1502 and A*3101.
- In HIV, think of abacavir, co-trimoxazole and dapsone. Exam trap: infection and herpesvirus reactivation can mimic or complicate DRESS.

Sources

- ROOK V4 — Ch 118 Severe Cutaneous Adverse Reactions to Drugs — §AGEP, DRESS — pp. 118.4–118.11
- IADVL V3 — Ch 101 Drug Reactions — §DRESS — pp. 3007–3009
- [w1] EuroGuiDerm/international DRESS guidance cited in pack: RegiSCAR validation scoring for DRESS — 2026 pack check — <https://onlinelibrary.wiley.com/doi/10.1111/ddg.15515>

161. SJS/TEN: management & SCORTEN

ASKED 3* · 6-mark scope

Asked in: Dec 2020 · Paper II · Q4(b) (5 marks) · Dec 2021 · Paper II · Q10(a) (4 marks) · Apr 2023 · Paper IV · Q7(b) (6 marks)

Q: Management of toxic epidermal necrolysis. [5]

Q: SCORTEN (Scoring system in Toxic Epidermal Necrolysis). [4]

Q: Discuss principles of management of TEN. [6]

Management of toxic epidermal necrolysis

MANAGEMENT BASIS

India: IADVL task force — Guidelines for the management of SJS/TEN: An Indian perspective [w1]. International: BAD — U.K. guidelines for SJS/TEN in adults — 2016 [w2]; German S3 guideline on epidermal necrolysis [w3]. Book: Rook's 10e, ch. 118.

Immediate steps

- **Withdraw the culprit drug at once**, which decreases the risk of death. Continue the patient's other regular medicines.
- Identify the culprit from the onset date and a 2-month drug history with a timeline. Latency is typically 7–10 days (range 5–28).
- ALDEN assesses drug causality retrospectively, not in the acute phase.
- If a drug is essential (epilepsy, septicæmia), substitute a structurally unrelated drug.
- If epidermal loss is >10% BSA, transfer to an ICU or burns unit. Rapid transfer improves survival.
- The care setting is a barrier-nursed side room, a pressure-relieving mattress and an ambient temperature of 25–28 °C.
- The team is multidisciplinary, led by a dermatologist or burns surgeon, with ICU, ophthalmology and skin-care nursing.

Baseline assessment

- Record the extent of erythema and of epidermal detachment separately on a body map.
- Blood tests include FBC, urea and electrolytes, bicarbonate, glucose, LFT, CRP, coagulation and Mycoplasma serology.
- Take a lesional biopsy for histopathology and a periblistar biopsy for DIF. Send skin swabs and do a chest X-ray.
- Compute SCORTEN on admission.

Skin care

- Handle the skin with minimal shearing. Avoid BP cuffs, adhesive ECG leads and adhesive dressings.
- Conservative approach: leave detached epidermis in situ as a biological dressing. Decompress tense bullae and retain the roof.
- Cleanse with warm sterile water or chlorhexidine 1/5000.
- Apply **50% white soft paraffin/50% liquid paraffin** over the whole skin, including denuded areas. [w2]
- Use silicone dressings on exposed dermis, with an absorbent non-adherent secondary layer.
- Use topical antibiotic only on sloughy or crusted areas or proven infection.

Mucosal care

- **Eyes:** ophthalmology review at the start and daily. Apply lubricant 2-hourly and do daily ocular hygiene.
 - Add a topical antibiotic if there is fluorescein staining or ulceration.
 - Consider amniotic membrane transplantation for extensive epithelial loss unresponsive to conservative measures.
- **Mouth:** apply WSP to the lips and use benzydamine rinse 3-hourly. Add chlorhexidine mouthwash twice daily.

- **Genital:** use WSP and silicone sheets on eroded areas. Catheterisation prevents urethral stricture.

Systemic supportive care

- **Fluids:** IV crystalloid at 2 mL/kg/% BSA detached, or guided by urine output. Over-resuscitation causes oedema.
- **Nutrition:** enteral is preferred. Give 20–25 kcal/kg/day in the catabolic phase and 25–30 kcal/kg/day in recovery.
- **Analgesia:** simple analgesia plus opiates. Give morphine 0.1 mg/kg IV bolus 10–20 min before procedures. Ketamine 0.5 mg/kg may be used for procedural analgesia.
- **Prophylaxis:** a proton pump inhibitor if not absorbing food, and LMWH unless contraindicated.
- **Infection:**
 - Swab several sites serially.
 - Give antibiotics only for clinical signs of infection, not prophylactically.
 - Empirical cover is one antistaphylococcal, one Gram-negative and one anaerobic agent.
 - Treat suspected Mycoplasma with a macrolide.
- **Respiratory:** about 25% need ventilation. Bronchoscopic debris removal helps.

Active (immunomodulatory) therapy

- Rook states that no systemic therapy has unambiguous benefit.
- Thalidomide was stopped in an RCT because of excess deaths.

Table 1: Immunomodulators in SJS/TEN

Drug	Evidence/dose	Source
Corticosteroid	Prednisolone 1–2 mg/kg/day within 72 h, tapered over 7–10 days	[w1]
Pulsed steroid	IV methylprednisolone 1000 mg for 3 days in a small series	
Ciclosporin	3–5 mg/kg/day for 10–14 days, alone or with steroid (level II, grade B)	[w1]
IVIg	Pooled mortality OR 1.00 vs supportive care	
Etanercept	Single 50 mg SC dose in an uncontrolled series	
Plasmapheresis, cyclophosphamide	Listed options	[w1]

Table 2: India vs international on active therapy

India: IADVL task force, SJS/TEN guideline	International: BAD 2016 / German S3
Early steroid, or ciclosporin as an alternative [w1]	BAD: no clear evidence for any tablet or injection; culprit withdrawal and supportive care first [w2]
TNF- α inhibitors listed without a dose [w1]	German S3: etanercept may be considered (off-label, grade 0) [w3]

SCORTEN (Scoring system in Toxic Epidermal Necrolysis)

- **Purpose:** a validated prognostic score (Bastuji-Garin, 2000) using seven clinical parameters to predict hospital mortality.
- Score 1 point for each parameter present.
- Calculate it on admission and again within the first 5 days.

Number of parameters	Predicted mortality (%)
0	1.2
1	3.9
2	3.9
3	32.4
4	62.2
5	85.0
6	95.1
7	98.5

Table 118.5 — Rook's Textbook of Dermatology 10e V4, p. 118.18. SCORTEN (SCORe of Toxic Epidermal Necrolysis)

Table 3: SCORTEN parameters (1 point each)

Parameter	Cut-off
Age	>40 years
Malignancy	Present
Heart rate	>120 beats/min
Epidermal detachment at admission	>10% BSA
Serum urea	>10 mmol/L
Serum glucose	>14 mmol/L
Bicarbonate	<20 mmol/L

Source for Table 3:

Table 4: SCORTEN and predicted mortality

Number of parameters	Predicted mortality (%)
0	1.2
1	3.9
2	3.9
3	32.4
4	62.2
5	85.0
6	95.1
7	98.5

Source for Table 4:

WORKED EXAMPLE

Patient aged 55 years, no malignancy, heart rate 130/min, 40% BSA detachment, urea 12 mmol/L, glucose 9 mmol/L, bicarbonate 22 mmol/L.

1. Age >40: 1 point.
2. Malignancy absent: 0.
3. Heart rate >120: 1 point.
4. Detachment >10%: 1 point.
5. Urea >10 mmol/L: 1 point.
6. Glucose ≤14 mmol/L: 0.
7. Bicarbonate ≥20 mmol/L: 0.
8. SCORTEN = 4, so predicted mortality is **62.2%**.

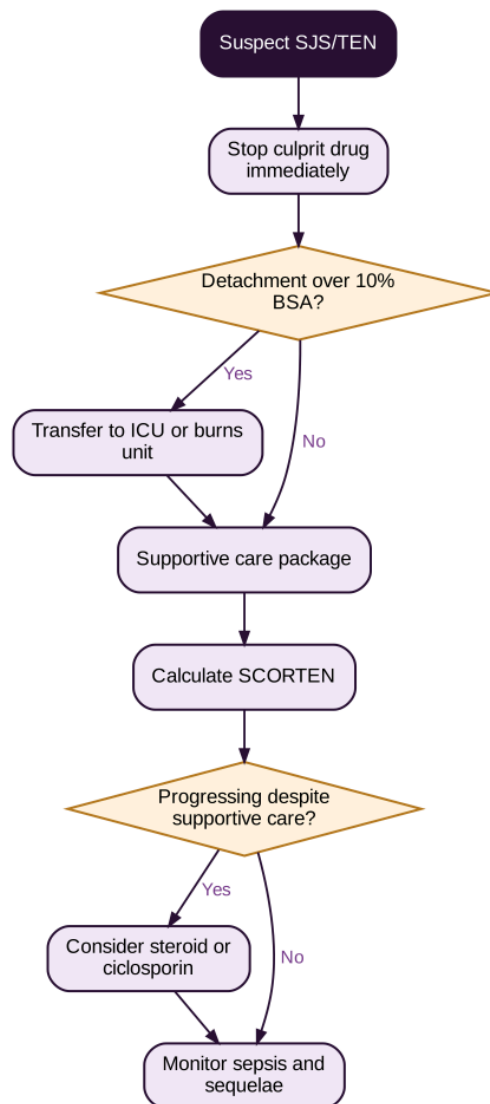
- Overall SJS/TEN mortality is about 22%, and about 30% in TEN.
- The commonest cause of death is septicaemia-induced multiorgan failure.

Principles of management of TEN

MANAGEMENT BASIS

India: IADVL task force — Guidelines for the management of SJS/TEN: An Indian perspective [w1]. International: BAD — U.K. guidelines for SJS/TEN in adults — 2016 [w2]; CPIC — HLA genotype and carbamazepine/oxcarbazepine — 2017 update [w4]. Book: Rook's 10e, ch. 118.

Flowchart 1: Management ladder in TEN



- **Principle 1: remove the cause.** Early withdrawal improves prognosis.
- **Principle 2: treat as acute skin failure, like a large burn.** Specialist unit, MDT and heated environment.
- **Principle 3: meticulous supportive care.** This covers fluids, nutrition, analgesia, thromboprophylaxis and infection surveillance. It is the fundamental element of management.
- **Principle 4: protect the mucosae.** Eyes, mouth and genitals need daily directed care, to prevent late sequelae.
- **Principle 5: no prophylactic antibiotics.** They increase skin colonisation, particularly with Candida.
- **Principle 6: prognosticate with SCORTEN** to guide the level of care.
- **Principle 7: immunomodulation is individualised.** Systemic steroids used indiscriminately may increase sepsis and impair re-epithelialisation.
- **Principle 8: prevent recurrence.** Give a Medic Alert card naming the culprit drug.
- **Principle 9: follow up for sequelae.** Ocular sequelae are the most disabling, so arrange long-term ophthalmology care.
- **HLA screening** is a preventive principle: CPIC advises avoiding carbamazepine, and oxcarbazepine, in HLA-B*15:02-positive drug-naïve patients. [W4]

243. Hidradenitis suppurativa: pathogenesis, staging, complications & management

ASKED 5* · 10-mark scope

Asked in: Dec 2020 · Paper IV · Q1(a) (5 marks) · Jun 2022 · Paper II · Q5 (10 marks) · Dec 2022 · Paper IV · Q2 (10 marks) · Apr 2023 · Paper II · Q7 (10 marks) · Jun 2025 · Paper II · Q6 (10 marks)

Q: Medical management of hidradenitis suppurativa. [5]

Q: Hidradenitis suppurativa: a) Pathogenesis. [3] b) Clinical staging. [2] c) Outline management for each staging. [5] [10]

Q: Hidradenitis Suppurativa: a) Hurleys staging. [2] b) Drug treatment. [3] c) Surgical treatment. [3] d) Lasers. [2] [10]

Q: Hidradenitis suppurativa: etiopathogenesis, clinical presentation and co-morbidities. [10]

Q: a) Discuss the complications of hidradenitis suppurativa. [5] b) Describe treatment modalities for refractory hidradenitis suppurativa. [5] [10]

Pathogenesis (etiopathogenesis)

- HS is a **primary disorder of the hair follicle**, not of apocrine glands.
- Sequence: infundibular hyperkeratosis with outlet narrowing → follicular dilatation/cyst → follicular rupture and inflammation → skin tunnels formed by epidermal strands.
- Perifollicular lymphocytic infiltrate in healthy-looking perilesional skin suggests subclinical inflammation comes before hyperkeratosis.
- Immune: the IL-23/Th17 pathway is activated, with raised IL-1 β , IL-17A, IL-12 and IL-23 in skin.
- Non-lesional keratinocytes show a pro-inflammatory state, with raised β -defensin-2, psoriasin and calgranulin.
- Genetics:
 - About one-third have an affected relative, often autosomal dominant.
 - Loss-of-function mutations in γ -secretase genes (Nicastrin, Presenilin-1, PSENEN) alter Notch signalling. They are mainly seen in Han Chinese patients.
 - There is no genetic test.
- Predisposing factors:
 - **Obesity** and **smoking** (OR 4.3) are the main environmental factors.
 - Hormonal influence is suggested by female preponderance and perimenstrual flares, but androgen levels are normal.
 - Friction and shear contribute. Poor hygiene, depilation and antiperspirants are not relevant.
 - Drugs: anti-TNF- α (paradoxical), lithium, sirolimus.
- Bacteria are secondary. Cultures are often negative or show normal flora (*S. epidermidis*, *S. aureus*).
- Histology:
 - Early: sparse lymphocytic infiltrate and sebaceous gland atrophy.
 - Later: follicular hyperplasia, cysts with free hair shafts, abscesses, and a dense mixed infiltrate with giant cells. This is followed by tunnels and fibrosis.

Clinical presentation

- Definition: chronic inflammatory follicular disease, presenting at or after puberty.
- Mean age is 24 years. Europe has a female excess (3:1), whereas Asia has a female:male ratio of 1:2.
- Diagnosis is clinical (San Francisco modification of the Dessau criteria). All three must be met:
 - Typical lesions.
 - Typical topography.
 - Chronicity, with at least two flares in 6 months.
- Index lesions:
 - Painful inflamed or non-inflamed nodules.
 - Rounded abscesses.
 - Draining or non-draining **tunnels** (the preferred term over sinus tract).
 - Rope-like bridged scars, which may be hypertrophic, atrophic or contractile.

- Paired, polyporous pseudocomedones (**tombstone**). Closed comedones are not seen.
- Nodules last 7–15 days, then regress, partly regress or rupture with malodorous, blood-stained pus.
- Sites: axillae, groin, perineal, perianal and gluteal skin, and infra- and intermammary folds. Retro/preauricular, scalp and truncal sites are less common.
- Males: perianal and gluteal disease. Females: genito-femoral and submammary disease.
- Regional lymphadenopathy is not routine.



Figure 90.6 — Rook's Textbook of Dermatology 10e V3, p. 90.5. Tombstone comedones in the axilla.

Co-morbidities

- Follicular occlusion tetrad: acne conglobata, dissecting cellulitis of the scalp, pilonidal sinus.
- Metabolic syndrome (40%; OR 2.2) and cardiovascular disease (MI incidence rate 1.6, stroke 1.3).
- Crohn disease (strong association), ulcerative colitis (weaker), and HLA-B27-negative spondyloarthropathy.
- Depression and anxiety, with a 2.4-fold higher rate of completed suicide.
- Polycystic ovary disease, Down syndrome (5 times more common), and obstructive sleep apnoea.
- Syndromic forms: PASH and PAPASH.

Hurley clinical staging

Table 1: Hurley stages (single region, not whole body)

Stage	Features
I	Recurrent inflammatory lesions, no tunnels or scarring
II	Recurrent lesions with widely separated tunnels and scarring
III	Multiple interconnected lesions, tunnels and scarring diffusely involving an entire region

Stage	Features
I (Figure 90.13)	Recurrent inflammatory skin lesions without skin tunnels and scarring
II (Figure 90.14)	Recurrent inflammatory skin lesions with widely separated skin tunnels and scarring
III (Figure 90.15)	Multiple interconnected inflammatory lesions, skin tunnels and scarring diffusely involving an entire skin region

Table 90.1 — Rook's Textbook of Dermatology 10e V3, p. 90.7. Definition of the three Hurley stages in hidradenitis suppurativa.

- Baseline staging stratifies therapy but is not useful for assessing response.
- **HiSCR** is the validated response endpoint: $\geq 50\%$ reduction in inflamed lesions, with no rise in abscesses or draining tunnels.
- IHS4 and HASI-R are candidate dynamic scores.
- Frequency in secondary care: stage I 45.5%, II 41.5%, III 13%.

Medical management and drug treatment

MANAGEMENT BASIS

Rook's 10e, ch. 90 (Management); BAD 2018 HS guideline is the source of the book's recommendations.

- Adjuvants:
 - Calculate BMI and refer for weight management.
 - Offer smoking cessation.
 - Advise loose clothing and wound dressings.
 - Give analgesia (paracetamol, NSAIDs). Avoid chronic opioids.
- Topical and intralesional:
 - Clindamycin 0.1% lotion for mild, superficial disease.
 - Intralesional triamcinolone **5–7 mg** for a single nodule.
 - Resorcinol 15% may help. Antiseptic washes and benzoyl peroxide are unproven.
- Oral tetracyclines are first-line oral therapy in mild–moderate disease. Double the acne dose may be needed, given for 12 weeks.
- Advanced disease: clindamycin **300 mg BD** plus rifampicin **300 mg BD** for 10–12 weeks, giving about 50% reduction in severity.
- Antiandrogens and others:
 - Metformin (also usable in pregnancy).
 - Spironolactone **75–100 mg** daily, in women only.
 - Finasteride if spironolactone is not tolerated.
 - Dapsone **50–100 mg** daily.
- Prednisolone **0.5–1.0 mg/kg**, short term, for severe disease unresponsive to other therapy.
- Retinoids: avoid isotretinoin for HS alone. Acitretin may give remission but is avoided in women of child-bearing potential.
- **Adalimumab** is the only FDA/EMA-approved drug: 160 mg at week 0, 80 mg at week 2, then 40 mg weekly from week 4. About 50% reach HiSCR.

Management by Hurley stage

MANAGEMENT BASIS

Rook's 10e, ch. 90, Figure 90.17 and text; sequence is flexible with patient preference, co-morbidity and lesion distribution.

Table 2: Stage-wise outline

Stage	Medical	Procedural
I	Adjuvants, topical clindamycin, intralesional steroid, oral tetracycline	Deroofing or localised excision if few stable lesions
II	Tetracycline, then clindamycin + rifampicin, then adalimumab; metformin or spironolactone/acitretin adjuncts	Deroofing, localised excision, hair-removal laser
III	Adalimumab, infliximab or other biologic; systemic therapy for multiple regions	Wide excision, with multidisciplinary care

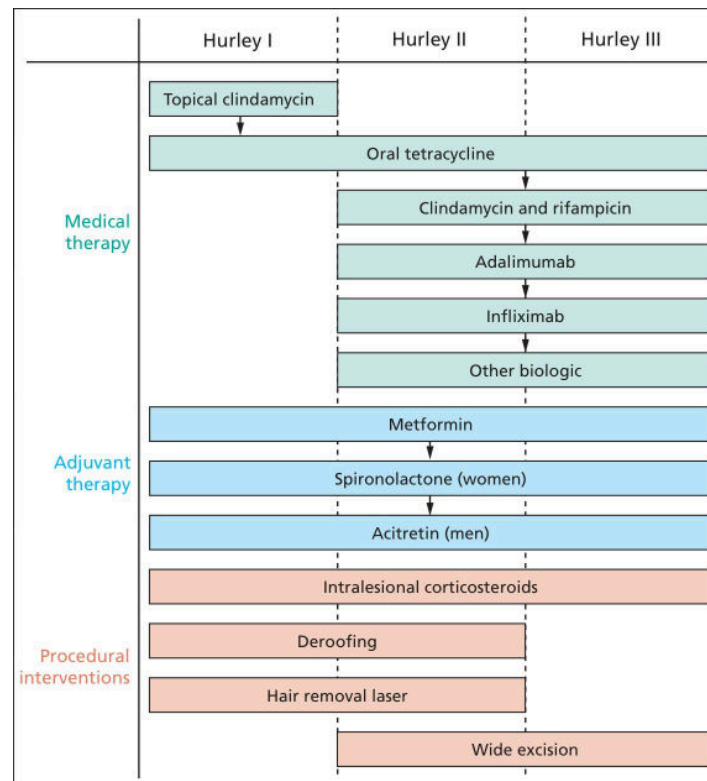


Figure 90.17 — Rook's Textbook of Dermatology 10e V3, p. 90.10. Management flow chart for the treatment of hidradenitis suppurativa

Surgical treatment

MANAGEMENT BASIS

Rook's 10e, ch. 90 (Surgery).

- Wider removal lowers recurrence but lengthens healing.
- **Incision and drainage:** only for frankly fluctuant abscesses. Recurrence is 100%.
- **Deroofing:** tissue-saving.
 - Probe the tunnel and remove its roof by loop electrosurgery or scalpel.
 - Leave the floor to heal by secondary intention.
- **Localised excision:** for few, stable, non-inflamed lesions. Recurrence is lower than after incision and drainage.
- **Extensive excision:** the only cure for whole-region disease.
 - Margins range from 1 cm to all hair-bearing skin of the region.
 - Heal by secondary intention, or close with split-skin graft or flap (e.g. thoracodorsal artery perforator flap).
 - Mobilise early to avoid axillary strictures.
 - Anogenital disease needs plastic and colorectal input.

Lasers

MANAGEMENT BASIS

Rook's 10e, ch. 90 (Surgery; non-ablative lasers).

- **CO₂ laser** vaporisation is ablative: it evaporates all affected tissue under vision until healthy tissue is reached. It is radical but tissue-sparing.
 - Defects heal by secondary intention.
 - Return to work takes 2–3 weeks, and full re-epithelialisation 8–10 weeks.
- **Hair removal** with Nd:YAG laser or IPL monthly gives significant improvement.

Complications

- Local: superinfection and scarring with contractures.
- Lymphatic obstruction causes lymphoedema, and scrotal elephantiasis can occur.
- Fistulae to the gut, urinary tract and peritoneum are rare and should prompt a search for Crohn disease.
- **SCC**: in chronic anogenital disease, mainly in men (perianal, perineal, buttock), with a poor prognosis.
- Systemic: anaemia, hypoalbuminaemia, hypergammaglobulinaemia, and rarely amyloidosis or sacral osteomyelitis.
- Psychosocial: depression, anxiety and suicide risk.
- Cardiovascular risk raises mortality. Mean life expectancy is 61 years, versus 75 in controls.

Treatment modalities for refractory HS

MANAGEMENT BASIS

Rook's 10e, ch. 90 (Management); BAD 2018 as cited there.

- Optimise adjuvants (weight, smoking) and add a tetracycline to adalimumab if needed.
- **Adalimumab** at the standard schedule, with concomitant oral tetracycline for extra control.
- Other biologics:
 - Infliximab: the primary endpoint was missed in the RCT, but post hoc analysis showed benefit.
 - Secukinumab (anti-IL-17) and ustekinumab (anti-IL-23): promising open data, RCTs awaited.
 - MABp1 (anti-IL-1α): benefit in a small RCT.
 - Etanercept: no benefit.
- Rescue IV **ertapenem 1 g daily for 6 weeks** in very severe suppurative disease.
- Systemic options: short-course prednisolone, dapsone, acitretin, repeat clindamycin–rifampicin.
- Surgery: derroofing, CO₂ laser, wide excision with flap or graft. Initial biologic therapy can delineate scarring for targeted surgery.
- Continue the biologic during and after surgery to prevent relapse.
- Multidisciplinary care and screening for depression, BP, HbA1c and lipids.

PEARLS

- HS = chronic follicular occlusion disease: nodules, tunnels, rope-like scars and tombstone comedones in flexures.
- Diagnosis is clinical (Dessau criteria). Hurley stage guides therapy, and HiSCR measures response.
- First-line oral drug: tetracycline for 12 weeks. Advanced disease: clindamycin plus rifampicin 300 mg BD each.
- Adalimumab is the only approved biologic (160/80/40 mg weekly).
- Avoid I&D except for fluctuant abscesses (100% recurrence). Isotretinoin is rarely effective.
- Look for Crohn disease, metabolic syndrome, depression and SCC.

Sources

- ROOK V3 — Ch 90 Hidradenitis Suppurativa — pp. 90.1–90.11



CLINICA
PUBLICATION

STRATUM CORNEUM

EPIDERMIS

DERMIS

HYPODERMIS

DNB Dermatology, Venereology & Leprosy

Solved Previous-Year Questions

VOLUME 3

Venereology & HIV, Leprosy, Dermatosurgery & Aesthetics, Recent Advances

- Part 17 · Genital Anatomy and Sexually Transmitted Infections
- Part 18 · Syndromic Management, HIV and STI Programmes
- Part 19 · Leprosy: Basic Science, Classification and Diagnosis
- Part 20 · Leprosy: Reactions, Neuritis, Treatment, Disability and Programme
- Part 21 · Dermatosurgery and Lasers
- Part 22 · Aesthetic Dermatology
- Part 23 · Recent Advances: AI, Teledermatology and COVID-19

REVIEWED BY

Dr Soumik Dey
DNB (Dermatology)

Dr Sirsa Ghosh
DNB (Dermatology)

Based on Rook's Textbook of Dermatology (10th ed.), IADVL Textbook of Dermatology (5th ed.),
IAL Textbook of Leprosy (3rd ed.) and current Indian & international guidelines

UPDATED TILL OCTOBER 2026

Order at www.clinicapublication.com

Index

HOW TO USE

Each answer groups every paper and year in which the question was asked (badge = how often) and answers exactly the parts asked, sized to the marks. The amber Management-basis strip names the guideline used (Indian programme or IADVL first, then international). [W#] tags mark guideline/web facts, listed in the Web-Sourced Facts Register. Book figures are reproduced with their book, volume and page; diagram questions also carry a schematic you can draw in the exam. Each answer ends with its Sources (book, chapter and pages).

A • Parts

Part 1 — Structure and Function of the Skin and Appendages	Volume 1	—
Part 2 — Immunology, Physiology, Photobiology and Genetics	Volume 1	—
Part 3 — Dermatopathology and Diagnostic Techniques	Volume 1	—
Part 4 — Topical and Systemic Therapeutics	Volume 1	—
Part 5 — Biologics, Small Molecules, Newer Drugs and Phototherapy	Volume 1	—
Part 6 — Severity Scores, Research Methods and Emergency Care	Volume 1	—
Part 7 — Papulosquamous and Eczematous Disorders	Volume 2	—
Part 8 — Urticaria, Drug Reactions and Mastocytosis	Volume 2	—
Part 9 — Bullous and Connective Tissue Diseases	Volume 2	—
Part 10 — Vasculitis, Neutrophilic, Granulomatous and Vascular Disorders	Volume 2	—
Part 11 — Pigmentary, Hair, Nail and Adnexal Disorders	Volume 2	—
Part 12 — Infections and Infestations	Volume 2	—
Part 13 — Genetic, Metabolic and Nutritional Disorders; Skin in Systemic Disease	Volume 2	—
Part 14 — Paediatric, Pregnancy, Erythroderma and Photodermatoses	Volume 2	—
Part 15 — Cutaneous Oncology	Volume 2	—
Part 16 — Psychodermatology, Environmental and Miscellaneous Dermatoses	Volume 2	—
Part 17 — Genital Anatomy and Sexually Transmitted Infections	35 answers	13
Part 18 — Syndromic Management, HIV and STI Programmes	34 answers	98
Part 19 — Leprosy: Basic Science, Classification and Diagnosis	20 answers	175
Part 20 — Leprosy: Reactions, Neuritis, Treatment, Disability and Programme	33 answers	222
Part 21 — Dermatosurgery and Lasers	18 answers	303
Part 22 — Aesthetic Dermatology	13 answers	347
Part 23 — Recent Advances: AI, Teledermatology and COVID-19	7 answers	379

B • This volume, question by question

341. Female genitalia: labelled diagram & Bartholin glands	3×	13
Asked in: Dec 2021 · Paper I · Q6(a) (4 marks) · Dec 2021 · Paper I · Q6(b) (3 marks) · Jun 2022 · Paper III · Q8(c) (3 marks)		
342. Male genital tract: labelled diagram	1×	15
Asked in: Dec 2020 · Paper III · Q3 (10 marks)		
343. Congenital syphilis: clinical features, diagnosis & management	7×	19
Asked in: Jun 2020 · Paper III · Q5(b) (5 marks) · Dec 2020 · Paper III · Q4(a) (5 marks) · Jun 2022 · Paper III · Q3(b) (4 marks) · Dec 2022 · Paper III · Q2(b) (5 marks) · Apr 2023 · Paper III · Q8(a) (4 marks) · Oct 2024 · Paper III · Q2(b) (7 marks) · Jun 2025 · Paper III · Q9(b) (4 marks)		
344. Syphilis in pregnancy	2×	25
Asked in: Dec 2020 · Paper III · Q4(b) (5 marks) · Jun 2022 · Paper III · Q3(a) (6 marks)		
345. Chancre redux, serofast state & reinfection	2×	28
Asked in: Jun 2021 · Paper III · Q5(b) (5 marks) · Dec 2025 · Paper III · Q4(b) (3 marks)		

346. Jarisch–Herxheimer reaction	2×	29
Asked in: Jun 2021 · Paper III · Q5(a) (5 marks) · Jun 2025 · Paper III · Q9(c) (3 marks)		
347. Biological false-positive VDRL	2×	31
Asked in: Dec 2021 · Paper III · Q4(b) (3 marks) · Oct 2023 · Paper III · Q7(b) (5 marks)		
348. Prozone phenomenon	2×	33
Asked in: Dec 2021 · Paper III · Q4(c) (3 marks) · Apr 2023 · Paper III · Q8(b) (3 marks)		
349. Serological tests, reverse algorithm & monitoring of treatment	2×	33
Asked in: Jun 2022 · Paper III · Q10(b) (3 marks) · Jun 2025 · Paper III · Q9(a) (3 marks)		
350. Secondary syphilis & classification of syphilis	2×	35
Asked in: Apr 2023 · Paper III · Q10(b) (5 marks) · Oct 2024 · Paper III · Q2(a) (3 marks)		
351. Animal models of syphilis	1×	37
Asked in: Jun 2022 · Paper III · Q5(b) (5 marks)		
352. Complications of gonorrhoea	5×	38
Asked in: Jun 2020 · Paper III · Q4(c) (4 marks) · Jun 2021 · Paper III · Q3(a) (5 marks) · Oct 2023 · Paper III · Q2(a) (5 marks) · May 2024 · Paper III · Q2(c) (3 marks) · Dec 2025 · Paper III · Q1(b) (5 marks)		
353. PPNG & antimicrobial resistance in gonorrhoea	5×	42
Asked in: Jun 2020 · Paper III · Q4(b) (4 marks) · Dec 2021 · Paper III · Q2(c) (3 marks) · Jun 2022 · Paper III · Q4(b) (4 marks) · Oct 2023 · Paper III · Q2(b) (5 marks) · May 2024 · Paper III · Q2(b) (3 marks)		
354. Neisseria gonorrhoeae: structure & gonococcal urethritis	2×	45
Asked in: Jun 2020 · Paper III · Q4(a) (2 marks) · May 2024 · Paper III · Q2(a) (4 marks)		
355. Mycoplasma genitalium & genital mycoplasmas	3×	47
Asked in: Jun 2022 · Paper III · Q4(a) (3 marks) · Dec 2022 · Paper III · Q2(a) (5 marks) · Jun 2025 · Paper III · Q8(a) (5 marks)		
356. Urethral discharge in men: approach & investigations	2×	49
Asked in: Dec 2021 · Paper III · Q3(a) (4 marks) · Apr 2023 · Paper III · Q9(a) (5 marks)		
357. Non-gonococcal urethritis: aetiology & management	2×	51
Asked in: Apr 2023 · Paper III · Q9(b) (5 marks) · Oct 2024 · Paper III · Q4(a) (6 marks)		
358. Herpes in pregnancy & neonatal herpes	4×	55
Asked in: Dec 2021 · Paper III · Q1(c) (2 marks) · Apr 2023 · Paper III · Q5(a) (4 marks) · Dec 2025 · Paper III · Q5(a) (5 marks) · Dec 2025 · Paper III · Q5(b) (5 marks)		
359. Acyclovir-resistant / persistent genital herpes	3×	58
Asked in: Jun 2022 · Paper III · Q4(c) (3 marks) · Dec 2022 · Paper III · Q4(a) (5 marks) · May 2024 · Paper III · Q3(b) (3 marks)		
360. Genital herpes: diagnosis (incl. type-specific serology)	2×	60
Asked in: Dec 2021 · Paper III · Q1(b) (4 marks) · May 2024 · Paper III · Q3(c) (4 marks)		
361. Recurrent genital herpes: suppressive therapy	1×	62
Asked in: May 2024 · Paper III · Q3(a) (3 marks)		
362. Anogenital warts: treatment (incl. persistent & paediatric)	3×	63
Asked in: Jun 2021 · Paper III · Q2(b) (5 marks) · May 2024 · Paper III · Q9(b) (5 marks) · Jun 2025 · Paper III · Q7(a) (5 marks)		
363. HPV vaccines (incl. nonavalent)	3×	66
Asked in: Jun 2021 · Paper III · Q2(a) (5 marks) · Dec 2021 · Paper III · Q5(c) (3 marks) · Jun 2022 · Paper III · Q7(a) (3 marks)		
364. HPV genotypes, clinical manifestations & HPV-related cancers	3×	68
Asked in: Dec 2021 · Paper III · Q5(b) (3 marks) · Jun 2022 · Paper III · Q6(a) (3 marks) · Dec 2022 · Paper III · Q10(a) (5 marks)		
365. Bowenoid papulosis	2×	70
Asked in: May 2024 · Paper III · Q10(b) (3 marks) · Dec 2025 · Paper III · Q4(a) (4 marks)		
366. Buschke–Löwenstein tumour	1×	72
Asked in: Apr 2023 · Paper III · Q2(a) (4 marks)		

367. Inguinal bubo: approach, syndromic management & chancroid vs LGV	3×	73
Asked in: Jun 2020 · Paper III · Q1(a) (5 marks) · Oct 2023 · Paper III · Q4(b) (5 marks) · Oct 2024 · Paper III · Q5(b) (6 marks)		
368. Lymphogranuloma venereum	3×	77
Asked in: Jun 2020 · Paper III · Q2 (10 marks) · Jun 2022 · Paper III · Q7(c) (4 marks) · Apr 2023 · Paper III · Q8(c) (3 marks)		
369. Genital ulcer disease: differential diagnosis & approach	1×	81
Asked in: Dec 2021 · Paper III · Q1(a) (4 marks)		
370. Bacterial vaginosis: diagnosis (Amsel, Nugent, clue cells) & complications	6×	83
Asked in: Jun 2021 · Paper III · Q4 (10 marks) · Jun 2022 · Paper III · Q9(b) (3 marks) · Dec 2022 · Paper III · Q8(a) (3 marks) · Dec 2022 · Paper III · Q8(b) (3 marks) · Jun 2025 · Paper III · Q3(a) (5 marks) · Dec 2025 · Paper III · Q4(c) (3 marks)		
371. Vaginal discharge: approach (incl. pregnancy & prepubertal girls)	3×	87
Asked in: Dec 2021 · Paper III · Q2(a) (4 marks) · Oct 2024 · Paper III · Q1(b) (7 marks) · Dec 2025 · Paper IV · Q6(a) (5 marks)		
372. Normal flora / microbiome of female genital tract	2×	92
Asked in: Jun 2022 · Paper I · Q9(b) (4 marks) · Oct 2024 · Paper III · Q1(a) (3 marks)		
373. Vulvovaginal candidiasis (incl. complicated VVC)	2×	94
Asked in: Jun 2022 · Paper III · Q9(c) (4 marks) · Oct 2023 · Paper III · Q8(a) (5 marks)		
374. Trichomoniasis: laboratory diagnosis	1×	96
Asked in: Jun 2022 · Paper III · Q9(a) (3 marks)		
375. Cytolytic vaginitis	1×	97
Asked in: Dec 2025 · Paper IV · Q3(c) (4 marks)		
376. Syndromic management of vaginal discharge & cervicitis	3×	98
Asked in: Dec 2021 · Paper III · Q2(b) (3 marks) · Dec 2022 · Paper III · Q4(b) (5 marks) · Jun 2025 · Paper III · Q3(b) (5 marks)		
377. Syndromic management of genital ulcer disease	2×	102
Asked in: Jun 2020 · Paper III · Q1(b) (5 marks) · May 2024 · Paper III · Q4(a) (4 marks)		
378. Syndromic management of lower abdominal pain (PID)	3×	104
Asked in: Dec 2020 · Paper III · Q1(a) (6 marks) · Dec 2020 · Paper III · Q1(b) (4 marks) · May 2024 · Paper III · Q4(c) (3 marks)		
379. Syndromic management of urethral discharge	1×	109
Asked in: May 2024 · Paper III · Q4(b) (3 marks)		
380. Syndromic case management: principles, advantages & limitations	1×	110
Asked in: Dec 2025 · Paper III · Q3 (10 marks)		
381. Antiretroviral drugs: classification, targets & newer drugs	3×	113
Asked in: Jun 2020 · Paper III · Q3(a) (6 marks) · Dec 2022 · Paper III · Q6(b) (5 marks) · Jun 2025 · Paper IV · Q9(a) (4 marks)		
382. HIV & other STIs: impact on GUD, herpes, chancroid, viral STIs	4×	117
Asked in: Dec 2021 · Paper III · Q5(a) (4 marks) · Apr 2023 · Paper III · Q2(b) (6 marks) · Oct 2023 · Paper III · Q5(a) (5 marks) · Oct 2023 · Paper III · Q5(b) (5 marks)		
383. Integrase inhibitors	2×	121
Asked in: Jun 2020 · Paper III · Q3(b) (4 marks) · Dec 2020 · Paper III · Q5(a) (5 marks)		
384. PrEP & PEP for HIV	3×	122
Asked in: Dec 2020 · Paper III · Q5(b) (5 marks) · Jun 2025 · Paper IV · Q2(b) (3 marks) · Jun 2025 · Paper IV · Q2(c) (4 marks)		
385. Structure & life cycle of HIV / retrovirus	2×	125
Asked in: Jun 2021 · Paper III · Q1(a) (4 marks) · Dec 2022 · Paper III · Q6(a) (5 marks)		
386. Cutaneous manifestations & opportunistic infections in HIV	2×	128
Asked in: Jun 2022 · Paper III · Q7(b) (3 marks) · Apr 2023 · Paper III · Q4(a) (5 marks)		
387. Adverse effects of ART & their cutaneous manifestations	2×	130
Asked in: Dec 2022 · Paper III · Q10(b) (5 marks) · Apr 2023 · Paper III · Q4(b) (5 marks)		

388. ART regimens (adult & paediatric) & monitoring	2×	132
Asked in: Apr 2023 · Paper III · Q5(b) (6 marks) · Oct 2024 · Paper III · Q4(b) (4 marks)		
389. Immune reconstitution inflammatory syndrome (IRIS)	1×	134
Asked in: Jun 2021 · Paper III · Q1(b) (6 marks)		
390. Kaposi sarcoma	1×	138
Asked in: Jun 2022 · Paper III · Q10(a) (3 marks)		
391. HIV testing: pre- & post-test counselling	1×	139
Asked in: Apr 2023 · Paper III · Q10(a) (5 marks)		
392. Persistent generalized lymphadenopathy	1×	140
Asked in: Oct 2024 · Paper III · Q3(a) (5 marks)		
393. Child sexual abuse, POCSO Act & STI evaluation in children	5×	142
Asked in: Jun 2022 · Paper III · Q8(a) (3 marks) · Jun 2022 · Paper III · Q8(b) (4 marks) · Oct 2023 · Paper III · Q4(a) (5 marks) · Jun 2025 · Paper III · Q10(b) (6 marks) · Dec 2025 · Paper III · Q1(a) (5 marks)		
394. Anorectal STIs: proctitis, gay bowel syndrome, anorectal discharge	4×	146
Asked in: Dec 2021 · Paper III · Q3(b) (3 marks) · Dec 2021 · Paper III · Q3(c) (3 marks) · Oct 2024 · Paper III · Q3(b) (5 marks) · Dec 2025 · Paper III · Q2 (10 marks)		
395. STIs in men who have sex with men (MSM)	2×	150
Asked in: Oct 2023 · Paper III · Q3(a) (5 marks) · Dec 2025 · Paper IV · Q8(b) (5 marks)		
396. Arthritis associated with STIs	1×	152
Asked in: Jun 2021 · Paper III · Q3(b) (5 marks)		
397. Partner management in STIs	1×	154
Asked in: Jun 2025 · Paper III · Q10(a) (4 marks)		
398. Venereophobia	1×	155
Asked in: Dec 2025 · Paper III · Q10(b) (5 marks)		
399. Non-venereal genital ulcers & Lipschutz ulcer	3×	157
Asked in: Dec 2020 · Paper III · Q2(b) (5 marks) · Oct 2023 · Paper III · Q10(b) (5 marks) · Dec 2025 · Paper IV · Q6(b) (5 marks)		
400. Vulvar pruritus & non-venereal vulvar dermatoses	3×	159
Asked in: Dec 2021 · Paper I · Q6(c) (3 marks) · May 2024 · Paper III · Q8(b) (5 marks) · Jun 2025 · Paper III · Q8(b) (5 marks)		
401. Lichen sclerosus (genital & extragenital)	2×	163
Asked in: Jun 2022 · Paper III · Q6(c) (4 marks) · Oct 2023 · Paper II · Q5(b) (5 marks)		
402. Plasma cell balanitis	1×	165
Asked in: Jun 2022 · Paper III · Q6(b) (3 marks)		
403. Genital Crohn's disease	1×	166
Asked in: May 2024 · Paper III · Q10(c) (4 marks)		
404. Red scrotum	1×	168
Asked in: May 2024 · Paper III · Q10(a) (3 marks)		
405. Point-of-care tests for STIs	2×	169
Asked in: Jun 2020 · Paper III · Q5(a) (5 marks) · May 2024 · Paper III · Q9(a) (5 marks)		
406. PCR / NAAT in STIs	2×	171
Asked in: Dec 2021 · Paper III · Q4(a) (4 marks) · Oct 2023 · Paper III · Q7(a) (5 marks)		
407. National AIDS Control Programme (NACP-V)	1×	172
Asked in: Dec 2022 · Paper III · Q8(c) (4 marks)		
408. Doxy-PEP	1×	173
Asked in: Jun 2025 · Paper IV · Q2(a) (3 marks)		
409. EGASP (Enhanced Gonococcal AMR Surveillance)	1×	174
Asked in: Jun 2025 · Paper IV · Q9(b) (2 marks)		

410. Host factors, HLA & immune response in leprosy	3×	175
Asked in: Jun 2021 · Paper III · Q6(a) (5 marks) · Oct 2023 · Paper III · Q6(a) (5 marks) · Oct 2024 · Paper III · Q6(a) (5 marks)		
411. Experimental leprosy & animal models	2×	178
Asked in: Jun 2021 · Paper III · Q10 (10 marks) · Jun 2022 · Paper III · Q5(a) (5 marks)		
412. Lepromin test	1×	180
Asked in: Jun 2021 · Paper III · Q6(b) (5 marks)		
413. Nodular lesions in leprosy: differential diagnosis	2×	182
Asked in: Oct 2023 · Paper III · Q10(a) (5 marks) · Dec 2025 · Paper III · Q8(b) (5 marks)		
414. Ridley–Jopling classification	1×	184
Asked in: Jun 2021 · Paper III · Q7(a) (7 marks)		
415. Differentiating points across the leprosy spectrum (sub-polar LL, histoid, BT vs BB)	1×	186
Asked in: May 2024 · Paper III · Q5 (10 marks)		
416. Atypical presentations of leprosy	4×	190
Asked in: Jun 2020 · Paper III · Q10(c) (3 marks) · Dec 2020 · Paper III · Q9 (10 marks) · Oct 2023 · Paper III · Q9(b) (5 marks) · Dec 2025 · Paper III · Q6(a) (5 marks)		
417. Childhood leprosy	4×	194
Asked in: Jun 2020 · Paper III · Q10(a) (3 marks) · Dec 2021 · Paper III · Q7(c) (3 marks) · Oct 2023 · Paper III · Q9(a) (5 marks) · Jun 2025 · Paper III · Q2(b) (5 marks)		
418. Histoid leprosy (clinical, diagnosis, histopathology)	4×	196
Asked in: Jun 2020 · Paper III · Q8 (10 marks) · Dec 2020 · Paper III · Q7(b) (5 marks) · Dec 2021 · Paper III · Q10(a) (4 marks) · Oct 2024 · Paper III · Q10(b) (4 marks)		
419. Leprosy & pregnancy	3×	200
Asked in: Jun 2020 · Paper III · Q10(b) (4 marks) · Dec 2021 · Paper III · Q7(a) (4 marks) · Dec 2025 · Paper III · Q6(b) (5 marks)		
420. Pure neuritic leprosy	3×	204
Asked in: Jun 2021 · Paper III · Q9(a) (5 marks) · Jun 2022 · Paper III · Q1(a) (5 marks) · Jun 2025 · Paper III · Q2(a) (5 marks)		
421. Lazarine & autoaggressive leprosy	2×	207
Asked in: Dec 2021 · Paper III · Q9(c) (3 marks) · Jun 2025 · Paper III · Q7(c) (2 marks)		
422. Leprosy & HIV	1×	207
Asked in: Dec 2021 · Paper III · Q7(b) (3 marks)		
423. Indeterminate leprosy	1×	208
Asked in: Dec 2022 · Paper III · Q9(c) (2 marks)		
424. Serology & biomarkers (anti-PGL-1)	4×	210
Asked in: Jun 2022 · Paper III · Q2(a) (4 marks) · Apr 2023 · Paper III · Q7(a) (5 marks) · Oct 2024 · Paper III · Q6(b) (5 marks) · Jun 2025 · Paper III · Q1(b) (5 marks)		
425. Bacterial index & morphological index	3×	212
Asked in: Jun 2022 · Paper III · Q2(c) (3 marks) · Oct 2024 · Paper III · Q7(a) (2 marks) · Jun 2025 · Paper III · Q6(c) (3 marks)		
426. Nerve biopsy	2×	214
Asked in: Jun 2022 · Paper III · Q1(b) (5 marks) · Dec 2025 · Paper III · Q9(a) (5 marks)		
427. Histopathology of leprosy (TT vs LL)	1×	215
Asked in: Jun 2021 · Paper III · Q7(b) (3 marks)		
428. PCR in leprosy	1×	217
Asked in: Jun 2022 · Paper III · Q2(b) (3 marks)		
429. Slit-skin smear & AFB staining (incl. Fite stain)	1×	217
Asked in: May 2024 · Paper III · Q7 (10 marks)		
430. Thalidomide: role, adverse effects & monitoring	4×	222
Asked in: Dec 2020 · Paper III · Q8(a) (5 marks) · Dec 2021 · Paper III · Q6(b) (3 marks) · Dec 2022 · Paper III · Q7(b) (3 marks) · Dec 2025 · Paper III · Q7(b) (5 marks)		

431. ENL: pathogenesis, cytokines, investigations & management	4×	225
Asked in: Jun 2020 · Paper III · Q7 (10 marks) · Dec 2021 · Paper III · Q6(a) (3 marks) · Dec 2021 · Paper III · Q6(c) (4 marks) · May 2024 · Paper III · Q1(c) (4 marks)		
432. Recalcitrant / steroid-dependent ENL	3×	232
Asked in: Dec 2022 · Paper III · Q7(a) (4 marks) · Oct 2024 · Paper III · Q9(b) (5 marks) · Dec 2025 · Paper III · Q10(a) (5 marks)		
433. Type 1 (reversal/downgrading) reaction & differences between reactions	2×	235
Asked in: Dec 2021 · Paper III · Q9(a) (4 marks) · May 2024 · Paper III · Q1(b) (3 marks)		
434. ENL: systemic involvement in type 2 reaction	2×	237
Asked in: May 2024 · Paper III · Q1(a) (3 marks) · Dec 2025 · Paper III · Q7(a) (5 marks)		
435. Lucio phenomenon	1×	239
Asked in: Jun 2025 · Paper III · Q6(a) (3 marks)		
436. Nerve damage: classification & assessment (VMT, ST, SW filaments)	4×	241
Asked in: Dec 2021 · Paper III · Q10(c) (3 marks) · Dec 2022 · Paper III · Q3(c) (3 marks) · Apr 2023 · Paper III · Q6(b) (6 marks) · Jun 2025 · Paper III · Q1(a) (5 marks)		
437. Neuritis: diagnosis & management (incl. silent neuritis)	2×	244
Asked in: Jun 2021 · Paper III · Q9(b) (5 marks) · Dec 2022 · Paper III · Q3(b) (4 marks)		
438. Foot drop: differential diagnosis, investigations & treatment	2×	246
Asked in: Dec 2021 · Paper III · Q8(a) (3 marks) · Dec 2021 · Paper III · Q8(b) (4 marks)		
439. Nerve abscess	1×	249
Asked in: Apr 2023 · Paper III · Q6(a) (4 marks)		
440. Relapse in leprosy (definition, causes, diagnosis, relapse vs reaction)	5×	250
Asked in: Jun 2020 · Paper III · Q6 (10 marks) · Dec 2021 · Paper III · Q10(b) (3 marks) · Dec 2022 · Paper III · Q5(c) (3 marks) · Oct 2023 · Paper III · Q1 (10 marks) · Oct 2024 · Paper III · Q7(c) (6 marks)		
441. Dapsone hypersensitivity syndrome	2×	254
Asked in: Dec 2020 · Paper III · Q8(b) (5 marks) · Dec 2022 · Paper III · Q7(c) (3 marks)		
442. WHO MDT: current regimens, modifications & newer drugs	2×	257
Asked in: Dec 2020 · Paper III · Q10(b) (4 marks) · Oct 2023 · Paper III · Q3(b) (5 marks)		
443. Second-line anti-leprosy drugs (minocycline, clofazimine)	2×	259
Asked in: Dec 2021 · Paper III · Q9(b) (3 marks) · Jun 2025 · Paper III · Q6(b) (4 marks)		
444. Drug resistance in leprosy (MDR)	2×	260
Asked in: Apr 2023 · Paper III · Q7(b) (5 marks) · May 2024 · Paper III · Q8(a) (5 marks)		
445. Persisters in leprosy	2×	262
Asked in: Oct 2024 · Paper III · Q7(b) (2 marks) · Jun 2025 · Paper III · Q7(b) (3 marks)		
446. WHO MDT in special situations (defaulter, pregnancy, TB, HIV)	1×	263
Asked in: Jun 2025 · Paper III · Q5 (10 marks)		
447. Trophic ulcer, foot care & care of anaesthetic hands/feet	5×	265
Asked in: Jun 2020 · Paper III · Q9(a) (5 marks) · Jun 2022 · Paper III · Q10(c) (4 marks) · Dec 2022 · Paper III · Q1(a) (6 marks) · Dec 2022 · Paper III · Q9(a) (4 marks) · Oct 2024 · Paper III · Q8(b) (5 marks)		
448. Hand deformities (claw hand): enumeration & surgical management	3×	268
Asked in: Jun 2020 · Paper III · Q9(b) (5 marks) · Dec 2020 · Paper III · Q10(a) (6 marks) · May 2024 · Paper III · Q6(a) (4 marks)		
449. Facial deformities (leonine facies) & surgical correction	3×	273
Asked in: Dec 2020 · Paper III · Q7(a) (5 marks) · Dec 2022 · Paper III · Q1(b) (4 marks) · Dec 2025 · Paper III · Q8(a) (5 marks)		
450. MCR footwear	2×	276
Asked in: Dec 2020 · Paper III · Q6(a) (5 marks) · Dec 2021 · Paper III · Q8(c) (3 marks)		
451. WHO disability grading	2×	278
Asked in: Dec 2020 · Paper III · Q6(b) (5 marks) · Dec 2022 · Paper III · Q5(b) (3 marks)		

452. Splints & splinting	1×	280
Asked in: Dec 2022 · Paper III · Q5(a) (4 marks)		
453. Paralytic deformities	1×	282
Asked in: Apr 2023 · Paper III · Q3(b) (5 marks)		
454. Ocular leprosy	3×	284
Asked in: Dec 2022 · Paper III · Q9(b) (4 marks) · Oct 2024 · Paper III · Q8(a) (5 marks) · Jun 2025 · Paper III · Q4(b) (5 marks)		
455. Renal involvement	2×	287
Asked in: Apr 2023 · Paper III · Q3(a) (5 marks) · Jun 2025 · Paper III · Q4(a) (5 marks)		
456. Musculoskeletal involvement	1×	289
Asked in: Jun 2021 · Paper III · Q8(b) (7 marks)		
457. Systemic involvement in leprosy (overview)	1×	291
Asked in: Jun 2021 · Paper III · Q8(a) (3 marks)		
458. WHO Global Leprosy Strategy 2021–2030 & elimination targets	3×	292
Asked in: May 2024 · Paper III · Q6(c) (3 marks) · Oct 2024 · Paper III · Q9(a) (5 marks) · Oct 2024 · Paper III · Q10(a) (6 marks)		
459. Chemoprophylaxis (SDR-PEP)	2×	296
Asked in: Apr 2023 · Paper III · Q1(a) (4 marks) · May 2024 · Paper III · Q6(b) (3 marks)		
460. Leprosy vaccines (incl. LepVax)	2×	297
Asked in: Apr 2023 · Paper III · Q1(b) (6 marks) · Dec 2025 · Paper IV · Q3(b) (3 marks)		
461. Leprosy in India: current status & epidemiological indicators	2×	299
Asked in: Oct 2023 · Paper III · Q6(b) (5 marks) · Dec 2025 · Paper III · Q9(b) (5 marks)		
462. Leprosy-cured person	1×	302
Asked in: Dec 2022 · Paper III · Q3(a) (3 marks)		
463. Radiofrequency & electrosurgery	3×	303
Asked in: Jun 2022 · Paper I · Q6(c) (3 marks) · Jun 2022 · Paper IV · Q4(b) (3 marks) · Dec 2022 · Paper IV · Q7 (10 marks)		
464. Cryotherapy / cryosurgery	2×	307
Asked in: Dec 2020 · Paper IV · Q4(b) (5 marks) · Apr 2023 · Paper IV · Q4 (10 marks)		
465. Procedural dermatology: definition & classification	2×	309
Asked in: Jun 2022 · Paper IV · Q6(b) (3 marks) · May 2024 · Paper IV · Q5(a) (3 marks)		
466. Sutures & suturing techniques	5×	310
Asked in: Jun 2022 · Paper I · Q6(a) (3 marks) · Jun 2022 · Paper I · Q6(b) (4 marks) · May 2024 · Paper IV · Q4(a) (2 marks) · May 2024 · Paper IV · Q4(b) (2 marks) · May 2024 · Paper IV · Q4(c) (3 marks)		
467. Local anaesthesia: facial blocks, ring block, lignocaine toxicity	3×	315
Asked in: Dec 2021 · Paper I · Q8(a) (4 marks) · Dec 2021 · Paper I · Q8(b) (3 marks) · Dec 2021 · Paper I · Q8(c) (3 marks)		
468. Lasers in pigmentary disorders & picosecond laser	4×	317
Asked in: Dec 2021 · Paper IV · Q9(b) (3 marks) · Jun 2022 · Paper IV · Q3(b) (3 marks) · Oct 2023 · Paper IV · Q6(a) (5 marks) · Dec 2025 · Paper IV · Q7(a) (5 marks)		
469. Laser hair removal (diode vs Nd:YAG)	2×	320
Asked in: Jun 2020 · Paper IV · Q10(b) (5 marks) · May 2024 · Paper IV · Q1(b) (4 marks)		
470. Lasers for tattoo removal	2×	322
Asked in: Jun 2020 · Paper IV · Q10(a) (5 marks) · Jun 2025 · Paper IV · Q3(a) (5 marks)		
471. Lasers for vascular lesions	1×	324
Asked in: Dec 2021 · Paper IV · Q9(a) (4 marks)		
472. Ablative & fractional lasers	1×	325
Asked in: Jun 2022 · Paper IV · Q10(c) (4 marks)		
473. Principles of lasers	1×	327
Asked in: Oct 2024 · Paper IV · Q6(a) (5 marks)		

474. Microneedling / dermaroller	3×	329
Asked in: Dec 2020 · Paper IV · Q2(b) (5 marks) · Jun 2022 · Paper IV · Q4(c) (3 marks) · Dec 2022 · Paper IV · Q5(a) (3 marks)		
475. Microneedling radiofrequency (MNRF)	3×	331
Asked in: Dec 2021 · Paper IV · Q9(c) (3 marks) · Oct 2023 · Paper IV · Q6(b) (5 marks) · Dec 2025 · Paper IV · Q9(a) (5 marks)		
476. Acne scars: management (surgical & procedural)	3×	333
Asked in: Dec 2022 · Paper IV · Q1(b) (5 marks) · Apr 2023 · Paper IV · Q10(a) (6 marks) · Jun 2025 · Paper IV · Q10(b) (6 marks)		
477. Acne scars: classification	1×	337
Asked in: Dec 2020 · Paper IV · Q2(a) (5 marks)		
478. Keloids & hypertrophic scars: treatment options	3×	338
Asked in: Jun 2022 · Paper IV · Q3(a) (3 marks) · Apr 2023 · Paper IV · Q10(b) (4 marks) · May 2024 · Paper IV · Q6 (10 marks)		
479. Hair restoration surgery: evaluation & techniques (FUT/FUE)	7×	342
Asked in: Dec 2020 · Paper IV · Q5(b) (5 marks) · Dec 2021 · Paper IV · Q6(a) (4 marks) · Dec 2022 · Paper IV · Q5(c) (3 marks) · Dec 2022 · Paper IV · Q9(b) (5 marks) · Oct 2024 · Paper IV · Q9(a) (5 marks) · Jun 2025 · Paper IV · Q6(a) (6 marks) · Jun 2025 · Paper IV · Q6(b) (4 marks)		
480. Complications of hair transplantation	1×	346
Asked in: Apr 2023 · Paper IV · Q9(b) (4 marks)		
481. Botulinum toxin: mechanism & uses	3×	347
Asked in: Jun 2020 · Paper IV · Q8(a) (6 marks) · Dec 2021 · Paper IV · Q5(c) (3 marks) · Apr 2023 · Paper IV · Q2(b) (5 marks)		
482. Fillers: complications	2×	351
Asked in: Dec 2021 · Paper IV · Q7(c) (3 marks) · Apr 2023 · Paper IV · Q2(a) (5 marks)		
483. Chemical peels: classification & newer peels	3×	353
Asked in: Dec 2021 · Paper IV · Q6(c) (3 marks) · May 2024 · Paper IV · Q10(c) (3 marks) · Jun 2025 · Paper IV · Q3(b) (5 marks)		
484. Skin resurfacing (ablative methods)	2×	356
Asked in: Dec 2022 · Paper IV · Q9(a) (5 marks) · Apr 2023 · Paper IV · Q9(a) (6 marks)		
485. Facial rejuvenation (non-surgical)	2×	360
Asked in: May 2024 · Paper IV · Q10(b) (4 marks) · Oct 2024 · Paper IV · Q6(b) (5 marks)		
486. Platelet-rich plasma (PRP)	2×	362
Asked in: Dec 2020 · Paper IV · Q5(a) (5 marks) · Oct 2023 · Paper IV · Q10(a) (5 marks)		
487. Platelet-rich fibrin (PRF)	2×	364
Asked in: Jun 2022 · Paper IV · Q10(a) (3 marks) · May 2024 · Paper IV · Q1(c) (3 marks)		
488. Autologous fat transfer	1×	365
Asked in: Jun 2022 · Paper IV · Q10(b) (3 marks)		
489. Growth factors & exosomes in aesthetics	2×	366
Asked in: Jun 2025 · Paper IV · Q5(a) (5 marks) · Jun 2025 · Paper IV · Q5(b) (5 marks)		
490. Nail cosmetics & their adverse effects	2×	370
Asked in: Dec 2022 · Paper IV · Q6(c) (2 marks) · May 2024 · Paper I · Q5(b) (4 marks)		
491. Cosmetics & cosmeceuticals (cleansers, hair treatments)	2×	372
Asked in: Dec 2022 · Paper IV · Q6(a) (5 marks) · Dec 2022 · Paper IV · Q6(b) (3 marks)		
492. Phytochemicals & plant-derived products in cosmetic dermatology	1×	375
Asked in: Oct 2024 · Paper IV · Q7(b) (5 marks)		
493. Pitfalls in cosmetic dermatology	1×	377
Asked in: Oct 2024 · Paper IV · Q9(b) (5 marks)		
494. Artificial intelligence in dermatology	5×	379
Asked in: Jun 2020 · Paper IV · Q1(a) (5 marks) · Dec 2020 · Paper IV · Q9 (10 marks) · Jun 2022 · Paper IV · Q8(a) (4 marks) · Oct 2023 · Paper IV · Q1 (10 marks) · Jun 2025 · Paper IV · Q4 (10 marks)		

495. Teledermatology	2×	382
Asked in: Jun 2020 · Paper IV · Q1(b) (5 marks) · Jun 2022 · Paper IV · Q1(a) (4 marks)		
496. Cutaneous manifestations of COVID-19	2×	383
Asked in: Jun 2020 · Paper IV · Q3(a) (4 marks) · Jun 2022 · Paper IV · Q1(b) (6 marks)		
497. COVID-19: laboratory inflammatory markers	1×	386
Asked in: Jun 2021 · Paper I · Q4(a) (5 marks)		
498. Hand sanitizers	1×	387
Asked in: Jun 2021 · Paper IV · Q2(a) (5 marks)		
499. Online learning during COVID-19	1×	388
Asked in: Jun 2021 · Paper IV · Q2(b) (5 marks)		
500. Post-COVID opportunistic mycoses	1×	390
Asked in: Apr 2023 · Paper IV · Q8(a) (5 marks)		

C · High-yield list (asked 3× or more)

Congenital syphilis: clinical features, diagnosis & management	7×	19
Hair restoration surgery: evaluation & techniques (FUT/FUE)	7×	342
Bacterial vaginosis: diagnosis (Amsel, Nugent, clue cells) & complications	6×	83
Complications of gonorrhoea	5×	38
PPNG & antimicrobial resistance in gonorrhoea	5×	42
Child sexual abuse, POCSO Act & STI evaluation in children	5×	142
Relapse in leprosy (definition, causes, diagnosis, relapse vs reaction)	5×	250
Trophic ulcer, foot care & care of anaesthetic hands/feet	5×	265
Sutures & suturing techniques	5×	310
Artificial intelligence in dermatology	5×	379
Herpes in pregnancy & neonatal herpes	4×	55
HIV & other STIs: impact on GUD, herpes, chancroid, viral STIs	4×	117
Anorectal STIs: proctitis, gay bowel syndrome, anorectal discharge	4×	146
Atypical presentations of leprosy	4×	190
Childhood leprosy	4×	194
Histoid leprosy (clinical, diagnosis, histopathology)	4×	196
Serology & biomarkers (anti-PGL-1)	4×	210
Thalidomide: role, adverse effects & monitoring	4×	222
ENL: pathogenesis, cytokines, investigations & management	4×	225
Nerve damage: classification & assessment (VMT, ST, SW filaments)	4×	241
Lasers in pigmented disorders & picosecond laser	4×	317
Female genitalia: labelled diagram & Bartholin glands	3×	13
Mycoplasma genitalium & genital mycoplasmas	3×	47
Acyclovir-resistant / persistent genital herpes	3×	58
Anogenital warts: treatment (incl. persistent & paediatric)	3×	63
HPV vaccines (incl. nonavalent)	3×	66
HPV genotypes, clinical manifestations & HPV-related cancers	3×	68
Inguinal bubo: approach, syndromic management & chancroid vs LGV	3×	73
Lymphogranuloma venereum	3×	77
Vaginal discharge: approach (incl. pregnancy & prepubertal girls)	3×	87
Syndromic management of vaginal discharge & cervicitis	3×	98

Syndromic management of lower abdominal pain (PID)	3×	104
Antiretroviral drugs: classification, targets & newer drugs	3×	113
PrEP & PEP for HIV	3×	122
Non-venereal genital ulcers & Lipschutz ulcer	3×	157
Vulvar pruritus & non-venereal vulvar dermatoses	3×	159
Host factors, HLA & immune response in leprosy	3×	175
Leprosy & pregnancy	3×	200
Pure neuritic leprosy	3×	204
Bacterial index & morphological index	3×	212
Recalcitrant / steroid-dependent ENL	3×	232
Hand deformities (claw hand): enumeration & surgical management	3×	268
Facial deformities (leonine facies) & surgical correction	3×	273
Ocular leprosy	3×	284
WHO Global Leprosy Strategy 2021–2030 & elimination targets	3×	292
Radiofrequency & electrosurgery	3×	303
Local anaesthesia: facial blocks, ring block, lignocaine toxicity	3×	315
Microneedling / dermaroller	3×	329
Microneedling radiofrequency (MNRF)	3×	331
Acne scars: management (surgical & procedural)	3×	333
Keloids & hypertrophic scars: treatment options	3×	338
Botulinum toxin: mechanism & uses	3×	347
Chemical peels: classification & newer peels	3×	353

PEARLS

- Male genital tract = testis → epididymis → vas deferens → ejaculatory duct → urethra, with accessory glands (seminal vesicles, prostate, Cowper glands).
- Male urethra is 18–20 cm: anterior about 16 cm, posterior 4 cm.
- Narrowest part of the urethra is the external meatus. Widest part is the bulbar urethra.
- Seminal vesicles give 60–70% of ejaculate volume. The prostate gives about 30%.
- Penile and scrotal skin drain to the superficial inguinal nodes. The glans drains to the deep inguinal and external iliac nodes.
- Draw the labels in order from bladder to glans. Add the penile cross-section: two corpora cavernosa and a corpus spongiosum containing the urethra.

Sources

- ROOK V3 — Ch 109 Dermatoses of the Male Genitalia — §Structure and function of the male genitalia; History and examination; Miscellaneous inflammatory dermatoses — pp. 109.3–109.4, 109.25
- IADVL V3 — Ch 125 Overview of Sexually Transmitted Diseases — Anatomy of genital organs — pp. 3406–3410

343. Congenital syphilis: clinical features, diagnosis & management

ASKED 7* · 7-mark scope

Asked in: Jun 2020 · Paper III · Q5(b) (5 marks) · Dec 2020 · Paper III · Q4(a) (5 marks) · Jun 2022 · Paper III · Q3(b) (4 marks) · Dec 2022 · Paper III · Q2(b) (5 marks) · Apr 2023 · Paper III · Q8(a) (4 marks) · Oct 2024 · Paper III · Q2(b) (7 marks) · Jun 2025 · Paper III · Q9(b) (4 marks)

Q: Diagnosis of congenital syphilis. [5]

Q: Clinical manifestations of congenital syphilis. [5]

Q: Management of early congenital syphilis. [4]

Q: Clinical manifestations of: Early congenital syphilis. [5]

Q: Management of congenital syphilis. [4]

Q: Clinical features, diagnosis, management and elimination of congenital syphilis. [7]

Q: Laboratory diagnosis of congenital syphilis. [4]

Diagnosis of congenital syphilis

- **Why it is difficult:** *T. pallidum* is non-cultivable and hard to demonstrate in specimens. Transplacental maternal antibodies confuse serology. Most neonates have no sign of infection at birth.
- **Evaluate** infants born to seropositive mothers if any of the following applies:
 - the mother had no documented treatment at least 4 weeks before delivery
 - the mother received a non-penicillin regimen
 - relapse or reinfection is suspected
- Table 1 sets out the evaluation.

Table 1: Evaluation of a neonate for congenital syphilis

Test	Finding / interpretation
Examination	Stigmata and early signs of CS
Long-bone X-ray	Periostitis; Wimberger sign
CSF examination	Needed to exclude neurosyphilis
Dark-field microscopy / PCR	Exudate from suspicious lesions or fluids
Dark-field microscopy	Identifies <i>T. pallidum</i> in early congenital lesions; sinusoidal organism with typical motility
Direct immunofluorescence / silver stain	Used where dark-field is unavailable, or on biopsy (Warthin–Starry)
PCR	Useful in CS, where serology is hard to interpret; mostly confined to reference laboratories
Serum non-treponemal titre	Infection suggested if the titre is ≥ 4 times the mother's
Specific IgM treponemal test	Positive result suggests neonatal infection

- Compare maternal (at delivery) and infant titres using the same test, preferably in the same laboratory.
- Passively transferred maternal IgG can persist for up to **12 months**.
- WHO 2024 recommends dual treponemal/non-treponemal rapid tests and syphilis self-testing as additional testing options [w].
- Placental or umbilical cord examination helps diagnose prenatal syphilis. The commonest lesion is necrotizing funisitis.
- Histology shows obliterative endarteritis with mononuclear and plasma cell infiltrate.

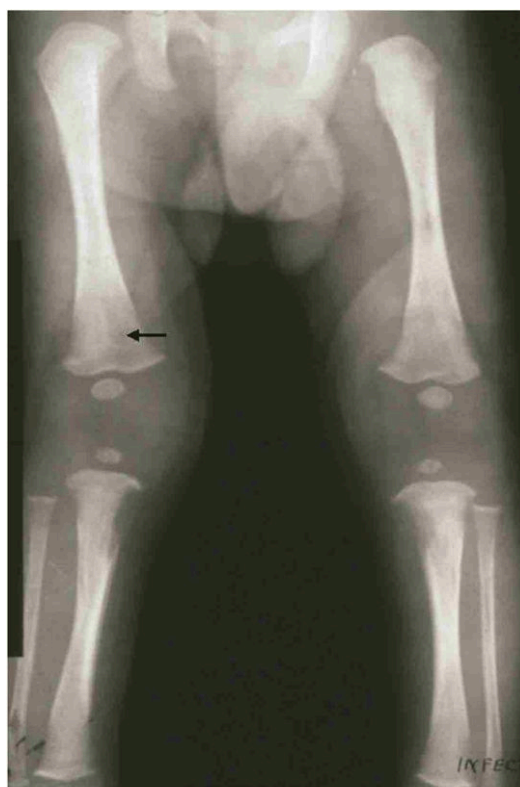


Figure 29.31 — Rook's Textbook of Dermatology 10e V1, p. 29.25. Wimberger sign (arrow) – destruction of the proximal metaphyses of the

Clinical manifestations of congenital syphilis

- **Definition:** transplacental passage of *T. pallidum*, or infection at delivery through maternal genital lesions.
- **Divisions:**
 - **early:** presents within 2 years
 - **late:** presents from age 3 onwards
 - **stigmata:** scars and deformities
- Only severe cases are apparent at birth.

Early congenital syphilis

- Many infants are asymptomatic. Signs appear from week 3–8 in about two-thirds of untreated cases, and nearly always within 3 months.
- Low birth weight, possibly <2500 g.
- **Snuffles** is the commonest and most important sign. It is a profuse serous nasal discharge rich in treponemes, and later causes **saddle nose**.
- **Skin:**
 - coppery red papular rash, mainly on the palms and soles
 - fissured papules at the mouth angles and around the nares
 - papulosquamous eruptions and anal condylomata
 - paronychia
 - rhagades, healing with radiating scars
- **Pemphigus syphiliticus:** bullae on the palms and soles, with highly infectious contents.
- Mucous patches, alopecia, condylomata lata and desquamation.
- **Bone:**
 - periostitis
 - osteochondritis at the lower tibia and fibula, with Parrot pseudoparalysis
 - positive **Wimberger sign**
 - dactylitis
- **Visceral:**
 - hepatosplenomegaly, with jaundice in some
 - pneumonia alba
 - anaemia and thrombocytopenia
 - nephritis and DIC
- **CNS and eye:**
 - meningitis with convulsions and later hydrocephalus
 - chorioretinitis; anterior uveitis is rare
- Napkin dermatitis is a differential. Syphilitic papules are less hypertrophic and less eczematous.

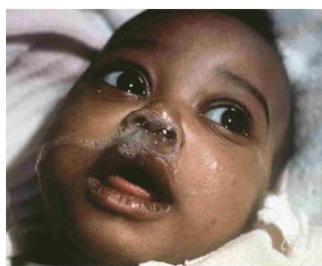
Late congenital syphilis

- Late CS is a consequence of inflammation at sites of early infection.
- **Interstitial keratitis** is the commonest and most serious late lesion.
 - It is usually bilateral and relapsing, with photophobia and pain.
 - It is not influenced by antisyphilitic treatment. Treat with topical or subconjunctival corticosteroids and refer to an ophthalmologist.
- **Eighth nerve deafness** is bilateral in most cases and is uninfluenced by treatment.
- **Clutton joints:** painless knee synovitis, uninfluenced by treatment.
- **Bone:** sabre tibiae, gummata and palatal perforation.
- **Neurosyphilis:** juvenile general paralysis at 6–21 years; tabes is less frequent.
- Cardiovascular syphilis is extremely rare.

Stigmata

- **Hutchinson teeth:** conical or barrel-shaped upper central incisors with a notched free edge.
- **Mulberry (moon) molar:** usually the first molar, with a flat surface and poor cusps.
- **Hutchinson triad:** interstitial keratitis, Hutchinson teeth and eighth nerve deafness.
- **Other stigmata:**
 - rhagades
 - saddle nose and dish face

- Parrot nodes
- "pepper and salt" fundus
- optic atrophy



(a)



(b)

Figure 29.28 — Rook's Textbook of Dermatology 10e V1, p. 29.24. (a) Early congenital syphilis showing serous nasal discharge from



(a)



(b)

Figure 29.37 — Rook's Textbook of Dermatology 10e V1, p. 29.28. Malformations of the secondary dentition in late congenital syphilis. (a)

Table 29.6 Features of congenital syphilis.

	Early features	Late features
Birth weight	Low birth weight: may be <2500 g	
Mucocutaneous	Snuffles	Rhagades
	Maculopapular rash	Gummata
	Pustules	Palatal perforation
	Mucous patches	
	Condylomata lata	
	Vesiculobullous lesions	
	Rhagades	
	Desquamation	
	Alopecia	
Dental		Hutchinson teeth
		Mulberry molars
Laryngeal	Hoarse cry	
Ocular	Chorioretinitis	Interstitial keratitis
	Uveitis	Uveitis
	Glaucoma	Glaucoma
Gastrointestinal	Hepatosplenomegaly	
	Pancreatitis	
Renal	Enteritis	
	Nephritis	
	Oedema and ascites	
Lymphatics	Lymphadenopathy	
Haematological	Thrombocytopenia	
	Anaemia	
	Disseminated intravascular coagulation	
Central nervous system	Aseptic meningitis	Mental delay
	Chronic meningovascular syphilis	Eighth nerve deafness
	Cranial nerve palsies	Convulsive disorders
	Hydrocephalus	Paresis and paralysis
Skeletal	Periostitis	Tabes dorsalis
		Periostitis causing frontal and parietal bossing
	Osteochondritis causing pseudoparalysis	Sabre tibiae, scaphoid scapula
	Osteitis	Thickening of medial part of clavicle
		Short maxilla and protuberant mandible
		Saddle nose
		Clutton joints

Table 29.6 — Rook's Textbook of Dermatology 10e V1, p. 29.24. Features of congenital syphilis.

Management of early congenital syphilis

MANAGEMENT BASIS

Rook's 10e, ch. 29 (treatment ladder for congenital syphilis). International: WHO — Updated recommendations for treatment of *N. gonorrhoeae*, *C. trachomatis* and *T. pallidum* — 2024 [w].

- **Treatment decisions** rest on:
 - maternal syphilis and the adequacy of maternal treatment
 - clinical, laboratory or radiographic evidence in the infant
 - comparison of maternal and infant titres

Table 2: Treatment ladder for congenital syphilis

Line	Group	Regimen
First	Neonate	Aqueous crystalline penicillin G 60 000–150 000 units/kg/day, as 30 000–50 000 units/kg/dose IV every 12 h in the first 7 days of life and every 8 h thereafter, for 10 days
First	Neonate (alternative)	Procaine penicillin G 50 000 units/kg/dose IM once daily for 10 days
First	Older infants and children	Aqueous crystalline penicillin G 0.2–0.3 mega units/kg/day IV, as 50 000 units/kg every 4–6 h for 10 days
Second	Infants ≥30 days	Ceftriaxone 75 mg/kg IV/IM OD for 10–14 days
Second	Older infants	Ceftriaxone 100 mg/kg OD, single dose

- IV therapy is preferred because IM injections are painful.
- Where a case is **not clinically evident** (the infant is normal on examination and the other criteria are met), treatment is optional with close monitoring. The criteria are:
 - infant titre ≤4 times the mother's
 - mother treated in pregnancy, appropriately for the stage
 - treatment given >4 weeks before delivery
 - no maternal reinfection or relapse
- If treated, give benzathine penicillin G 50 000 units/kg as a single IM injection.

- Data on non-penicillin agents are insufficient. If one is used, follow up closely with serology and CSF examination.
- Infants with penicillin allergy should be desensitised if necessary, then given penicillin.
- **Follow-up:**
 - non-treponemal tests every 2–3 months until non-reactive or the titre falls fourfold
 - titres should fall by 3 months and be non-reactive by 6 months
 - if titres are stable or rising after 6–12 months, do a CSF examination and give a 10-day course of parenteral penicillin G

Clinical features, diagnosis, management and elimination of congenital syphilis

- Clinical features, diagnosis and management are as above.

Elimination

- CS is the second commonest cause of preventable stillbirth after malaria. WHO launched a global elimination initiative in 2007.
- The **target** is <50 cases per 100 000 live births.
- **Prevention** depends on prenatal care and timely penicillin treatment.
- **Strategies:**
 - antenatal screening, with repeat screening at 30 weeks for at-risk women
 - point-of-care tests where return for results is uncertain
 - dual syphilis and HIV screening, which is more cost-effective
 - WHO 2024 also recommends STI partner services [wi]
- **Barriers:** unassured benzathine penicillin supply and other practical issues in low-income countries.
- Up to one-third of women attending antenatal clinics are not tested.

PEARLS

- CS is caused by transplacental *T. pallidum*. Early disease presents within 2 years and late disease from age 3.
- Snuffles is the commonest early sign. Wimberger sign marks osteochondritis.
- Diagnose by comparing infant and maternal titres (≥ 4 times suggests infection), IgM, dark-field/PCR, long-bone X-ray and CSF.
- Maternal IgG may persist for up to 12 months.
- Hutchinson triad: interstitial keratitis, Hutchinson teeth and eighth nerve deafness. Keratitis and deafness do not respond to penicillin.
- Treat with 10 days of IV penicillin G; ceftriaxone is second line.
- The elimination target is <50 per 100 000 live births.

Sources

- ROOK V1 — Ch 29 Syphilis and Congenital Syphilis — §Investigations; Follow-up; Congenital syphilis — pp. 29.18, 29.22–29.29
- IADVL V3 — Ch 126 Syphilis — §Syphilis and Pregnancy — p. 3439
- [wi] WHO — Updated recommendations for the treatment of *N. gonorrhoeae*, *C. trachomatis* and *T. pallidum* (syphilis), and new recommendations on syphilis testing and partner services — 2024 — <https://www.who.int/publications/i/item/9789240090767>

Table 3: Monitoring schedule

Stage	Parameter	Frequency or action
Baseline	Consent and ability to comprehend risk	Before starting
Baseline	Pregnancy test (child-bearing potential)	Serum or urine, adequate sensitivity
Baseline	CBC with absolute neutrophil count, HIV RNA	Once
Baseline	Clinical neurological exam, EMG and NCV	Once. SNAP amplitude if history or exam suggests
Follow-up	Pregnancy test, regular menses	Weekly for 4 weeks, then monthly
Follow-up	Pregnancy test, irregular menses	Every 2 weeks
Follow-up	CBC with platelets and absolute neutrophil count	Periodic
Follow-up	Neurological exam	Monthly for 3 months, then 6-monthly
Follow-up	EMG and NCV	Biannually, or each 10 g rise in cumulative dose
Follow-up	SNAP amplitude	Fall of 30% : reduce dose. Fall of 40% : stop

PEARLS

- Thalidomide suppresses TNF- α . It is the best drug for severe, recurrent or steroid-dependent ENL in men and postmenopausal women.
- Start at 400 mg/day. ENL is controlled within 48–72 hours. Taper slowly.
- Never stop it abruptly. Flare-ups, including necrotic ENL, can follow.
- Category X: phocomelia from exposure on days 20–36. Two contraceptives are needed from 4 weeks before to 4 weeks after.
- Neuropathy occurs in 20% in year 1. A fall in SNAP of 30% means reduce the dose, and 40% means stop.
- DVT risk rises with steroid co-administration. Somnolence is reduced by bedtime dosing.

Sources

- IAL — Ch 31 Reactions in Leprosy: Management — pp. 508, 511, 515–517, 519
- [w] NLEP (MoHFW/DGHS) — Operational Guidelines for DPMR — 2007 — <https://clinicalestablishments.mohfw.gov.in/sites/default/files/standard-treatment-guidelines/516.pdf>

431. ENL: pathogenesis, cytokines, investigations & management

ASKED 4* · 10-mark scope

Asked in: Jun 2020 · Paper III · Q7 (10 marks) · Dec 2021 · Paper III · Q6(a) (3 marks) · Dec 2021 · Paper III · Q6(c) (4 marks) · May 2024 · Paper III · Q1(c) (4 marks)

Q: Erythema nodosum leprosum (ENL) a) Current concepts in the etiopathogenesis. [5] b) Management of ENL. [3] c) Newer treatment modalities. [2] [10]

Q: A 35-year-old male presents with recurrent Erythema Nodosum Leprosum (ENL). Enumerate the various investigations in this case. [3]

Q: A 35-year-old male presents with recurrent Erythema Nodosum Leprosum (ENL). Cytokines in ENL. [4]

Q: Management of Erythema Nodosum Leprosum. [4]

Current concepts in the etiopathogenesis of ENL

- **ENL (type 2 reaction)** is a multisystem immune-mediated complication of **LL and BL** leprosy.
- Exact pathogenesis is unclear. It is often considered a **neutrophilic, immune-complex-mediated** condition, with features shared with the Arthus reaction.
 - Complement, proteins and immunoglobulins are deposited in the dermis of ENL biopsies. Whether immune complexes are causal or an epiphenomenon is not clear.
- **Early phase:** monocytes between foamy cells become active macrophages and probably destroy the inert foamy macrophages.

- Released antigens are presented to the immune system, which stimulates CMI. Antigens not engulfed form **immune complexes** with local antibodies.
- **Neutrophils** are the histological hallmark, but direct evidence for their causal role is lacking. They may be recruited by chemokines such as **IL-8**.
- **CD64 (FcγRI) upregulation:** after MDT starts, fragmented intracellular *M. leprae* components upregulate CD64 on neutrophils. This increases proinflammatory cytokine release and explains the higher incidence of ENL after MDT.
- **T-cell role:** CD4⁺ cells rise and surpass CD8⁺ cells, so the CD4/CD8 ratio increases. **Treg** numbers and proportion fall, giving unregulated inflammation.
- **Th17 axis:** IL-17 and TGF-β are raised in ENL lesions.
- **Genetic:** polymorphisms of **NOD2** and **NRAMP1** raise the risk.
- **Autoimmunity:** NCAM expression is increased and NCAM⁺ CD8⁺ cells are found in nerve tissue. CD8⁺ cells cause cytolysis of NCAM-expressing Schwann cells, and **IL-15** (which induces NCAM) is in excess.
- **Risk factors:**
 - Lepromatous leprosy.
 - **BI >4+**: 5.2 times more likely to develop ENL than BI <4.
 - Intercurrent infections, and co-infection with HIV or hepatitis B/C.
 - Trauma or surgery, physical and mental stress, vaccination.
 - Pregnancy, postpartum and lactation.
 - Potassium iodide.
 - Low C4 and high anti-*M. leprae* antibodies at diagnosis.

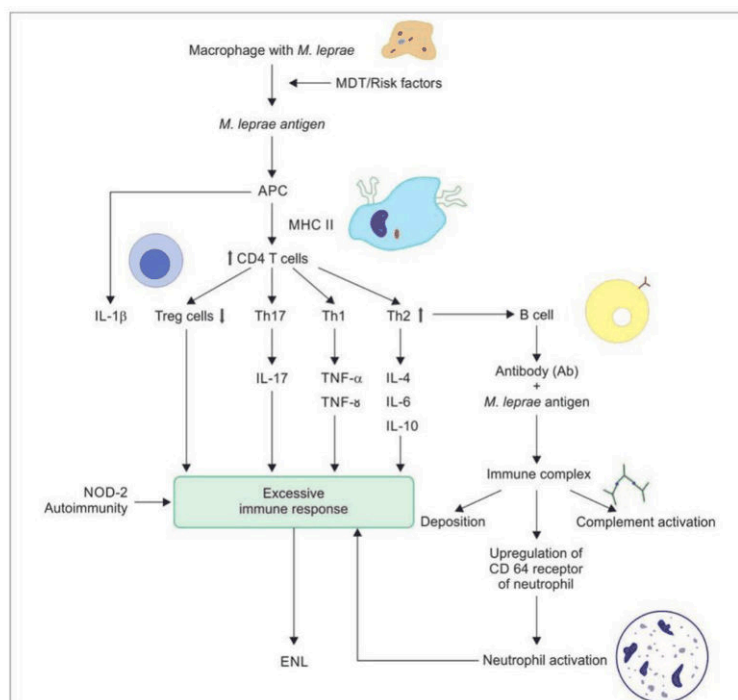


Fig. 30.9 — IAL Textbook of Leprosy 3e, p. 494. The immunopathomechanism of type 2 reaction (T2R).

Cytokines in ENL

Table 1: Cytokines and mediators in ENL

Mediator	Change / role
TNF- α	Raised. Pyrogen causing fever and tissue damage. Level does not correlate with severity but falls after steroids or thalidomide
IFN- γ	Raised
IL-1 β	Raised. A high level may indicate higher risk of reaction after starting treatment
IL-6	Raised. Also a biomarker, along with IL-7
IL-10	Raised
IL-17 (Th17)	Raised in lesions
TGF- β	Increased in lesional skin
IL-8	Chemokine that may recruit neutrophils
IL-15	Induces NCAM expression, so Schwann cell damage
Others	C3 and PDGF-BB. CCL-11 is a potential marker

Gene expression	NOD2 gene, NRAMP1 polymorphism
Cytokines	$\uparrow$ TNF- α , IFN- γ , IL-1 β , IL-10, IL-6, IL-17 $\uparrow$ TGF- β
Others	$\uparrow$ C3, platelet-derived growth factor β polypeptide b (PDGF-BB)
(IL: interleukin; NOD: nucleotide-binding oligomerization domain; NRAMP: natural resistance-associated macrophage protein; T2R: type 2 reaction; TGF: transforming growth factor; TNF: tumor necrosis factor)	

TABLE 30.5 — IAL Textbook of Leprosy 3e, p. 495. Mediators of T2R.

Investigations in a 35-year-old man with recurrent ENL

- **Definition:** recurrent ENL is repeated episodes occurring 28 days or more after stopping ENL treatment. **Chronic ENL** is ENL for 24 weeks or more, needing continuous treatment or with treatment-free gaps of 27 days or less.
- **No specific laboratory test confirms ENL.**

Table 2: Investigations in recurrent ENL

Investigation	Finding / purpose
Clinical tests	Ryrie test (burning pain on stroking the sole). Ellis test (painful wrist squeeze). Naafs criteria: 1 major or ≥ 3 minor
Severity	ENLIST ENL severity scale (pain VAS, fever, lesion number, oedema, nerve tenderness etc.)
Slit-skin smear / BI	Fragmented, granular AFB. High BI is a risk factor
CBC, ESR, CRP	Leukocytosis 20,000–50,000/mm ³ , ESR markedly raised, CRP present, anaemia possible
Urine routine and microscopy	Albuminuria, RBCs, casts to detect kidney involvement
LFT	Raised bilirubin or transaminases indicate hepatic or muscle damage
Skin biopsy	Dilated capillaries, intense neutrophil infiltrate, microabscesses and vasculitis, on a background of LL/BL granuloma. Few, fragmented bacilli
Triggers	Screen for HIV, hepatitis B/C, chronic infections (oral-dental, urinary, sinus), TB, parasites, filariasis
Baseline for thalidomide	CBC with absolute neutrophil count, HIV RNA, neurological examination, EMG/NCV
Research biomarkers	PTX3, $\alpha 1$ -AGP, complement C1q (low), IL-6/IL-7, CD64, anti-LID-1

Clinical examination	• Ryrie test • Ellis test
Hematological parameters	• Complete blood count (leukocytosis, high ESR, and anemia) • Liver function tests • CRP (C-reactive protein) levels • Routine and microscopic urine examination
Histopathological examination	Intense neutrophilic infiltration in dermis, and blood vessels. Bacilli are few and are mostly fragmented and granular
Biomarkers	PTX-3, α 1-AGP, complement C1q, interleukin-6 and -7, CD-64, anti-LID-1 antibody
(ESR: erythrocyte sedimentation rate; LID-1: leprosy IDRI diagnostic; PTX-3: pentraxin-3; AGP: acid glycoprotein)	

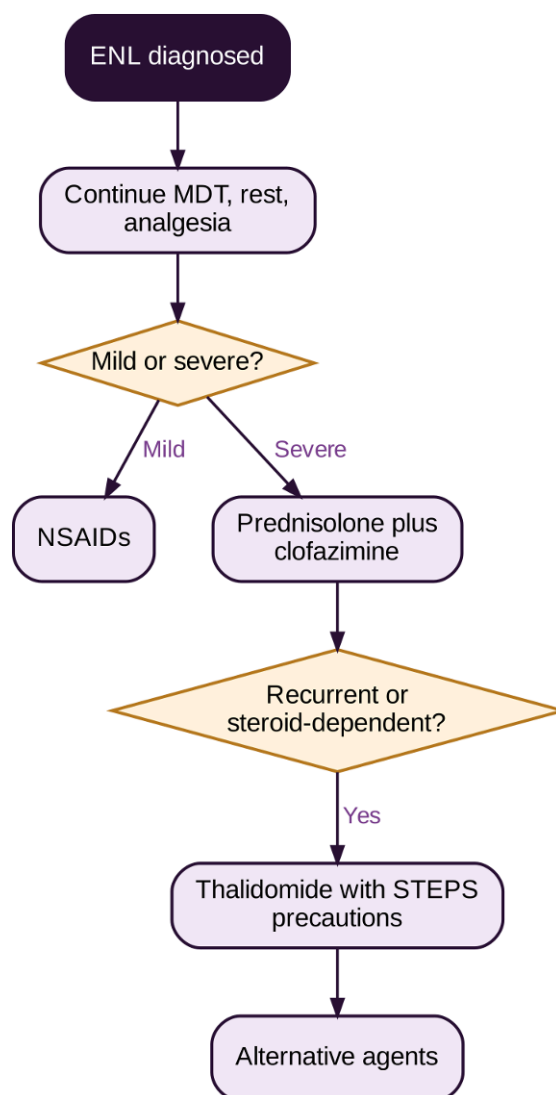
TABLE 30.10 — IAL Textbook of Leprosy 3e, p. 501. Evaluation for type 2 reaction (T2R).

Management of ENL

MANAGEMENT BASIS

NLEP (MoHFW) — National Leprosy Eradication Programme Guidelines (standard treatment guidelines) [w1]; WHO — Leprosy: management of reactions and prevention of disabilities, technical guidance — 2020 [w2]; IAL Textbook of Leprosy 3e, ch. 31.

Flowchart 1: Management ladder for ENL



General and mild disease

- Treat ENL with prednisolone, bed rest, and rest to the affected nerves with splints and analgesics [w1].
- Mild ENL: **NSAIDs** (aspirin, indomethacin, ibuprofen).
- Severe ENL: prednisolone, high-dose clofazimine, thalidomide, with other agents listed.

Corticosteroids and clofazimine

Table 3: India (NLEP) versus WHO regimens

India: NLEP, standard treatment guidelines	International: WHO, technical guidance 2020
Prednisolone 1 mg/kg/day as a single morning dose after breakfast [w1]	Prednisolone 40 mg daily [w2]
After control, taper by 10 mg fortnightly to 20 mg/day, then 5 mg fortnightly until withdrawal [w1]	Taper by 5 mg every fortnight for 24 weeks [w2]
Clofazimine with steroids in every case, 100 mg TDS , tapering to 100 mg after every 12 weeks [w1]	Clofazimine 300 mg/day for 12 weeks, 200 mg/day for 12 weeks, then 100 mg/day for 12–24 weeks [w2]

- For neuritis, prolong prednisolone to **four weeks from 20 mg onwards** [w1].
- Record tablets in the **Prednisolone Card**. Give on an empty stomach with a salt-restricted diet [w1].

Thalidomide

- **Mechanism:** markedly reduces **TNF-α** (represses transcription, inhibits NF-κB) and inhibits neutrophil chemotaxis.

- **Indications:** men and postmenopausal women with difficult ENL, recurrent ENL, steroid-dependent cases, and where steroids are contraindicated.
 - Women of child-bearing age may be treated if the risks are understood and precautions taken.
- **Effect:** response >90%. Suppresses all manifestations in 48–72 hours, with complete resolution in 2 weeks. It is faster than aspirin, clofazimine and pentoxifylline.
- **Monitoring:**
 - Pregnancy test in women of child-bearing potential: weekly for 4 weeks, then monthly.
 - CBC with neutrophil count.
 - Neurological examination monthly for 3 months, then 6-monthly.
 - EMG/NCV: if SNAP amplitude falls 30% reduce the dose, and if 40% stop.
- **STEPS precautions:**
 - Two reliable contraceptives from 4 weeks before until 4 weeks after treatment.
 - Monthly pregnancy tests.
 - Condoms for men.
 - No blood donation and no sharing of tablets.
 - Signed informed consent.
- **Adverse effects:**
 - **Teratogenicity** (phocomelia).
 - Irreversible peripheral neuropathy in 20% in the first year.
 - Thromboembolism/DVT, especially with steroids.
 - Somnolence in up to 11%; give at bedtime.
 - Cutaneous reactions (EM, erythroderma, TEN) in 3%.

Newer treatment modalities in ENL

- **Agents listed for severe or refractory ENL:**
 - Pentoxifylline, azathioprine, cyclosporine, methotrexate and mycophenolate mofetil.
 - Corticosteroid pulse therapy (dexamethasone/betamethasone).
 - **Anti-TNF- α agents, minocycline, apremilast**, and immunotherapy/immunomodulators.
- **Pentoxifylline:** 1200 mg/day in three doses. Slow, with maximum effect in 30–60 days. A useful option in HIV co-infection or when thalidomide and steroids are contraindicated.
- **Azathioprine:** 2–3 mg/kg/day for 6–8 months, as a steroid-sparing drug. Check TPMT before starting.
- **Cyclosporine A:** 7.5 mg/kg/day, promising in acute ENL. No significant steroid-sparing effect in chronic ENL.
- **Methotrexate:** 7.5–15 mg weekly, effective in steroid-resistant ENL.
- **Mycophenolate mofetil:** 1 g/day, maintained at 0.5 g/day for 4–6 months. An option in chronic ENL when steroids are contraindicated.

	Mild	Severe
Type 1 reaction	NSAIDs, e.g., aspirin, indomethacin, ibuprofen, etc.	• Corticosteroids (Prednisolone) • Methotrexate • Cyclosporine • Azathioprine • Mycophenolate mofetil • Tacrolimus (Topical)
Type 2 reaction	NSAIDs, e.g., aspirin, indomethacin, ibuprofen, etc. Colchicine	• Corticosteroids (Prednisolone) • Clofazimine (high dose) • Thalidomide • Pentoxifylline • Azathioprine • Cyclosporine • Methotrexate • Mycophenolate mofetil • Corticosteroid pulse therapy (dexamethasone/betamethasone) • Anti-TNF-α agents • Minocycline • Apremilast • Immunotherapy and immunomodulators

(NSAIDs: nonsteroidal anti-inflammatory drugs; TNF: tumor necrosis factor)

TABLE 31.1 — IAL Textbook of Leprosy 3e, p. 508. Drugs used in the management of type 1 and type 2 lepra reactions.

PEARLS

- ENL is a type III hypersensitivity (neutrophilic, immune-complex) reaction of **LL/BL**, commonly in the first year of MDT.
- Hallmark: crops of tender, evanescent pink nodules on the outer thighs, legs and face, with fever. Original lesions are unchanged.
- Histology: neutrophilic infiltrate on a lepromatous granuloma, with fragmented bacilli.
- Key cytokine: **TNF- α** . It falls with thalidomide and steroids.
- Drug of choice in recurrent or steroid-dependent ENL is **thalidomide**. Exclude pregnancy first, and monitor for neuropathy.
- NLEP: prednisolone 1 mg/kg/day plus clofazimine, with the 100 mg TDS start tapering after every 12 weeks [w1].
- Exam trap: ENL must be distinguished from erythema nodosum. Erythema nodosum is pretibial, usually has up to 10 lesions, and is common in young women.

Sources

- IAL — Ch 30 Reactions in Leprosy: Pathogenesis and Clinical Features — pp. 493–496, 500–501
- IAL — Ch 31 Reactions in Leprosy: Management — pp. 508, 515, 517
- [w1] NLEP (MoHFW) — National Leprosy Eradication Programme Guidelines (standard treatment guidelines, primary care) — year not stated — <https://clinicaestablishments.mohfw.gov.in/sites/default/files/standard-treatment-guidelines/516.pdf>
- [w2] WHO — Leprosy/Hansen disease: management of reactions and prevention of disabilities, technical guidance — 2020 — <https://afro.who.int/health-topics/leprosy>

442. WHO MDT: current regimens, modifications & newer drugs

ASKED 2* · 5-mark scope

Asked in: Dec 2020 · Paper III · Q10(b) (4 marks) · Oct 2023 · Paper III · Q3(b) (5 marks)

Q: Latest modification in MDT of leprosy. [4]

Q: Newer drug regimens in leprosy. [5]

Latest modification in MDT of leprosy

MANAGEMENT BASIS

India: NLEP — Revised Classification and Treatment Protocol for leprosy (revised 2023; nationwide from 1 April 2025) [w1]. International: WHO — Guidelines for the diagnosis, treatment and prevention of leprosy — 2018 [w2].

- **3-drug MDT** (rifampicin, dapsone, clofazimine) is now used for **both PB and MB** cases [w1].
- Before 1 April 2025, India's PB regimen was 2-drug (rifampicin + dapsone, 6 months) [w1].
- WHO gives the same 3-drug regimen to all patients: **6 months for PB, 12 months for MB** [w2].
- Revised NLEP classification: **PB** is 1–5 skin lesions, no nerve involved, smear negative. **MB** is more than 5 lesions, one or more nerves involved, or smear positive [w1].
- Adult PB, 6 months: dapsone 100 mg daily; clofazimine 300 mg monthly + 50 mg daily; rifampicin 600 mg monthly [w1].
- Child 10–14 years PB, 6 months: dapsone 50 mg daily; clofazimine 150 mg monthly + 50 mg alternate days; rifampicin 450 mg monthly [w1].
- Child under 10 years or 20–40 kg PB, 6 months: dapsone 2 mg/kg daily; clofazimine 100 mg monthly + 50 mg twice weekly; rifampicin 10 mg/kg monthly [w1].
- MB adult course is 12 months. Children under 40 kg need single-formulation drugs, as no blister packs exist for them [w1].
- Earlier evolution: dapsone monotherapy → WHO-MDT 24 months (FDT-24) → **FDT-12 for MB and FDT-6 for PB** (1997).
- Single-dose ROM for single-lesion leprosy was withdrawn by WHO and NLEP for operational reasons.
- Rifampicin-resistant leprosy (WHO): at least two second-line drugs (clarithromycin, minocycline or a quinolone) plus clofazimine daily for 6 months, then clofazimine plus one of these drugs for 18 months [w2].

Table 1: Old vs current NLEP regimen

Feature	Before 1 April 2025 (NLEP)	Current (NLEP 2023 revision) [w1]
PB drugs	Rifampicin + dapsone	Rifampicin + dapsone + clofazimine
PB duration	6 months	6 months
MB duration	12 months	12 months

Newer drug regimens in leprosy

- **ROM** (rifampicin, ofloxacin, minocycline) single dose was used for single-skin-lesion PB leprosy, then withdrawn.
- In a 1,526-patient trial of single-dose ROM in PB with 2–5 lesions, relapse was higher than with WHO-PB-MDT (1.13 vs 0.35 per 100 person-years).
- **ROM-12 for MB and ROM-6 for PB** (monthly) are listed as recommended trial regimens.
- **PMxM-1**: rifapentine 900 mg or rifampicin 600 mg + moxifloxacin 400 mg + clarithromycin 1,000 mg (or minocycline 200 mg), all once monthly under supervision. This was recommended at the 2008 Hyderabad workshop.
- **MxRM**: moxifloxacin 400 mg + rifampicin 600 mg + minocycline 200 mg, monthly for 12 months in MB and 6 months in PB. **MxRMC** adds clofazimine.
 - Adding clofazimine did not appear to change the occurrence of reactions.
- **Moxifloxacin** is the most powerful bactericidal agent against *M. leprae*, acting by inhibiting DNA gyrase.
- Other agents: clarithromycin (bactericidal), minocycline (bacteriostatic), rifapentine and rifabutin.

- **Bedaquiline** is bactericidal in mice, but no human trials have been done.
- **Linezolid** is weakly bactericidal in mice; **pretomanid** has low activity against *M. leprae*.
- Special situation, MB patients who cannot take rifampicin (adults): clofazimine 50 mg daily + ofloxacin 400 mg + minocycline 100 mg (or clarithromycin 500 mg) daily for 6 months.
- Parenteral clofazimine does not produce skin pigmentation.

Regimen	Classification	Duration
1. DDS monotherapy	MB PB	Life-long (continuous) 10 years
2. WHO-MDT (21 days of intensive therapy)	MB	24 months or till skin smear negativity
3. WHO-MDT (modified)	MB	24 months or till skin smear negativity
4. WHO-MDT (FDT-24)	MB	24 months
5. WHO-MDT (FDT-12)	MB	12 months
6. WHO-MDT (FDT-6)	PB	6 Months
7. ROM-12	MB	12 months (monthly)
8. ROM-6	PB	6 months (monthly)
9. RO-28	PB/MB	28 days (daily)
10. ROM-1	SSL	1 day (single dose)
11. PMXM-1	MB	1 day (single dose)
12. MXRM – 12	MB	Intermittent (monthly)
13. MXRM – 6	PB	Intermittent (monthly)

(DDS: dapsone; FDT: fixed duration treatment; M: minocycline; MB: multibacillary; MDT: multidrug therapy; MX: moxifloxacin; O: ofloxacin; P: rifapentine; PB: paucibacillary; R: rifampicin; SSL: single skin lesion; WHO: World Health Organization)

Note: No. 1–4 and 9: Not in use; No. 5 and 6: Universally recommended by WHO; No. 7, 8, 12, and 13: Recommended trials; No. 10: In use for some time, now given up. No. 11 PM₁M-1 -MB-1 day (single dose) (Experimentally tested in mice using mouse footpad system and recommended for clinical trials).³⁴

TABLE 33.3 — IAL Textbook of Leprosy 3e, p. 549. Evolution of treatment regimens in leprosy.

PEARLS

- Current NLEP: 3-drug MDT for PB (6 months) and MB (12 months).
- PB = 1–5 lesions, no nerve, smear negative; MB = more than 5 lesions, nerve involved, or smear positive.
- Moxifloxacin is the most bactericidal drug against *M. leprae*.
- Single-dose ROM was withdrawn because of higher relapse and operational problems.
- Bedaquiline has no human leprosy trials.

Sources

- IAL — Ch 33 Chemotherapy: Development and Evolution of WHO MDT and Newer Treatment Regimens — pp. 548–550
- IADVL V3 — Ch 144 Leprosy: Multidrug Therapy and Management of Reactions — p. 3847
- [w1] NLEP (MoHFW/DGHS) — Revised Classification and Treatment Protocol for leprosy — 2023 — <https://dghs.mohfw.gov.in/nlep.php>
- [w2] WHO — Guidelines for the diagnosis, treatment and prevention of leprosy — 2018 — <https://www.who.int/publications/i/item/9789290226383>

- **Follicular pustules** appear at 2–3 months when grafts are badly placed.
 - Causes: more than two follicular units in one slit (piggybacking), or damaged hair fragments.
 - They are usually sterile and self-limiting. Persistent collections are drained.
- **Loss of grafts over time in primary cicatricial alopecia:** transplanted hair is not immune to the disease process.
- **Misplaced hairline:** surgery is not usually done under 25 years because later balding progression makes the hairline look unnatural.
- **Prevention:**
 - Operate only by trained physicians.
 - Use a skilled team and a stereomicroscope for graft dissection.
 - Give proper postoperative care.

PEARLS

- The most common complications are minor: bleeding, infection, oedema and shock loss.
- Strip method: linear occipital scar. FUE: higher transection rate and a shaved donor area.
- Shock loss is a temporary anagen effluvium.
- Piggybacking and damaged grafts cause postoperative sterile pustules.
- Trichophytic closure reduces a wide donor scar.
- Poor patient selection (age under 25, progressive loss, scarring alopecia) leads to long-term failure.

Sources

- ROOK V3 — Ch 87 Acquired Disorders of Hair — §Surgery — p. 87.97
- IADVL V3 — Ch 110 Hair Transplantation — pp. 3209, 3213–3214

PART 22

Aesthetic Dermatology

481. Botulinum toxin: mechanism & uses

ASKED 3* · 6-mark scope

Asked in: Jun 2020 · Paper IV · Q8(a) (6 marks) · Dec 2021 · Paper IV · Q5(c) (3 marks) · Apr 2023 · Paper IV · Q2(b) (5 marks)

Q: Role of botulinum toxin in dermatology. [6]

Q: Mechanism of action of Botulinum Toxin. [3]

Q: Therapeutic uses of Botulinum toxin in dermatology. [5]

Role of botulinum toxin in dermatology

- **Botulinum toxin (BTX)** is a neurotoxin that blocks acetylcholine release at the neuromuscular junction and so inhibits muscle contraction.
- The first dermatological use was **glabellar frown lines**. This was noticed during strabismus treatment and reported by Carruthers and Carruthers in 1992.
- It reduces **hyperfunctional, muscle-induced lines (dynamic wrinkles)**. This was confirmed in double-blind placebo-controlled studies.
- The FDA approved Ona BTX-A for glabellar lines in 2002.
- Botox was also approved for crow's feet by the FDA in October 2013.
- Severe **axillary hyperhidrosis** is a second approved dermatological indication. It is supported by evidence-based multicentre studies.
- Its role in dermatology can be grouped as:
 - Aesthetic: dynamic rhytides, brow shaping, facial contouring and reshaping.
 - Autonomic: focal hyperhidrosis and gustatory sweating.

- Combination: with dermal fillers for folds and volume loss, with radiofrequency for tightening, and with lasers for rejuvenation.
- Products approved for facial lines are Botox, Dysport and Xeomin (all type A). Only Botox is approved for hyperhidrosis. Myobloc/NeuroBloc (type B) is not approved for facial lines.
- No topical BTX is approved yet. Early topical studies in hyperhidrosis and facial lines were not confirmed.
- The first and most critical step is accurate examination at rest and at maximum contraction, with photographs.
- The therapeutic window for cosmetic patients is narrower than for medical use, so a **conservative approach** is best.
- Indian practice (IADVL Dermatosurgery Task Force) [w1]:
 - Any qualified dermatologist may give BTX after adequate training, and it can be given in a minor procedure room.
 - Consent should state the toxin type, expected longevity and possible complications.
 - Pre- and postoperative photographs are recommended.
 - Dose depends on area, muscle mass and gender.
 - Beginners should treat the upper third of the face first, and the middle and lower face only after adequate experience.

Mechanism of action of Botulinum Toxin

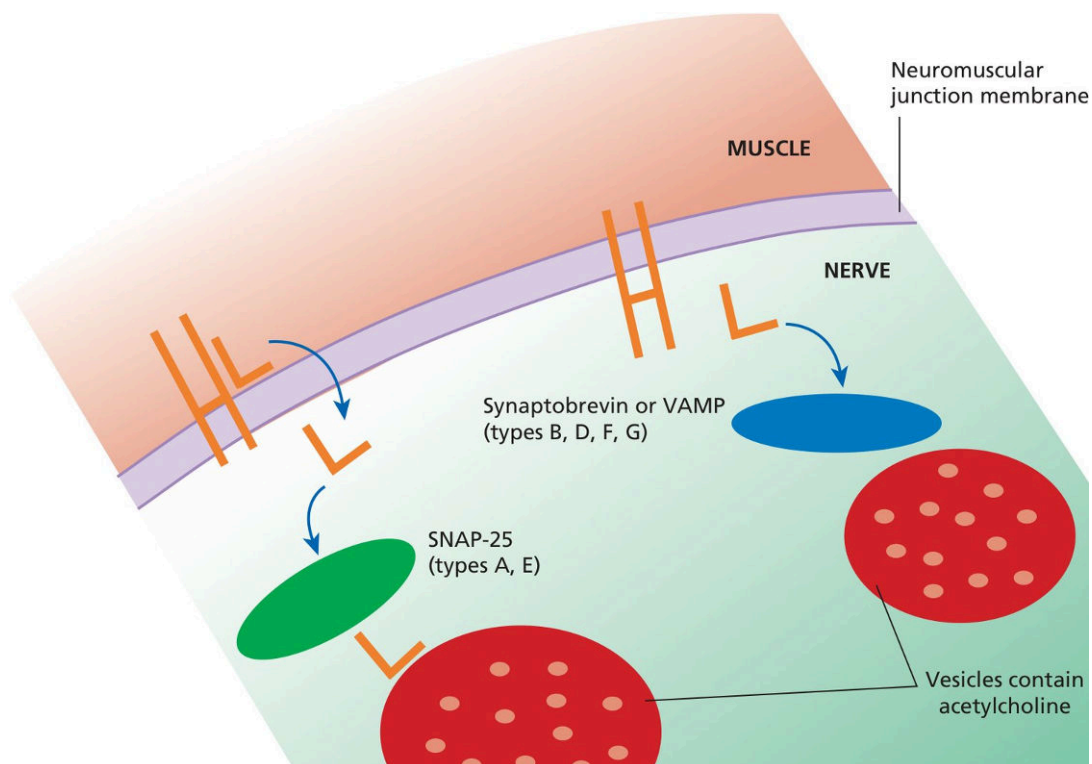
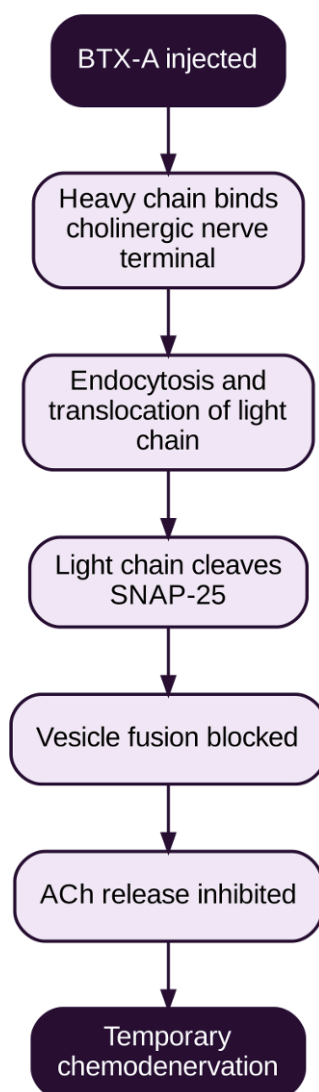


Figure 159.1 — Rook's Textbook of Dermatology 10e V4, p. 159.2. Different botulinum toxins block

- BTX acts at the neuromuscular junction by **blocking release of acetylcholine** from the nerve endplate. This inhibits muscle contraction.
- The core molecule is a **150 kDa protein with three domains**. Accessory neurotoxin-associated proteins (NAPs) vary by serotype.
 - **Binding domain:** docks on specific neuronal surface receptors.
 - **Translocation domain:** lets the catalytic domain enter the cell.
 - **Catalytic domain:** binds specific intracellular proteins and blocks neurotransmitter release.
- Target proteins differ by type:
 - **BTX-A** binds intracellular **SNAP-25**, which prevents exocytosis of ACh from the synaptic vesicle.
 - **BTX-B** binds **synaptobrevin (VAMP)**, which also prevents ACh transport across the membrane.
 - Types A and E act on SNAP-25. Types B, D, F and G act on synaptobrevin/VAMP.
- Chain structure: the single-chain toxin (150 kD) is converted to a **dichain molecule**, held by a disulfide link. This conversion is essential for activity.
 - **Light chain (~50 kD):** zinc-associated endopeptidase that cleaves SNARE proteins.

- **Heavy chain (~100 kD):** the C-terminal half binds and the N-terminal half translocates.
- Three steps of action: binding, internalisation (receptor-mediated endocytosis, energy dependent) and inhibition of transmitter release.
- Autonomic and smooth muscle effect: BTX inhibits transmitter release at postganglionic parasympathetic cholinergic fibres. The same mechanism explains the effect in hyperhidrosis.
- Pain disorders: SNARE proteins are likely involved in release of substance P and glutamate from nociceptive C fibres. This explains its use in chronic migraine.
- The effect is temporary because **new synaptic proteins are synthesised**, ACh release is re-established and muscle activity returns.
- Onset is 12–72 hours, with peak effect at 1–3 weeks and a plateau for 2–3 months. Reinjection is usually needed every 4–6 months.

Flowchart 1: Pathway of BTX-A action



Therapeutic uses of Botulinum toxin in dermatology

Product (trade name)	Toxin type	Molecular weight (kDa)	pH	Approved for facial etc. lines	Approved for hyperhidrosis	Approved for some medical indications, e.g. cervical dystonia, blepharospasm
Botox ^a	A	900	7	Yes	Yes	Yes
Dysport	A	500–900	7	Yes	No	Yes
Xeomin	A	150	7	Yes	No	Yes
Myobloc/NeuroBloc	B	300–500	5.6	No	No	Yes

^a Botox was also approved for crow's feet by the US Food and Drug Administration, October 2013.

Table 159.2 — *Rook's Textbook of Dermatology 10e V4, p. 159.3. Some characteristics of the botulinum neurotoxins.*

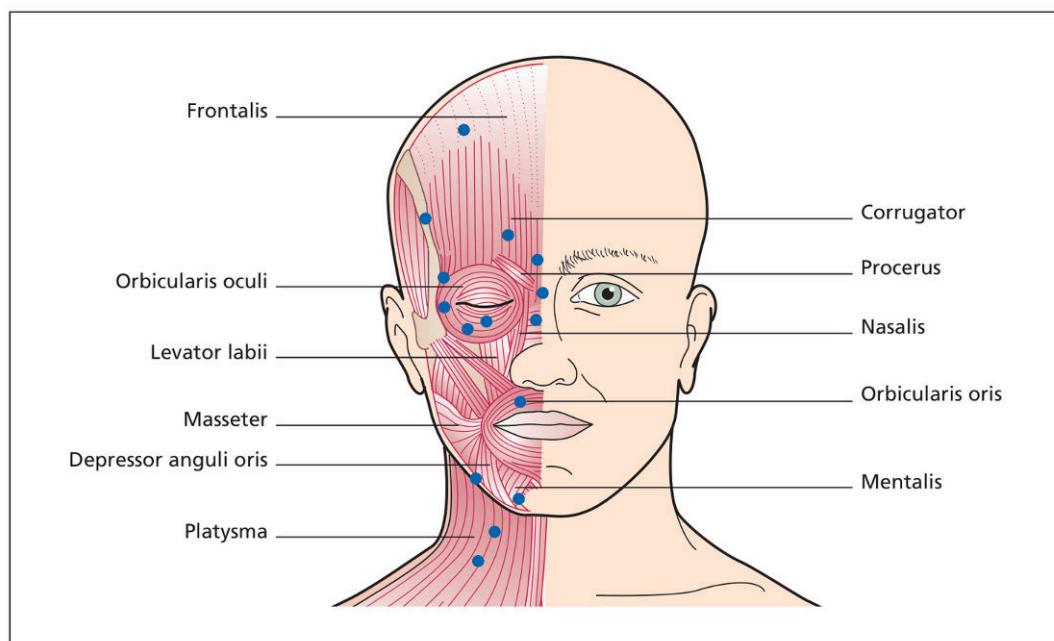


Figure 159.2 — Rook's Textbook of Dermatology 10e V4, p. 159.3. Idealised diagram of the facial muscles

Aesthetic uses

Table 1: Aesthetic indications by site

Site	Target muscle	Indication
Glabella	Corrugator, procerus	Frown lines, brow depression
Forehead	Frontalis	Horizontal lines; use small doses
Brow	Corrugator, procerus, superolateral orbicularis oculi	Lateral brow lift
Lateral canthus	Lateral orbicularis oculi	Crow's feet; 2–4 injections per side, 1–2 cm lateral to the bony rim
Mid face	Nasalis, depressor septi nasi, levator labii superioris alaeque nasi	Nasal lines, nasal tip droop, gummy smile
Lower face	Depressor anguli oris	Downturned mouth corners
Upper lip	Orbicularis oris	Smoker's lines; may combine with HA filler
Chin	Mentalis	Puckered or pebbled chin
Jaw	Masseter	Masseter hypertrophy, reshaping
Neck	Platysma	Platysmal bands

- The frontalis is the main brow elevator. The corrugator and procerus are brow depressors. Brow depressor/elevator balance is key to upper face treatment.
- Glabellar injection: usually 1–2 sites in the procerus and 2 in each corrugator.
- Example dose: 4 Ona BTX-A units per site for a symmetrical glabella and forehead.
- Men generally need **higher doses and more injection sites**. A male forehead example used a total of 36 Ona units.
- Infraorbital lines can be reduced by very small, carefully placed doses.
- Other cosmetic uses: marionette lines and the Nefertiti lift for jawline definition. The Nefertiti lift uses 15–20 U in total.
- Typical IADVL doses:
 - Forehead: 5–7 U in females and 7–12 U in males.
 - Crow's feet: 8–10 U per side in females and 10–12 U per side in males.
- Reconstitution (IADVL): 2.5 mL of 0.9% non-preserved saline in a 100 U vial gives 4.0 U/0.1 mL. Higher dilution means more diffusion and shorter action.
- Ona 1 U is roughly equal to Inco 1.25 U and Abo 3 U, but each is a separate drug.

Medical and other dermatological uses

- Severe **axillary hyperhidrosis** is an approved indication.
- Autonomic uses: focal hyperhidrosis, gustatory sweating, hyperlacrimation and sialorrhoea.
- **Painful hypertrophic scars** are a listed non-cosmetic indication.
- Non-dermatological uses include blepharospasm, cervical dystonia, spasticity, migraine and overactive bladder.
- Palmar hyperhidrosis: caution in those needing fine hand control, such as jewellers and musicians.

Adverse effects and contraindications

- Serious effects are uncommon because the doses are low. Adverse effects are usually mild and transient. The main causes are poor technique, wrong dose and wrong site.
 - Bruising and pain. Postinflammatory hyperpigmentation after bruising is a higher risk in darker phototypes.
 - Persistent headache, rarely.
 - **Brow ptosis or eyelid ptosis**, from a high dose in the lower lateral or mediolateral forehead.
 - Facial asymmetry from unequal effect.
 - Rarely paraesthesia, diplopia, dry eyes, dysphagia and dysarthria.
- Side effects are more frequent in older patients.
- **Contraindications:** myasthenia gravis or other neuromuscular disorder, and pregnancy and breastfeeding.
- Oral erythromycin and zinc can increase the response.
- Aminoglycosides potentiate the effect, and serotypes are not interchangeable. [w]
- **Acquired resistance** to BTX-A is rare and not always antibody related. It can be managed with **BTX-B**, which acts via a different synaptic protein.

PEARLS

- BTX-A cleaves **SNAP-25**. BTX-B cleaves **synaptobrevin (VAMP)**.
- Units are **not interchangeable**. Ona 1 U is roughly equal to Inco 1.25 U and Abo 3 U, but treat each as a separate drug.
- Frontalis overdose causes **brow ptosis**. The frontalis is the main brow elevator.
- The effect is temporary because new synaptic proteins are synthesised.
- Dermatological approvals are glabellar lines, crow's feet and axillary hyperhidrosis.
- Assess the patient at rest and at maximal contraction, with photographs.

Sources

- ROOK V4 — Ch 159 Aesthetic Uses of Botulinum Toxins — pp. 159.2–159.9
- IADVL V3 — Ch 105 Basics of Injectables — pp. 3077–3084, 3092
- [w] IADVL Dermatosurgery Task Force — Guidelines on the use of botulinum toxin type A (IJDVL 2008;74 Suppl:S13–22) — 2008 — <https://ijdv.com/guidelines-on-the-use-of-botulinum-toxin-type-a/>

482. Fillers: complications

ASKED 2* · 5-mark scope

Asked in: Dec 2021 · Paper IV · Q7(c) (3 marks) · Apr 2023 · Paper IV · Q2(a) (5 marks)

Q: Complication of fillers. [3]

Q: Complications of soft tissue fillers. [5]

Complications of fillers (soft-tissue fillers)

- **Overall risk:** no injectable filler is free of adverse reactions.
- **Onset:** early, within hours to days, or delayed by weeks to years.
- **HA fillers:** hyaluronidase can reverse overcorrection, nodules and inflammation. It is essential in vascular occlusion or embolisation.
- **Non-HA fillers:** all other biodegradable fillers lack an antidote.

- **Permanent fillers:** the product stays implanted. Immune reactivity can change with time, causing delayed large granulomas. These are much harder to treat than HA granulomas.

Table 1: Complications of soft-tissue fillers

Group	Complication	Key point
Early, injection site	Erythema, oedema, itching, bruising	Common and mild
Early, technique-related	Infection or abscess	Non-sterile technique; disinfection is mandatory
Early, technique-related	Overcorrection, asymmetry, haematoma	More frequent with rushed or high-pressure injection
Early, technique-related	Bumps or sausage-like lines	High G-prime filler placed too superficially; HA is corrected with hyaluronidase
Early, vascular	Arterial occlusion: blanching, severe pain, necrosis, blindness	Emergency
Late	Nodules	Immune-response nodules after HA or non-HA fillers
Late	Granulomas	Permanent fillers may cause them years later
Late	Persistent erythema, lumping or clumping	
Late	Infection	Atypical mycobacteria
Hypersensitivity	Allergy	Bovine collagen reactions in about 3%

Avoidable causes

- **Fillers without an antidote:** intravascular injection cannot be dissolved emergently.
- **Poor anatomical knowledge:** needed to avoid asymmetry and arterial occlusion. This includes muscles, fat compartments, ligaments, nerves, vessels and bone.
- **Excess volume:** more than 3 mL per site in the cheeks gives very visible changes.
- **Products without good clinical data:** raise the risk of early reactions such as abscess.

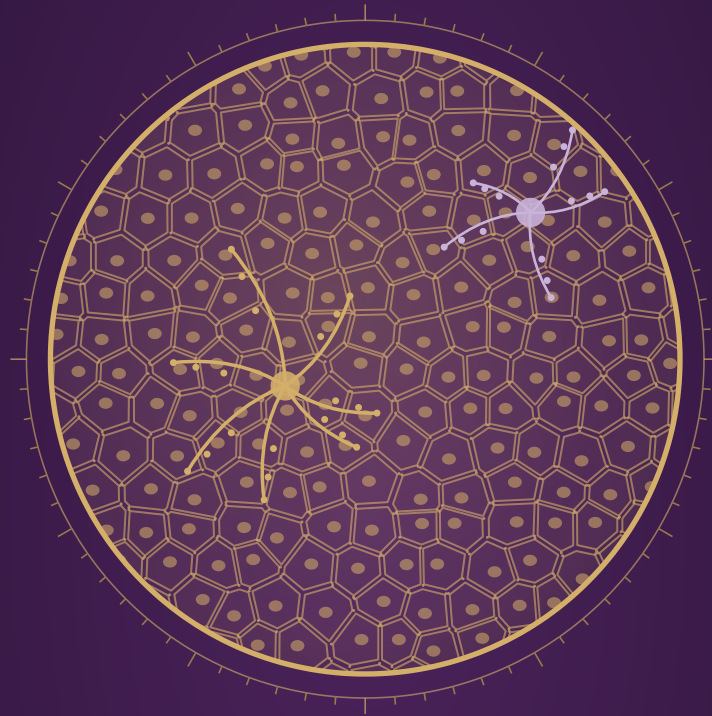
Points by filler type

- **Vycross HA products:** some evidence suggests more inflammatory reactions.
- **Hyaluronidase:** may itself cause redness, oedema, pruritus, pain, and immediate or delayed allergy.
- **Calcium hydroxylapatite:**
 - Lip nodules: 7 of 90 patients had persistent visible mucosal nodules. Four needed intervention such as surgical extrusion.
 - Occlusion: local necrosis or blindness has been reported.
- **Bovine collagen:** a further 1.3% reacted despite a negative skin test.
- **DermaLive (permanent):** highest risk of adverse reactions: nodules, abscesses and ulceration.
- **Skin types IV–VI:** reaction rates are similar. Postinflammatory hyperpigmentation is more likely after events such as abscess.

Treatment of immune-response nodules

- **HA filler, first line:** hyaluronidase around or in the nodule.
- **Second line:** intralesional steroid and/or 5-fluorouracil, or pulse oral prednisolone. Doxycycline 100 mg twice a day for 3 weeks may be added.
- **Non-HA filler:** immunomodulation is first line, and methotrexate 10–15 mg once a week is an option.
- **Third line:** excision, or diode or CO2 laser for non-HA granulomas.

Arterial occlusion: treatment



DNB DERMATOLOGY, VENEREOLOGY & LEPROSY

Solved Previous-Year Questions

Questions from previous DNB DVL theory papers, answered in exam-ready form and grouped by how often it has been asked. Answers are sized to the marks, carry drawable schematics for diagram questions, and are anchored to Rook's, IADVL and the IAL Textbook of Leprosy, with Indian programme and IADVL guidance placed first and every source listed under the answer.

THE THREE-VOLUME SERIES

- VOL 1** Basic Science, Diagnostics & Therapeutics · Parts 1–6
- VOL 2** Clinical Dermatology · Parts 7–16
- VOL 3** Venereology & HIV, Leprosy, Dermatosurgery & Aesthetics, Recent Advances · Parts 17–23

REVIEWED BY

Dr Soumik Dey
DNB (Dermatology)

Dr Sirsa Ghosh
DNB (Dermatology)



CLINICA
PUBLICATION

TO ORDER, VISIT

www.clinicapublication.com

Free sample · Updated till October 2026