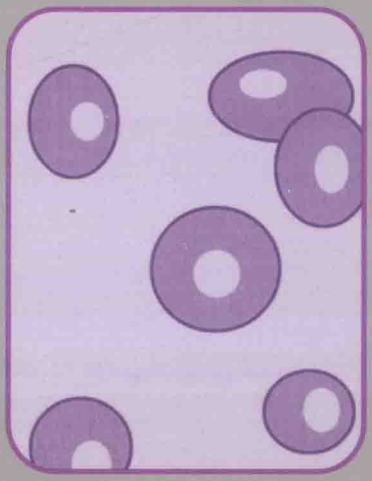
DERMATOLOGY

QUICK GLANCE



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To my life partner, Kishwar, my children, Laila and Jamal,
and my pillars of support, Aboo, Mom, and Tehmina.
—Saeed

In loving memory of my late father, Aleem, my mother,
Kulsum, my wife, Laura, and our children, Afnaan and Danish.
—Abrar

FOREWORD

The value of the printed word, whether in books or journals, has evolved greatly during the past decade. A new edition of *Harrison's Principles of Internal Medicine* is published every three and a half years; the current edition is also available on the Internet, where it is updated daily. Full text articles published in medical journals can also be read on the Internet, often prior to publication. Search engines such as PubMed from the National Library of Medicine can gather references within a matter of seconds on limitless subjects.

What then is the value of a new book in dermatology in this era of electronic publishing? The old adage "A picture is worth a thousand words" certainly is true for dermatologic textbooks. *Dermatology Quick Glance* by Jaffer and Qureshi demonstrates that tables and lists can be worth a thousand words. This new dermatology textbook summarizes the entire discipline of dermatology in fewer than 300 pages, providing easy access to thousands of factoids. Drs. Jaffer and Qureshi have compiled the search for a thousand and one subjects, which we all may have had in our memory banks at one point but have quickly forgotten after board or recertification examinations.

Who will find *Dermatology Quick Glance* of value? Individuals at the beginning a their careers, medical students, dermatology residents, and those studying for Dermatology Board Examinations and recertification. The book is also informative to leaf through, providing new associations, both new and/or forgotten. This presentation of factoids we hope will be updated in subsequent editions.

Richard Allen Johnson, MDCM

PREFACE

Dermatology Quick Glance is our collection of teaching materials and practical tidbits gathered over the years at the Boston area teaching hospitals affiliated with the medical schools of Harvard University, Boston University, and Tufts University.

For all those primary care providers on the front lines: you are aware that skin problems make up more than one-third of patient complaints. This book was written to help answer questions at the point of care and make dermatology as much fun and as exciting as we know it to be. It is meant to serve as a versatile, concise companion and to give providers that extra edge in providing complete care to their patients on a daily basis. It serves the needs of a wide audience:

- Medical students
- Residents and fellows of all specialties
- Nurse practitioners
- Physician assistants
- Nurses
- Emergency room physicians
- Family practitioners
- Pediatricians
- Internists

In particular, dermatology residents, dermatopathologists, and practicing dermatologists will use this book as a "peripheral brain" that allows rapid access both to simple facts and in-depth information. The format makes the book's contents readily accessible; it should prove functional in both a clinical environment and in studying for board examinations.

There is no one dermatology text like this one. The book is divided into 12 chapters that cover major topics in alphabetical order. Two chapters, "Infections and Parasites" and "Dermatopharmacology," are subdivided to facilitate quick reference. The text includes useful tables, illustrations, and flowcharts to explain complicated ideas in a simple, easy-to-understand fashion. Because of the wide variety of sources behind each component, references have been left out for the sake of brevity. Standard dermatologic abbreviations are utilized to present the information in a succinct manner.

We hope that you will find this book to be a valuable resource and guide. We will continue to strive to update and improve the information presented herein.

ACKNOWLEDGMENTS

There is one person who deserves more credit for the production of this work than any other: my wife, Kishwar Bano, who stuck with me through the worst and best at MIT, Harvard, UCLA, Boston University, and Tufts University. Thank you also to the people who first opened my eyes to the wonderful world of medicine and dermatology: my father, Dr. Shahnawaz S. Jaffer, Dr. Harley Haynes, and Dr. Richard Johnson. None of this would have been possible without the guidance and motivation provided by my mother, Aqueela Jaffer, and my sister, Tehmina Jaffer. Thanks to all my colleagues who have inspired and educated me throughout the years: Dan Loo, Abrar Qureshi, Vandana Chatrath, Rana Shahab, Mehran Nowfar-rad, Vince Afsahi, and Soma Wali.

Saeed N. Jaffer

There are many people who have contributed to my training and teaching from the Aga Khan University and Harvard Medical School. I simply cannot thank everyone. Saeed has been a true friend, colleague, and coauthor. My late father, Dr. Aleem S. Qureshi, a dermatologist and educator, was my best friend. He would have been proud of this work. For my mother, Dr. Kulsum Aleem: I truly appreciate all you have done. This book would not have been possible without my lovely wife Laura's outstanding strength, courage, and patience. For my sister Jazibeh and brothers Wasif and Tabarak: thank you for being there. Dr. Ethan Lerner has been like a father to me; I am grateful that he believed in me at a time when few others did. Dr. Harley Haynes is a role model whom I will always aspire to be like but will probably never be. Dr. Michael Bigby: thank you for instilling in me the thirst to learn more about evidence-based medicine.

Abrar A. Qureshi

SPECIAL MENTION

It is rare when a mentor comes along who significantly changes the lives of many pupils and colleagues at the same time. Dr. Daniel Loo is such an individual. His untiring commitment to teaching is the main inspiration behind the completion of this text.

We should like to credit, acknowledge, and thank Dr. Loo for his teaching. Although almost everyone completing dermatology residency compiles a list of factoids for studying, Dr. Loo helped initiate this project by putting these down on paper. In fact, a number of mnemonics incorporated in this book have his stamp of ingenuity on them. Although the final product presented here is an altogether different version, we are indebted to Dr. Loo for his tremendous guidance and support.

Saeed and Abrar

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CHAPTER

1

BASIC SCIENCE AND BLISTERING DISEASES

β-CATENIN

- Intracellular calcium-binding protein that is an "armadillo" protein (type of protein that permits cell-cell interaction)
- Increased expression in pilomatricoma
- Involved in morphogenesis of hair follicle
- Accumulates in stem cells
- Localizes to nucleus in melanoma

TABLE 1–1
AMINO ACIDS

Amino Acid	Fiber
Isodesmosine	Elastin
Desmosine	Elastin
Citrulline	Vimentin
Hydroxylysine	Collagen
Hydroxyproline	

TABLE 1–2
BLISTERING DISORDERS (AUTOIMMUNE)

Disease	Antigen	kDa	DIF	IIF	Salt Split
Pemphigoid (BP)	BPAG1 (less) BPAG2 (NC16 only)	230 180	100% linear C3 at BMZ; most also IgG	75% IgG1, IgG4 at BMZ Titer no help	Roof; 20% roof and base
Protein 105 pemphigoid	105-kDa protein in lower lamina lucida	105	Linear IgG at DEJ		All on the base
Cicatricial pemphigoid (CP)	Laminin 5 Laminin 6 BPAG1 BPAG2 (NC16 and collagenous domain)	150, 140 200 230 180 145/290	80+% linear IgG, C3 along BMZ, occasional IgM, IgA	20–30% IgG1, IgG4, IgA1	Roof or both or base only Laminin 5 or coll. VII
Ocular only	Collagen VII β_4 integrin	205			
Herpes gestationis (HG)	BPAG2 (NC16 only) BPAG1 (less)	180 230	100% linear C3 BMZ 40% IgG	25% IgG1 HG factor = identified by complement factor enhanced ELISA)	Roof
Pemphigus foliaceus	Desmoglein 1	160	100% IgG intercellular 50% C3	80–85% IgG4 guinea pig esophagus	
Pemphigus herpetiformis (neonatal)	Desmoglein 1 Desmoglein 3	160 130	100% IgG intercellular in upper or entire epidermis	Most have circulating IgG	

Pemphigus erythematosus (Senear-Usher disease)	Desmoglein 1	160	Intercellular IgG, C3 25% granular IgG, C3 at BMZ, 50% linear, positive lupus band	Circulating IgG Positive ANA
IgA pemphigus	Intraepidermal neutrophilic (IEN) desmoglein 3 Subcorneal pustular dermatosis (SPD) desmocollin 1 desmocollin 2	130 115 105	IEN: intercellular IgA1 in entire or lower epidermis SPD: intercellular IgA in upper epidermis only	50% have circulating IgA Skin culture best substrate
Pemphigus vulgaris	100% Dsg3 50–75% Dsg1	130 160	100% IgG intercellular 50% C3, IgA, IgM	80–85% IgG4 Best: monkey esophagus may follow titer
Paraneoplastic pemphigus	Plectin Desmoplakin I BPAG1 Desmoplakin II/envoplakin Periplakin Unknown Dsg1 Dsg3	500 250 230 210 190 170 160 130	Intercellular IgG, C3; may have linear C3 along BMZ	75–80% positive IgG on monkey esophagus Rodent bladder epithelium (best substrate)
Dermatitis herpetiformis	Transglutaminase (IgA antibodies)		100% granular IgA in papillary dermis	+Antigliadin Ab +Antiendomyseal Ab +Antireticulin Ab

TABLE 1–2
BLISTERING DISORDERS (AUTOIMMUNE) (*Continued*)

Disease	Antigen	kDa	DIF	IIF	Salt Split
Linear IgA disease Chronic bullous disease of childhood	LABD Ag (NC16 + coll. domain) LAD-1 (ladinin) Collagen VII	97/120 285 145/290	100% linear IgA BMZ on noninvolved skin	80%+ in children 30%+ in adults	Roof > both > base
EB acquisita	Collagen VII (noncollagenous)	145/290 dimer	100% linear IgG BMZ can be C3, IgM/A	25–50% IgG to BMZ	Floor
Bullous LE	Collagen VII (collagenous) Laminin 5 Laminin 6 BPAG1	145/290 dimer	100% linear IgG BMZ 50–60% linear IgA/M some C3	IgG, IgA when present	Floor, roof, or absent

Key: BMZ, basement membrane zone; DEJ, dermoepidermal junction; Ag, antigen; EB, epidermolysis bullosa; NC, noncollagenous.
BP230 and plectin homologous to desmoplakin 1.
97 kDa of LABD; Ag = extracellular domain of BPAg2 (180 kDa).
ELISA, enzyme-linked immunosorbent assay; NC16, noncollagenous domain of BP Ag2; NC domain collagen VII, fibronectin, von Willebrand factor, cartilage matrix protein; hemidesmosomes analogous to desmoplakin.

TABLE 1–3
BLISTERING DISORDERS (INHERITED VS. AUTOIMMUNE)

Antigen	Inherited Disorder	Autoimmune
K5, K14	EB[a] simplex	
Plectin	EB simplex–muscular dystrophy	Paraneoplastic pemphigus
BPAG2	Generalized atrophic benign EB (GABEB)	BP, HG, CP
α6β4 integrin	Junctional EB (JEB) with pulmonary atresia	β4 integrin: ocular CP only
Laminin V	JEB (occasional GABEB)	CP
Collagen VII	Dystrophic EB (collagenous domain)	Epidermolysis bullosa acquisita (noncollagenous domain), bullous lupus (collagenous domain)[a]

[a] EB = Epidermolysis bullosa.
[b] Transient bullous dermolysis of newborn heals spontaneously within months; may be dominant EB variant.

BLOTS

- Southern = DNA
- Northern = RNA
- Western = protein

CADHERINS

- Ca-dependent adhesion molecules
- Four types: E-cadherin, P-cadherin, desmoglein, desmocollin
- Cadherins present in both desmosomes and adherens junctions

TABLE 1–4
CD MARKERS

Cell Type	Associated With
CD1a+	Langerhans cells: histiocytosis X, Gianotti-Crosti syndrome
CD2, 3, 5, 7	Pan T cell, usually need fresh tissue
CD4+	Parapsoriasis, LyP type B, pityriasis lichenoides chronica (PLC)
CD4+, often CD7–, ↑CD4, ↓CD8 Th1 Th2	Cutaneous T-cell lymphoma (CTCL) • Mycosis fungoides • Sézary syndrome
CD8+	Pityriasis lichenoides et varioliformis acuta (PLEVA), lichen planus, graft-vs.-host disease, psoriasis, alopecia areata
CD8+	Actinic reticuloid
CD8+ CD4–, CD8–	Pagetoid reticulosis: • Local (Woringer-Kolopp disease) • Disseminated (Ketron-Goodman disease)
CD10+	Folliculocentric B-cell lymphomas
CD15+, (may be CD30+)	Hodgkin disease
CD18	β_2-Integrin; defect = leukocyte adhesion deficiency type 1
CD20	Pan B-cell marker: ++ in B-cell lymphomas
CD21	Binds to EBV
CD25	Activated T cells (IL-2 receptor/Ontak® anti CD25 tagged with diptheria toxin)
CD30+	Lymphomatoid papulosis type A (type B = CD4+)
(Ki-1)	Large-cell anaplastic lymphoma (CTCL)
(Ber-H2)	Hodgkin disease
CD31	Endothelial cells, PECAM-1, angiosarcomas, Kaposi's sarcoma
C34+	Endothelials, DFSP (factor XIIIa-), Kaposi sarcoma, spindle cell lipoma
CD43	T cells, sialophorin (defect in Wiskott-Aldrich syndrome)
CD44	Extensive staining in Merkel cells indicates poor prognosis
CD45 (LCA)	White cells, Langerhans cells
CD45RO	Memory T cells
CD54	ICAM-1, which binds LFA-1
CD56	Natural killer (NK) cells (NK cell lympoma)
CD68+	Macrophages, JXG
CD79	Granulocytes, + myeloperoxidase, + chloracetate esterase = leukemia
CD95	Fas ligand (anti-Fas Ab used in toxic epidermal necrolysis) (Fas to ligand leads to apoptosis)
CD117	c-*kit* proto oncogene