

# Renal Glomerular Diseases

Atlas of Electron Microscopy with  
Histopathological Bases and Immunofluorescence  
Findings

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## Dedication

To my wife Joana and daughters Laura and Sandra  
whose support and understanding  
made this work possible

# Renal Glomerular Diseases

## Atlas of Electron Microscopy with Histopathological Bases and Immunofluorescence Findings

Presentation of 110 Cases of Patients Undergoing Kidney Biopsies

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## Abbreviations

### Glomerular Diseases

AS	Alport's syndrome
BN	Berger's nephropathy
FSGS	Focal segmental glomerulosclerosis
GBMN	Glomerular basement membrane nephropathy
GWCI	Glomerulonephritis with crescentic features
HSP	Henoch-Schönlein purpura
IGA	IgA nephropathy
INS	Infant nephrotic syndrome
KW	Kimmelstiel-Wilson disease
MCD	Minimal change disease
MN	Membranous nephropathy
MPGN	Membranoproliferative glomerulonephritis
PIAGN	Postinfectious acute glomerulonephritis
PICRGN	Pauci-Immune crescentic glomerulonephritis
SLE	Systemic lupus erythematosus
TBMN	Thin basement membrane nephropathy
TMA	Thrombotic microangiopathic nephropathy
UGN	Uncommon glomerulonephritis

### Miscellaneous

AA	Amyloid with serum A protein	HPF	High-power field
AL	Amyloid with light chain globulin	I	Interstitial tissue
Am	Amyloid	INR	International normalized ratio
ANA	Antinuclear antibodies	JGC	Juxtaglomerular cell
ANCA (C and P)	Antineutrophilic cytoplasmic autoantibodies	L	Capillary lumen
ASO	Antistreptolysin-O	LD	Lamina densa
BC	Bowman's capsule	LGH	Lutheran General Hospital
BP	Blood pressure	LRE	Lamina rara externa
bpm	Beats per minute	LRI	Lamina rara interna
Br	Break or defect of basement membrane	M	Mesangium
BUN	Blood urea nitrogen	MC	Mesangial cell
Cap	Capillary	MI	Mesangial interposition in the capillary wall
Col	Collagen	MM	Mesangial matrix
Cr	Crescent	MN	Mesangial nodule
Cr cell	Cell of crescent	Mon	Mononuclear cell or monocyte
CRP	C-reactive protein	MV	Microvillus or microvilli
D	Deposit	P	Podocyte
En	Endothelial cell	PAS	Periodic acid-Schiff reagent
Ep	Epithelial cell	Pmn	Polymorphonuclear leukocyte
ESR	Erythrocyte sedimentation rate	PT	Prothrombin time
F	Fibrin	PTT	Partial thromboplastin time
FC	Foam cell	RBC	Red blood cell
FP	Foot processes	S	Spike or spikes
GBM	Glomerular basement membrane	SSA	Sjögren's syndrome antibodies A
Gr	Granular material or granules	SSB	Sjögren's syndrome antibodies B
H&E	Hematoxylin and eosin	SM	Smooth muscle
H	Hyalin	TBM	Tubular basement membrane
Hct	Hematocrit	TC	Tubular cell
Hgb	Hemoglobin	U	Urinary space
		WNL	Within normal limits

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## Preface

The *Atlas* presents the results of a long-term study by a hospital pathologist with an interest and an active participation in the study of renal biopsy pathology. It is clear that the increased frequency of the use of diagnostic renal biopsies is related to a number of factors. The early effort using improved light microscopy and combined use of electron microscopy together with immunohistochemistry led to the characterization of significant glomerular lesions and made possible both a workable nomenclature and classification of many glomerulopathies.

With the introduction of dialysis and transplantation, there was a sharp increase in published series of renal biopsy findings that resulted in new approaches to the patient. It is now clear that the diagnosis of renal lesions by light microscopy alone should be considered provisional until supplemented by electron and immunofluorescence microscopy, particularly for early lesions and where clinically significant disease may occur with only slight glomerular changes. In practice, it is evident that critical clinical and laboratory data are especially important in interpreting significant findings properly. With serial renal biopsies at different times in the natural history of the disease and at critical times during treatment, the prognostic significance of specific lesions can be appreciated.

The glomerulus, while complex in its physiologic reactions, has relatively few structural components. Therefore, the patterns of reactions to injury are limited. Early classification of renal disease recognized this limitation by having few morphologic subgroups of glomerular disease, but many related clinical syndromes. With early and serial renal biopsies, improved histologic techniques, and the addition of tagging antibody techniques, there has been a rapid advance in improved clinicopathologic correlation and more accurate prognosis, especially with the use of a wide range of effective therapies.

This monograph documents frequent diagnostic lesions and a realistic range of variants occurring in one hospital practice. The importance of changes in frequency of some diseases over several decades is clearly demonstrated.

Benjamin Spargo, MD

## Introduction

Since 1961, following the Ciba Foundation Symposium in London on kidney biopsies, the percutaneous renal biopsy with light microscopic, electron microscopic, and immunofluorescence microscopic examination has become standard in the diagnosis of kidney pathology, particularly in the diagnosis of glomerular diseases. Today, it is widely recognized that the kidney biopsy specimen should undergo electron microscopic examination, in addition to light microscopic and immunofluorescence microscopic examination, as an essential part in the diagnosis of glomerular diseases.

The *Atlas* presents illustrations of electron micrographs, light micrographs, and immunofluorescence micrographic descriptions of 110 patients with glomerular disease, selected from 1,213 patients undergoing kidney biopsy examinations. The book's 18 sections reflect the classification of glomerular diseases as published in Rubin and Farber's textbook, *Pathology*, ed. 2, in the chapter Kidney Diseases, by Benjamin Spargo, MD, and Mark Haas, MD.

The format of the *Atlas* consists of clinical history, pertinent general laboratory findings (eg, urinalysis, CBC, BUN, creatinine, albumin, cholesterol), and special laboratory findings (eg, 24-hour urine protein, protein/creatinine ratio, complements C<sub>3</sub>, C<sub>4</sub>, CH<sub>50</sub>, and C<sub>1q</sub>, ANA, DNA, ASO, immunoelectrophoresis, kappa and lambda light chains, ANCA). In addition to electron microscopy, special attention has been given to light microscopic findings (H&E, PAS, trichrome, and silver Jones stains). The *Atlas* gives a detailed description of immunofluorescence microscopic findings in each case.

For better understanding of abnormal kidney electron and light microscopic findings, the normal glomerulus is illustrated and described. Multiple additional illustrations of electron microscopic pathology describe specific glomerular diseases, including minimal change disease, focal segmental glomerulosclerosis, membranous nephropathy, membranoproliferative glomerulonephritis, postinfectious acute glomerulonephritis, diabetic nephropathy, and amyloidosis.

The percutaneous renal biopsies were performed at Chicago-area hospitals (Lutheran General Hospital, Northwest Community Hospital, Swedish Covenant Hospital, Holy Family Hospital, Resurrection Hospital, Good Shepherd Hospital, Trinity Hospital, and Ravenswood Hospital). The specimens were examined and diagnosed by pathologists interested in renal pathology at Lutheran General Hospital (Yolanda Pinzòn-Trujillo, MD, Jerome B. Taxy, MD, Imad Almanaseer, MD, and Nitinchandra K. Bharani, MD, in addition to myself). Consultation was sought and rendered by nephropathologists Benjamin H. Spargo, MD, Maria Picken, MD, Conrad Pirani, MD, Robert L. Vernier, MD, and Melvin Schwartz, MD. The 110 cases presented in the *Atlas* were patients of the following nephrologists from the institutions previously mentioned: Harold Bregman, MD, David Burstein, MD, Margaret Bishel, MD, George Hvosik, MD, Ronald Kallen, MD, Richard Kaplan, MD, Daniel Kniaz, MD, Kenneth Miller, MD, Olive Oh, MD, and James Yeung, MD. The majority of the cases presented in this *Atlas* were discussed at Lutheran General Hospital's monthly renal biopsy conferences with the active participation of nephrologists, pathologists, residents, electron microscopists, and nephropathologist Benjamin Spargo, MD.