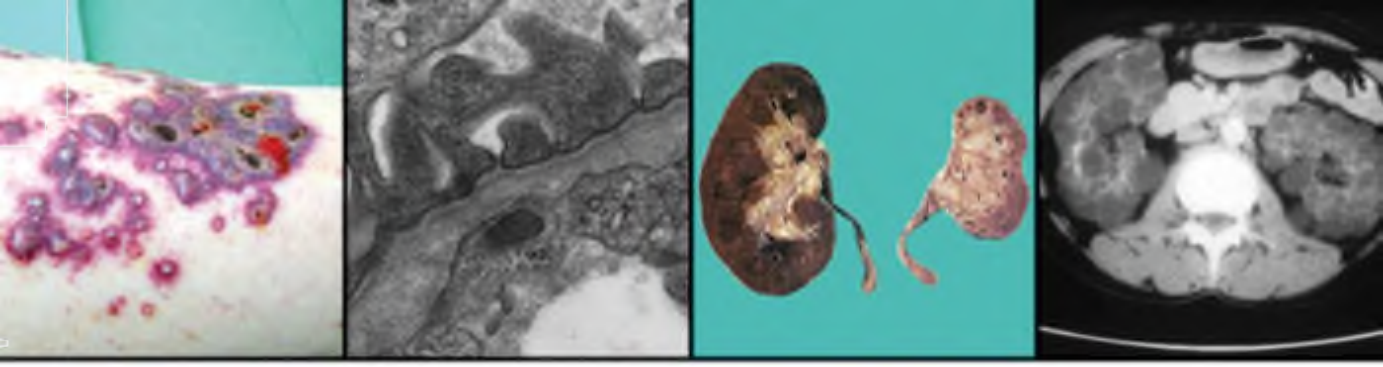

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► Except where otherwise stated, drug doses and recommendations are for the non-pregnant adult who is not breast-feeding.

Oxford Desk Reference
Nephrology

Jonathan Barratt
Senior Lecturer and Honorary Consultant Nephrologist,
John Walls Renal Unit,
Leicester General Hospital,
Leicester

Kevin Harris
Reader and Honorary Consultant Nephrologist,
John Walls Renal Unit,
Leicester General Hospital,
Leicester

and

Peter Topham
Reader and Honorary Consultant Nephrologist,
John Walls Renal Unit,
Leicester General Hospital,
Leicester

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Preface

In this era of evidence-based medicine, one of the biggest challenges confronting clinicians is keeping abreast with often rapidly changing recommendations that guide clinical practice. While it is clearly helpful to have research-based guidelines and protocols to draw upon it is not always easy to access this information, particularly at the time when it is needed most, such as on a ward round or in a busy outpatient clinic. These evidence-based guidelines, produced by national organizations (The Renal Association, British Hypertension Society, National Institute for Health and Clinical Excellence) and international organizations (International Society of Nephrology, National Kidney Federation – KDOQI) are often found in a variety of locations and published media and therefore timely access is not always possible.

To overcome this problem we have aimed to produce a comprehensive textbook of nephrology which focuses on aspects of renal disease that are important to the clinician. The book brings together the key recommendations found in current evidence-based guidelines and presents them in a uniform and accessible format. It has been designed and written so that locating information is both quick and simple, and the layout of the chapters allows the reader to identify and assimilate information rapidly.

The book is aimed at clinicians with a specialist interest in Nephrology (including consultants and specialist trainees in Nephrology) but it should also prove to be a valuable resource for any generalists who encounter a nephrological problem in their day-to-day practice.

We hope that this book will become an integral part of your working day.

Dr Jonathan Barratt
Dr Kevin Harris
Dr Peter Topham

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Brief contents

Detailed contents

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Abbreviations

ACEI	angiotensin-converting enzyme inhibitor
ACR	albumin:creatinine ratio
ADH	antidiuretic hormone
ADMA	asymmetric dimethyl arginine
ADPKD	autosomal dominant polycystic kidney disease
ADQI	Acute Dialysis Quality Initiative
AG	anion gap
AGE	advanced glycation end-products
AIDS	acquired immune deficiency syndrome
AIN	acute interstitial nephritis
AKI	acute kidney injury
AKIN	Acute Kidney Injury Network
ANA	antinuclear antibodies
ANCA	antineutrophil cytoplasmic antibodies
APD	automated peritoneal dialysis
ARAS	atheromatous renal artery stenosis
ARB	angiotensin-receptor blocker
ARPKD	autosomal recessive polycystic kidney disease
ARVD	atherosclerotic renovascular disease
ASOT	antistreptolysin O titre
ATN	acute tubular necrosis
AVF	arteriovenous fistula
AXR	abdominal X-ray
bd	twice daily
BCG	bacillus Calmette–Guérin
BNF	British National Formulary
BP	blood pressure
CAKUT	congenital abnormalities of the kidneys and urinary tract
CAPD	continuous ambulatory peritoneal dialysis
CAVH	continuous arteriovenous hemofiltration
CCPD	continuous cycling peritoneal dialysis
cfu	colony-forming units
CH_{50}	dose of complement required to hemolyse 50% of erythrocytes
CKD	chronic kidney disease
CMV	cytomegalovirus
CNI	calcineurin inhibitor
CNS	central nervous system
COX	cyclo-oxygenase
CRP	C-reactive protein
CRRT	continuous renal replacement therapy
CsA	ciclosporin
CT	computed tomography
CVP	central venous pressure
CVVHF	continuous venovenous hemofiltration
CVVHD	continuous venovenous hemodialysis
CVVHDF	continuous venovenous hemodiafiltration
CXR	chest X-ray
DBP	diastolic blood pressure
DEXA	dual energy X-ray absorptiometry
DI	diabetes insipidus
DIC	disseminated intravascular coagulation
DM	diabetes mellitus
DMSA	dimercaptosuccinic acid
DOPPS	Dialysis Outcomes & Practice Patterns Study
dsDNA	double-stranded DNA
DTPA	diethylenetriamine penta-acetic acid
ECF	extracellular fluid
ECG	electrocardiograph
EDD	extended daily dialysis
EDTA	ethylenediamine tetra-acetic acid
ENaC	epithelial sodium channel
eGFR	estimated glomerular filtration rate
eKt/V	equilibrated Kt/V
ELISA	enzyme-linked immunosorbent assay
EM	electon micrograph
ENA	extractable nuclear antigen
EPO	erythropoietin
ERF	established renal failure
ESA	erythropoiesis-stimulating agent
ESR	erythrocyte sedimentation rate
ESRD	end-stage renal disease
FSGS	focal and segmental glomerulosclerosis
FE_{Na}	fractional excretion of sodium
FMD	fibromuscular disease
FSGS	focal segmental glomerulosclerosis
GBM	glomerular basement membrane
GDP	glucose degradation products
GFR	glomerular filtration rate
GI	gastrointestinal
H&E	hemotoxylin and eosin
HAART	highly active antiretroviral therapy
HbSS	homozygous sickle cell anemia
HD	hemodialysis
HDF	hemodiafiltration
HELLP	Hemolytic anemia, Elevated Liver enzymes and Low Platelet count
HF	hemofiltration
HIT	heparin-induced thrombocytopenia
HIV	human immunodeficiency virus

HMG CoA	3-hydroxy-3-methylglutaryl coenzyme A
HP	hemoperfusion
HPF	high power field
HRS	hepatorenal syndrome
HTN	hypertension
HUS	hemolytic uremic syndrome
IF	immunofluorescence
IHD	intermittent hemodialysis
IHF	intermittent hemofiltration
IL	interleukin
iPTH	intact parathyroid hormone
ISPD	International Society of Peritoneal Dialysis
ITU	intensive therapy unit
IV	intravenous
IVU	intravenous urogram
K/DIGO	Kidney Disease Improving Global Outcomes
K/DOQI	Kidney Disease Outcomes Quality Iniative
KUB	kidneys, ureters and bladder
LDH	lactate dehydrogenase
LDL	low density lipoprotein
LFTs	liver function tests
LMWH	low molecular weight heparin
LV	left ventricle
MAG3	mercaptoacetylglycine
MARS	molecular adsorbent recirculating system
MCUG	micturating cystourethrogram
MDRD	Modification of Diet in Renal Disease study
MMF	mycophenolate mofetil
MRI	magnetic resonance imaging
MW	molecular weight
NIPD	nocturnal intermittent peritoneal dialysis
nPCR	normalized protein catabolic rate
NHANES	National Health and Nutrition Examination Surveys
NICE	National Institute for Health and Clinical Excellence (renamed in 2005)
NIDDKD	National Institute of Diabetes and Digestive and Kidney Diseases
NKF	National Kidney Foundation
NODAT	new-onset diabetes after transplantation
NPHP	nephronophthisis
nPNA	normalized protein equivalent of total nitrogen appearance
NSAID	nonsteroidal anti-inflammatory drug
NSF	nephrogenic systemic fibrosis
OAT-1	organic anion transporter-1
od	once daily
OCPD	optimized cycling peritoneal dialysis
PCR	protein:creatinine ratio
PD	peritoneal dialysis
PE	plasma exchange
PET	peritoneal equilibration test
PeT	per-eclampsia
pmp	per million population
PNA	protein equivalent of total nitrogen appearance
PO	per oral
PRA	panel reactive antibodies
PRCA	pure red cell aplasia
PSA	prostate-specific antigen
PTFE	polytetrafluoroethylene
PTH	parathyroid hormone
PTLD	post-transplant lymphoproliferative disease
PUJ	pelviureteric junction
qds	four times daily
RAS	renin–angiotensin–aldosterone system
RBF	renal blood flow
RCC	renal cell carcinoma
RCIN	radio contrast-induced nephropathy
RI	resistive index
RIFLE	risk, injury, failure, loss, end-stage disease
RTA	renal tubular acidosis
RR	relative risk
RRT	renal replacement therapy
SBP	systolic blood pressure
SC	subcutaneous
SEP	sclerosing encapsulating peritonitis
SGA	subjective global assessment
SHPT	secondary hyperparathyroidism
SIRS	systemic inflammatory response syndrome
SLEDD	slow low-efficiency daily dialysis
SNS	sympathetic nervous system
SPA	standardized permeability analysis
SPEP	serum protein electrophoresis
spKt/V	single-pool Kt/V
stdKt/V	standardized Kt/V
SVR	systemic vascular resistance
TCC	transitional cell carcinoma
tds	three times daily
TIPS	transjugular intrahepatic portosystemic shunt
TMP	transmembrane pressure
TNF-α	tumor necrosis factor-α
TNM	tumor, node, metastases
TPN	total parenteral nutrition
TRUS	transrectal ultrasound
TTP	thrombotic thrombocytopenic purpura

TURBT	transurethral resection of bladder tumor
TURP	transurethral resection of prostate
U&Es	urea, creatinine and electrolytes
UF	ultrafiltration
UFH	unfractionated heparin
UKM	urea kinetic modeling
UPEP	urine protein electrophoresis
URR	urea reduction ratio
USRDS	US Renal Data System
USS	ultrasound scan
UTI	urinary tract infection
VHL	von Hippel–Lindau
VUJ	vesicoureteric junction
VUR	vesicoureteric reflux

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Contributors

Dr Samuel Ajayi
Consultant Nephrologist
Department of Medicine
University of Abuja Teaching Hospital
Abuja, FCT
Nigeria

Dr Reem Al Jayyousi
Consultant Nephrologist and Honorary Senior Lecturer
John Walls Renal Unit
Leicester General Hospital
Leicester

Dr Karen Anderson
Consultant Nephrologist
The Richard Bright Renal Unit
Southmead Hospital
Bristol

Professor Mustafa Arici
Professor of Medicine
Hacettepe University Faculty of Medicine
Department of Nephrology
Ankara
Turkey

Dr Richard Baines
Clinical Lecturer in Nephrology
John Walls Renal Unit
Leicester General Hospital
Leicester

Mr Ali Bakran
Consultant Transplant & Vascular Surgeon
Royal Liverpool University Hospital
Liverpool

Mr Adam Barlow
Clinical Research Fellow
Department of Transplant Surgery
Leicester General Hospital
Leicester

Dr Jonathan Barratt
Senior Lecturer and Honorary Consultant Nephrologist
John Walls Renal Unit
Leicester General Hospital
Leicester

Professor Rashad Barsoum
Professor of Medicine
Kasr El-Aini Medical School
Cairo University
The Cairo Kidney Center
Cairo
Egypt

Dr Anne Barton
Reader and Honorary Consultant Rheumatologist
ARC Epidemiology Unit
Division of Epidemiology and Health Sciences
Stopford Building
Oxford Road
Manchester

Dr Linda M Barton
Consultant Haematologist
Department of Haematology
Leicester Royal Infirmary
Leicester

Dr Sunita Bavanandan
Consultant Nephrologist
Department of Nephrology
Institute of Urology and Nephrology
Hospital Kuala Lumpur
Kuala Lumpur
Malaysia

Professor Daniel Bichet
Professor of Medicine and Physiology
Renal Genetics
Montreal Sacré-Coeur Hospital
University of Montreal
Quebec
Canada

Dr Anne Blanchard
Centres d'Investigations Cliniques
Hopital Europeen Georges Pompidou
Paris
France

Dr Detlef Böckenhauer
Consultant Paediatric Nephrologist
Department of Nephrology
Great Ormond Street Hospital for Children NHS Trust
London

Professor Nigel Brunskill
Professor of Renal Medicine
John Walls Renal Unit
Leicester General Hospital
Leicester

Dr Katharine Buck
Consultant Nephrologist
Queen Margaret Hospital
Dunfermline

Mr Paul Butterworth
Consultant Urological Surgeon
Department of Urology
Leicester General Hospital
Leicester

Professor J Stewart Cameron
Emeritus Professor of Renal Medicine
Elm Bank
Melmerby
Penrith
Cumbria

Professor Giovambattista Capasso
Professor of Nephrology
Department of Internal Medicine
Second University of Naples
Naples
Italy

Dr S J Carr
Consultant Nephrologist and Honorary Senior Lecturer
John Walls Renal Unit
Leicester General Hospital
Leicester

Dr Claire S Chapman
Consultant Haematologist
Department of Haematology
Leicester Royal Infirmary
Leicester

Dr Chern Li Chow
Specialist Registar in Nephrology
Kidney Genetics Group
Academic Nephrology Unit
The Henry Wellcome Laboratories for Medical Research
School of Medicine and Biomedical Sciences
University of Sheffield
Sheffield

Miss Alexandra J Colquhoun
Specialist Registrar in Urology
Department of Urology
Leicester General Hospital
Leicester

Professor Christian Combe
Professor of Nephrology
Centre Hospitalier Universitaire de Bordeaux
Service de Néphrologie
Université de Bordeaux
Unité INSERM U889
Bordeaux
France

Dr Andrew Davenport
Consultant Nephrologist and Honorary Senior Lecturer
UCL Centre for Nephrology
Royal Free & University College Medical School
Hampstead Campus
London

Dr Frédéric Debelle
Consultant Nephrologist
Department of Nephrology
Hôpital Erasme
Université Libre de Bruxelles
Brussels
Belgium

Professor Chris Denton
Professor of Experimental Rheumatology
Department of Medicine
Royal Free Campus
University College
London

Dr Stephen J Dickinson
Specialist Registrar in Nephrology
John Walls Renal Unit
Leicester General Hospital
Leicester

Dr Haresh Dodeja
Consultant Nephrologist and Transplant Physician
Wockhadt Hospitals Ltd
Mulund-Mumbai
India

Dr Christopher Dudley
Consultant Nephrologist
The Richard Bright Renal Unit
Southmead Hospital
Bristol

Dr Tony Elliott
Consultant Clinical Oncologist
The Christie Hospital
Withington
Manchester

Dr Hossam El Shazly
Consultant Nephrologist
Department of Renal Medicine
Cairns Base Hospital
Cairns 4870
Australia

Dr Sally Feather
Consultant Paediatric Nephrologist
Department of Paediatric Nephrology
St James's University Hospital
Leeds

Professor John Feehally
Professor of Renal Medicine
John Walls Renal Unit
Leicester General Hospital
Leicester

Professor Jürgen Floege
Professor of Nephrology
Division of Nephrology
University Hospital
Aachen
Germany

Dr John Frew
Registrar in Clinical Oncology
Northern Centre for Cancer Treatment
Newcastle General Hospital
Westgate Road
Newcastle-upon-Tyne

Dr Trevor Friedman
Consultant Liaison Psychiatrist
Brandon Unit
Leicester General Hospital
Leicester

Dr Julian Gillmore
Senior Lecturer and Honorary Consultant Nephrologist
National Amyloidosis Centre
Royal Free and University College Medical School
Royal Free Hospital
London

Mr Leyshon Griffiths
Senior Lecturer and Honorary Consultant Urological Surgeon
Urology Section
Department of Cancer Studies & Molecular Medicine
University of Leicester Clinical Sciences Unit
Leicester General Hospital
Leicester

Prof Krishan Lal Gupta
Professor of Nephrology
Postgraduate Institute of Medical Education and Research
Chandigarh
India

Dr Pankaj Gupta
Specialist Registrar in Chemical Pathology
Leicester Royal Infirmary
Leicester

Dr Shikha Gupta
Specialist Registrar in Dermatology
Leicester Royal Infirmary
Leicester

Dr Jenny Hainsworth
Clinical Psychologist
Leicestershire Partnership NHS Trust and
Leicester General Hospital
Leicester

Dr Andrew Hall
Clinical Research Fellow
Centre for Nephrology
Royal Free and University College Medical School
London

Dr Matt Hall
Specialist Registrar in Nephrology
John Walls Renal Unit
Leicester General Hospital
Leicester

Dr Lorraine Harper
Senior Lecturer and Honorary Consultant Nephrologist
Division of Immunity and Infection
The Medical School
University of Birmingham
Edgbaston
Birmingham

Dr Steve Harper
Consultant Nephrologist
The Richard Bright Renal Unit
Southmead Hospital
Bristol

Dr Kevin Harris
Reader and Honorary Consultant Nephrologist
John Walls Renal Unit
Leicester General Hospital
Leicester

Professor Philip N Hawkins
Professor of Medicine
National Amyloidosis Centre
Department of Medicine
Royal Free and University College Medical School
Royal Free Hospital
London

Professor Friedhelm Hildebrandt
Professor of Pediatrics and of Human Genetics
Frederick G.L. Huetwell Professor for the Cure and Prevention of Birth Defects
Doris Duke Distinguished Clinical Scientist
University of Michigan
Department of Pediatrics
Ann Arbor
Michigan
USA

Dr Richard Holt
Consultant Paediatric Nephrologist
Royal Liverpool Children's Hospital
Alder Hey
Liverpool

Prof Pascal Houillier
Professor of Physiology
Departement de Physiologie
Hopital Europeen Georges Pompidou
Paris
France

Dr Peter Houtman
Consultant Paediatrician
Leicester Royal Infirmary
Leicester

Dr Alastair Hutchison
Consultant Nephrologist
Department of Renal Medicine
Manchester Royal Infirmary
Manchester

Dr David Jayne
Consultant Nephrologist
Renal Unit
Addenbrookes Hospital
Cambridge

Dr Graham Johnston
Consultant Dermatologist
Leicester Royal Infirmary
Leicester

Dr Caroline Jones
Consultant Paediatric Nephrologist
Royal Liverpool Children's Hospital
Alder Hey
Liverpool

Dr Philip A Kalra
Consultant Nephrologist and Honorary Senior Lecturer
Dept of Renal Medicine
Hope Hospital
Salford

Dr Jens Kannmacher
Renal Fellow
Division of Nephrology
University Hospital
Aachen
Germany

Professor Robert Kleta
Professor of Nephrology
Centre for Nephrology
Royal Free and University College Medical School
London

Mr Roger Kockelbergh
Consultant Urological Surgeon
Leicester General Hospital
Leicester

Dr George Kosmadakis
Nephrologist and Clinical Research Fellow
John Walls Renal Unit
Leicester General Hospital
Leicester

Dr Andrew J P Lewington
Consultant Nephrologist and Honorary Senior Lecturer
Department of Renal Medicine
St James's University Hospital
Leeds

Dr Robert Mactier
Consultant Nephrologist
Renal Unit
Glasgow Royal Infirmary
Glasgow

Dr Webster Madira
Consultant Chemical Pathologist
Leicester Royal Infirmary
Leicester

Dr Stephen D Marks
Consultant Paediatric Nephrologist
Department of Nephrology
Great Ormond Street Hospital for Children NHS Trust
London

Dr Nick Mayer
Consultant Histopathologist
Department of Pathology
Leicester General Hospital
Leicester

Dr Simon Maxwell
Senior Lecturer in Clinical Pharmacology
Queens Medical Research Institute
Edinburgh

Dr Catherine McBain
Consultant Clinical Oncologist
The Christie Hospital
Withington
Manchester

Professor John K Mellon
Professor of Urology
Urology Section
Department of Cancer Studies & Molecular Medicine
University of Leicester Clinical Sciences Unit
Leicester General Hospital
Leicester

Dr Christopher Mitchell
Consultant Paediatric Oncologist
John Radcliffe Hospital
Headington
Oxford

Dr Henry Morgan
Consultant Paediatric Nephrologist
Royal Liverpool Children's Hospital
Alder Hey
Liverpool

Dr Francis J Mussai
Fellow in Paediatric Haematology and Oncology
John Hopkins University Hospital
Baltimore
Maryland
USA

Dr Chas Newstead
Consultant Nephrologist
Department of Renal Medicine
St James's University Hospital
Leeds

Professor Michael Nicholson
Professor of Transplant Surgery
Department of Transplant Surgery
Leicester General Hospital
Leicester

Dr Joëlle Nortier
Head of the Department of Nephrology
Hôpital Erasme
Université Libre de Bruxelles
Brussels
Belgium

Dr Albert CM Ong
Reader and Honorary Consultant Nephrologist
Kidney Genetics Group, Academic Nephrology Unit
The Henry Wellcome Laboratories for Medical Research
School of Medicine and Biomedical Sciences
University of Sheffield
Sheffield

Dr Rakesh S Patel
Specialist Registrar in Nephrology and General Medicine
John Walls Renal Unit
Leicester General Hospital
Leicester

Dr Sue Pavord
Consultant Haematologist
Leicester Royal Infirmary
Leicester

Dr Henry Penn
Clinical Research Fellow
Centre for Rheumatology
Royal Free Hospital
London

Dr Liam Plant
Consultant Nephrologist
Department of Renal Medicine
Cork University Hospital
Cork
Ireland

Dr Pradeep Rao
Specialist Registrar in Psychiatry
Brandon Unit
Leicester General Hospital
Leicester

Dr Yvonne Rees
Consultant Radiologist
Department of Radiology
Leicester General Hospital
Leicester

Professor Vincent Rigalleau
Professor of Nutrition
Centre Hospitalier Universitaire de Bordeaux
Service de Diabétologie Nutrition
Université de Bordeaux
Bordeaux
France

Dr Alan D Salama
Senior Lecturer and Honorary Consultant Nephrologist
Renal Section
Division of Medicine
Hammersmith Hospital
London

Dr Moin A Saleem
Reader and Consultant Paediatric Nephrologist
University of Bristol
Children's Renal Unit
Bristol Royal Hospital for Children
Bristol

Dr Andy Salmon
Clinical Lecturer in Nephrology
The Richard Bright Renal Unit
Southmead Hospital
Bristol

Mr Richard FJ Stanford
Clinical Research Fellow
Department of Cancer Studies & Molecular Medicine
University of Leicester

Dr John Schollum
Consultant Nephrologist
Department of Nephrology
Dunedin Hospital
Dunedin
New Zealand

Dr John E Scoble
Consultant Nephrologist
New Guys House
London

Dr Adrian Stanley
Consultant Physician and Clinical Pharmacologist
Leicester Royal Infirmary
Leicester

Professor Vladisav Stefanovic
Professor of Medicine
Institute of Nephrology
Faculty of Medicine
University of Nis
Nis
Serbia

Dr Daniel Teta
Consultant Nephrologist
Privat-Docent & MER
Service de Néphrologie
CHUV, 1011 Lausanne
Switzerland

Mr James F Thorpe
Clinical Research Fellow
Department of Cancer Studies & Molecular Medicine
University of Leicester

Dr Charles Tomson
Consultant Nephrologist
The Richard Bright Renal Unit
Southmead Hospital
Bristol

Dr Peter Topham
Senior Lecturer and Honorary Consultant Nephrologist
John Walls Renal Unit
Leicester General Hospital
Leicester

Dr Kjell Tullus
Consultant Paediatric Nephrologist
Department of Nephrology
Great Ormond Street Hospital for Children NHS Trust
London

Professor Robert Unwin
St Peter's Professor of Nephrology
Centre for Nephrology
Royal Free and University College Medical School
London

Professor Jean-Louis Vanherweghem
Emeritus Professor of Nephrology
Department of Nephrology
Hôpital Erasme
Université Libre de Bruxelles (ULB)
Brussels
Belgium

Dr William Van't Hoff
Consultant Paediatric Nephrologist
Department of Nephrology
Great Ormond Street Hospital
London

Dr Stephen Walsh
Clinical Research Fellow
Centre for Nephrology,
Royal Free and University College Medical School,
London

Dr Graham Warwick
Consultant Nephrologist
John Walls Renal Unit
Leicester General Hospital
Leicester

Professor Alan Watson
Professor of Paediatric Nephrology
Children's Renal and Urology Unit
Nottingham University Hospitals
Nottingham

Dr Rachel Westacott
Specialist Registrar in Nephrology
John Walls Renal Unit
Leicester General Hospital
Leicester

Dr Stanley White
Senior Lecturer
Institute of Membrane and Systems Biology
University of Leeds
Leeds

Dr Christopher Winearls
Consultant Nephrologist
Oxford Radcliffe Hospitals
Oxford

Dr Matthias TF Wolf
Senior Research Fellow
Department of Pediatrics
University of Michigan
Ann Arbor
Michigan
USA

Dr Graham Woodrow
Consultant Renal Physician
Department of Renal Medicine
St James's University Hospital
Leeds

Dr Rosnawati Yahya
Consultant Nephrologist
Department of Nephrology
Hospital Kuala Lumpur
Jalan Pahang
Kuala Lumpur
Malaysia

Dr Janak R de Zoysa
Consultant Nephrologist
Department of Renal Medicine
Auckland City Hospital
Auckland
New Zealand

Glomerular disease in the tropics

Glomerulonephritis (GN) accounts for 30–60% of all end-stage renal disease (ESRD) in the tropics.

Infection is the commonest cause of glomerular disease in this setting with hypertension and diabetes mellitus accounting for the majority of the remainder. Infectious causes of glomerular disease in the tropics are listed in Table 3.13.1.

Human immunodeficiency virus (HIV)-associated disease is becoming increasingly common.

Renal biopsy confirmation of the diagnosis is often difficult due to lack of diagnostic facilities and to late presentation when, in the face of advanced disease, renal biopsies become difficult to interpret.

Epidemiology

There is variability in the etiology of glomerular disease in different parts of the tropics due to differences in the epidemiology of infections, standards of living, and infrastructure development.

The high prevalence of chronic GN points to variety of etiological agents.

GN usually presents in one of four ways in the tropics: acute nephritic syndrome, nephrotic syndrome, asymptomatic urinary abnormalities, and ESRD.

This chapter focuses on causes of the nephrotic and nephritic syndromes, and incorporates asymptomatic urinary abnormalities and ESRD where appropriate.

Acute nephritic syndrome

Etiology

Post-streptococcal GN (PSGN) is the commonest cause of rapidly progressive renal failure in the tropics.

It is most common in children.

It may occur during epidemics of post-streptococcal skin (impetigo, pyoderma) and throat infections.

Different nephritogenic serotypes have been identified:

- skin: M types 47, 49, 55, and 57;
- throat: M types 1, 2, 4, and 12.

The risk of nephritis is 5% with throat infection and up to 25% for pyoderma.

GN occurs 1–3 weeks after the onset of a streptococcal infection.

Pathogenesis

PSGN is generally assumed to be due to deposition of circulating immune complexes in the glomeruli. However, this should result in activation of the classical complement pathway (with C3 and C4 activation), but C4 levels are normal and only C3 is found in the glomerular immune deposits (suggesting alternative pathway activation).

The following possibly contribute to inflammation and injury:

- activation of the alternative complement pathway by glyceraldehyde-3-phosphate dehydrogenase (GAPDH);
- cationic streptococcal antigen promoting development of subepithelial immune deposits ('humps');
- cytokines IL-6, TNF-α and vasoactive mediators such as platelet activating factors;
- *in situ* deposition of antigen followed by antibody formation;
- IgG rheumatoid factor initiating an autoimmune reaction in the glomerulus.

Table 3.13.1. Infections associated with glomerulonephritis in the tropics

Bacterial	
	Streptococus pyogenes
	E. coli 0157 :H7 (HUS)
	Salmonella typhi
	Mycobacterium leprae
	Mycobacterium tuberculosis
	Staphylococcus epidermidis/aureus
	Treponema pallidum
	Streptococcus pneumoniae
Protozoal	
	Plasmodium falciparum
	Plasmodium malariae
	Leishmania donovani
	Toxoplasma gondii
	Trypanosoma cruzei
Viral	
	Hepatitis B
	Hepatitis C
	HIV
	Epstein–Barr virus
Helminthic	
	Schistosoma mansoni
	Schistosoma haematobium
	Wuchereria bancrofti
	Brugia malayi
	Loa loa
	Onchocerca volvulus

Clinical course

Most children (>90%) recover completely. Approximately 5% develop progressive acute kidney injury or acute GN.

In acute GN, there is rapid deterioration of renal function, oliguria, fluid overload from sodium and water retention, hypertension (80%), edema (80–90%), and pulmonary edema.

Approximately 20% have long-term sequelae with asymptomatic urinary abnormalities, nephrotic syndrome, hypertension, or very rarely progressive renal dysfunction.

Examination of renal histology of the nephrotic syndrome in nonmalarious parts of the tropical world would suggest that PSGN may be a cause in some patients. It may be the reason why minimal change disease is rarely seen in Black African children with nephrotic syndrome.

Chronic GN leading to progressive chronic kidney disease is more likely in adolescents than in children, especially in those with persistent proteinuria.

Elderly patients do less well (70% have azotemia, 40% heart failure, 25% early mortality). Longer-term prognosis is also less good than in children with up to 25% being left with abnormal renal function.

Diagnosis

This is usually easy to make from the clinical features and simple laboratory tests.

Oliguria may not be obvious in children, but 'coca-cola'-colored urine, due to macroscopic hematuria, is usually apparent.

Urinalysis

- Red blood cells and red cell casts.
- Variable degree of proteinuria.
- Some may have nephrotic range proteinuria.
- Leukocytes.

Serology

A positive antistreptolysin O (ASO) titer is seen in only ~30% of patients (perhaps because of early antibiotic use).

Serum C3 levels are reduced in >90% of patients in the first week, but return to normal within 2 months.

Serum C4 is typically normal.

Serum IgG and IgM are elevated in 75–80% of patients.

A low level of anti-DNA antibodies and ANCA may be seen.

Renal biopsy

This is rarely needed to confirm the diagnosis.

Indications:

- Nephrotic-range proteinuria.
- Acute kidney injury.
- Patients with sickle-cell anemia (a recognized cause of hematuria).

Light microscopy:

- Diffuse endocapillary, mesangial and endothelial proliferation. Neutrophil infiltration in glomeruli.
- Interstitial infiltration by monocytes and lymphocytes.

Immunofluorescence microscopy:

- C3, IgG, IgM deposition in capillary loops and the mesangium is invariable.

Electron microscopy:

- Classic subepithelial electron-dense deposits ('humps').

Differential diagnosis

Protein-energy malnutrition and nephrotic syndrome due to other causes.

Treatment

Treat ongoing streptococcal infection with appropriate antibiotics, e.g. benzathine penicillin (1.2 million U intramuscularly as a single dose).

Restrict salt and water intake.

Diuretics (e.g. furosemide) for volume overload.

Antihypertensive agents which may also prevent hypertensive encephalopathy: nifedipine, hydrallazine, Na nitroprusside.

Dialysis, although rarely needed in children, is a challenge in many tropical countries due to lack of dialysis infrastructure.

Prognosis

This is excellent in children who recover, but poor in adolescents who may progress to chronic GN.

Proteinuria may persist for months, and sometimes years.

Follow-up

It may be necessary to screen other family members – especially young children, who may have subclinical disease in epidemics of PSGN.

Nephrotic syndrome (NS)

NS is 2–10 times more common in the tropical countries than in Western Europe or North America.

It has recently been reported that NS accounts for 38% of nephrological admissions in the Democratic Republic of Congo.

Etiology

Even within the tropics, there are interracial differences. For example:

In South Africa minimal change disease is more common in Indian children than in Black children who have a preponderance of mesangiocapillary GN (MCGN). Minimal change disease is also common in India, suggesting a genetic predisposition.

MCGN predominates in Nigerian and Kuwaiti children. The disparities in histological features may suggest that NS may be a complication of PSGN and other forms of glomerular diseases. This may account for the rarity of minimal change disease as a cause of NS in African children.

The three main primary causes of NS are:

- MCGN.
- membranous GN.
- focal segmental glomerulosclerosis.

The most common secondary form of GN causing NS in the tropics is Quartan malarial nephrotic syndrome. Because this is peculiar to the tropics, it is discussed in more detail.

Quartan malarial nephrotic syndrome (QMNS)

Plasmodium malariae is the most important cause of secondary GN causing NS in children and adolescents.

QMNS is probably due to deposition of soluble immune complexes in the glomerulus but it is characterized by the absence of specific antibody and antigen. This has raised questions about the exact pathogenetic mechanism.

Pathology:

The characteristic appearance is of MCGN with progressive mesangial sclerosis leading to obliteration of glomerular capillaries with eventual involvement of tubules.

Immunofluorescence microscopy demonstrates coarse granular deposits of IgG, IgM and C3 mainly within the glomerular capillary walls.

Electron microscopy shows subendothelial electron-dense deposits and thickened GBM.

Clinical features:

Fever peaking once every 72 h is the earliest feature.

Gross edema and ascites are the main symptoms which develop several weeks after the onset of fever. This may easily be confused with protein-energy malnutrition (kwashiorkor).

Other features are nonselective proteinuria, microscopic hematuria, normal blood pressure, anemia, and hepatomegaly.

Hypoalbuminemia may be severe (10–20 g/L). Due to associated malnutrition in the majority of cases, the serum cholesterol is characteristically normal.

P. malariae may be detected in 69–70% of cases. The presence of parasites even when glomerular changes are present does not necessarily indicate cause and effect. Detection of a specific antigen has been demonstrated in only a few cases.

Clinical course:

Prognosis is poor, and spontaneous remission is rare. QMNS causes end-stage renal failure in <4 years.

Treatment:

There is a uniformly a poor response to corticosteroids, and to antimalarials. There are reports of remission of steroid-resistant patients with cyclophosphamide but this leads to no improvement in overall survival. Giving a therapeutic trial of corticosteroids before biopsy is therefore a practice that is increasingly being called into question.

Falciparum malaria

P. falciparum infection is mainly a disease of children in the tropics. Unlike QMNS, it is not progressive, and it resolves following eradication of *P. falciparum*. Indeed, GN may pass unnoticed especially with the widespread use of antimalarials.

There is nonselective proteinuria, microscopic hematuria and casts.

The typical pathological abnormalities are mesangial hypercellularity with mild matrix expansion changes without basement membrane involvement.

IgG, IgM and C3 can be identified in the mesangium. Immune deposits can be identified by EM in the mesangial and subendothelial areas.

The renal lesions recover with conventional antimalarial treatment.

Schistosoma mansoni-associated nephropathy (see Chapter 7.4 for more detail)

This is common in Brazil and Egypt, and patients often present with the nephrotic syndrome.

Glomerulonephritis is most common when there are hepatosplenic manifestations and chronic salmonella infection.

Males are twice more affected than females, probably due to outdoor activities in rivers and pools.

Clinical features include:

- overt peripheral edema and ascites;
- poorly selective proteinuria and, rarely, hematuria;
- low serum C3 with hypergammaglobulinemia;
- normal cholesterol.

Pathology:

Several patterns of glomerular pathology have been observed:

- class I mesangioproliferative lesions;
- class II diffuse proliferative GN;
- class III MCGN types I and III;
- class IV focal and segmental glomerulosclerosis (FSGS);
- class V amyloidosis.

Treatment:

Praziquantel 20 mg/kg body weight is effective in eradicating the parasites but this does not result in resolution of glomerular disease that has progressed beyond class II.

The treatment of chronic salmonella infection may cause resolution of glomerular disease (usually class II changes), suggesting a critical role for salmonella antigens in the pathogenesis of disease.

A good response to oxamniquine has been observed in class II disease.

The role of immunosuppression is unclear.

Hepatitis B virus (HBV)-associated GN

HBV infection is prevalent in certain tropical countries, and the kidneys may be affected by immune-complex-mediated disease.

Acute HBV infection is associated with a serum sickness-like syndrome characterized by fever, arthralgias/arthritis, hepatitis, rash (urticaria/maculopapular) and renal involvement (microscopic hematuria, sterile pyuria, and proteinuria)

Renal biopsy, if performed, shows mesangial proliferative GN.

GN resolves with remission of the acute phase of the disease.

Approximately 10% of patients acutely infected with HBV become chronic carriers and are at risk for other renal manifestations of HBV. These include membranous nephropathy, polyarteritis nodosa, MCGN, and IgA nephropathy.

This topic is covered in detail in Chapter 3.11.

The treatment of HBV-related renal disease is more effective for membranous GN than for MCGN.

Treatment regimens that have shown benefit usually include interferon-α and lamivudine.

However, since these agents are not widely available in the tropics, the management of HBV-mediated renal disease (and the other manifestations of HBV infection) can be suboptimal.

Steroids and cytotoxics are contraindicated since they enhance viral replication and may precipitate hepatitic flares.

Hepatitis C virus (HCV)-associated GN

Glomerular disease associated with HCV has not been well-described in the tropics, even though the pathological changes and clinical course are not expected to be different from those described in the Western world.

These include variable degrees of proteinuria, microscopic hematuria, and renal impairment.

Mesangiocapillary GN has been described.

HIV-associated renal disease

The tropical regions, especially sub-Saharan Africa currently bear the brunt of the HIV/AIDS pandemic. Therefore, occurrence of renal disease associated with HIV is on the increase, and impacting negatively on the outcome of HIV disease.

HIV is immunocytopathic, and it therefore can cause a variety of renal syndromes such as:

- HIV nephropathy (HIVAN) (in >60% it occurs with advanced disease).
- Immune complex GN ('lupus-like syndrome').
- Thrombotic microangiopathy.

In addition, chemotherapy can present a challenge because some antiretroviral (ARV) and adjunct medications can cause nephrotoxicity (pentamidine) or nephrolithiasis (indinavir).

HIVAN: Occurs in about 60% of patients with advanced disease, but may be subclinical in the majority of cases. It is rapidly progressive and may reach ESRD within 5 months.

HIVAN is a diagnosis of exclusion, and therefore other treatable causes such as hypovolemia from diarrhea should be excluded. In the tropics, a significant number of patients use herbal preparations for recurrent febrile illness, and this may cause nephrotoxicity.

Pathogenesis:

HIV infects tubular and glomerular epithelial cells and podocytes. This may explain the occurrence of NS in affected individuals.

Pathology:

This typically shows a preserved Bowman's space with collapsed/shrunken glomeruli ('collapsing glomerulopathy'), and focal dilatations of tubules presenting a 'microcystic' appearance.

Treatment:

RAS blockade can slow progression in those with moderate CKD (creat <176 μmol/L (2 mg/dL)).

HAART (highly active antiretroviral therapy) may cause remission of glomerulopathy.

Prednisolone may be beneficial in high risk patients.

Sickle cell-associated renal disease

Sickle cell disease is common in certain population groups in the tropics, particularly Black Africans and Africans in the Caribbean. It causes renal disease by one or all of the following mechanisms:

- hypoperfusion/ischemia;
- iron overload and deposition;
- intracapillary fragmentation and phagocytosis of sickled cells;
- immune complex formation;
- hyperfiltration and subsequent glomerular sclerosis.

Pathology:

The following appearances may be observed:

- congestion of capillary loops with sickled cells;
- FSGS ;
- MCGN ;
- tubular atrophy ;
- iron pigment deposits.

Clinical features:

- gross hematuria;
- acute kidney injury from renal papillary necrosis, pregnancy, multiple organ failure;
- nephrotic syndrome.
- ESRD.

Treatment:

RAS blockade improves proteinuria by reducing intraglomerular pressure, but large-scale studies are required to prove their long-term benefit for prevention/resolution of glomerular sclerosis.

NSAIDs and immunosuppressive drugs (steroids and cyclophosphamide) are not of proven benefit.

Leprosy-associated GN

Mycobacterium leprae may cause immune complex disease, secondary amyloidosis, interstitial nephritis, and renal tubular disorders.

Glomerular lesions occur more commonly in the lepromatous form, and the histological lesions include: mesangioproliferative, mesangiocapillary, and crescentic GN, especially when patient is being treated with rifampicin.

Clinical features:

Only a few patients present with nephrotic syndrome, nephritic syndrome and rapidly progressive GN. Most patients are asymptomatic with a variety of urinary abnormalities.

Treatment:

Early treatment of leprosy may prevent occurrence of renal involvement. Treatment with rifampicin and occurrence of erythema nodosum leprosum (ENL) may exacerbate glomerular lesions. Steroids have no proven benefit.

Other forms of secondary GN

The following organisms can cause GN, even though the prevalence varies from region to region depending on the level of preventive measures: filarial worms, helminths, protozoas, and fungi. They induce various forms of GN, e.g. MCGN, membranous GN.

Patients often present with asymptomatic proteinuria or hematuria, and sometimes nephrotic syndrome. Generally, glomerular lesions do not respond to treatment of causative organisms.

Further reading

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Internet resources

Information on schistosomiasis:

`www.who.int/ctd/schisto`

See also

Infection-related glomerulonephritis, p. 128

Acute endocapillary glomerulonephritis, p. 116

Mesangiocapillary glomerulonephritis, p. 112

Mixed cryoglobulinemia and hepatitis C infection, p. 176

Sickle cell disease, p. 192

Renal tuberculosis and other mycobacterial infections, p. 256

Schistosomiasis, p. 260

Fungal infections and the kidney, p. 264

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