AMT MRCP(UK) Parts 1 and 2

Renal Medicine Illustrated Revision Handbook

Renal Medicine

Part 1 foundations and Part 2 clinical application

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Version 8 | Physician-approved enhanced MRCP Part 2 visual edition | Chapter only

PUBLICATION STATUS: Physician approved on 27 October 2026. This handbook is an educational resource; prescribing and patient-care decisions must follow current local guidance, the BNF/SmPC and appropriate specialist advice.

How to use this chapter

Renal Medicine is the first chapter rebuilt under the unified AMT MRCP(UK) Parts 1 & 2 architecture. It teaches normal structure and function once, then extends the same knowledge into disease, investigation, management and complex Part 2 decisions without duplicating the disease profile.

Chapter learning system
Understand the mechanism -> see the anatomical or clinical pattern -> associate it with a concise memory cue -> retrieve it rapidly -> apply it to the next clinical decision. Memory aids support reasoning; they never replace it.

Layer	Purpose
PART 1 CORE	Anatomy, physiology, biochemistry, pathology, pharmacology, mechanisms and fundamental diagnosis.
PART 1 + PART 2	Core disease profiles, investigations, emergencies and management principles.
PART 2 APPLICATION	Complex presentation, competing diagnoses, treatment selection, transplantation, multimorbidity and prognosis.
QUESTION BANK	30 Part 1 + 30 Part 2 original AMT BOF questions with permanent IDs and full option rationales.

BLUEPRINT: Renal Medicine is currently an indicative 14 questions in Part 1 and 20 questions in Part 2. The 30+30 chapter set is an educational practice bank, not a claim about live exam composition.

Chapter map

Section	High-yield focus	Exam layer
Foundations	Anatomy, embryology, GFR, tubular transport, RAAS, water, K+, acid-base, endocrine renal function	P1 core
Pharmacology	RAAS blockers, SGLT2 inhibitors, finerenone, diuretics, potassium binders, anaemia/CKD-MBD drugs, transplant drugs	P1 + P2
AKI	Pre-renal, intrinsic, vascular, post-renal; urinalysis, microscopy, ultrasound, biopsy, RRT	P1 + P2
CKD	Cause-G-A classification, KFRE, progression, CVD risk, anaemia, CKD-MBD, RRT/transplant	P1 + P2
Glomerular/tubular	Nephritic/nephrotic patterns, GN, RTA, tubulointerstitial disease	P1 + P2
Complex renal	ACS in CKD, DKA in renal failure, transplant AKI and toxicity	P2 application
Revision	AMT 60-second recall panels; premium visual cards scheduled for Phase 2	Retrieval practice

1. Renal anatomy and embryology

PART 1 CORE • ANATOMY → PHYSIOLOGY → PATHOLOGY → PHARMACOLOGY

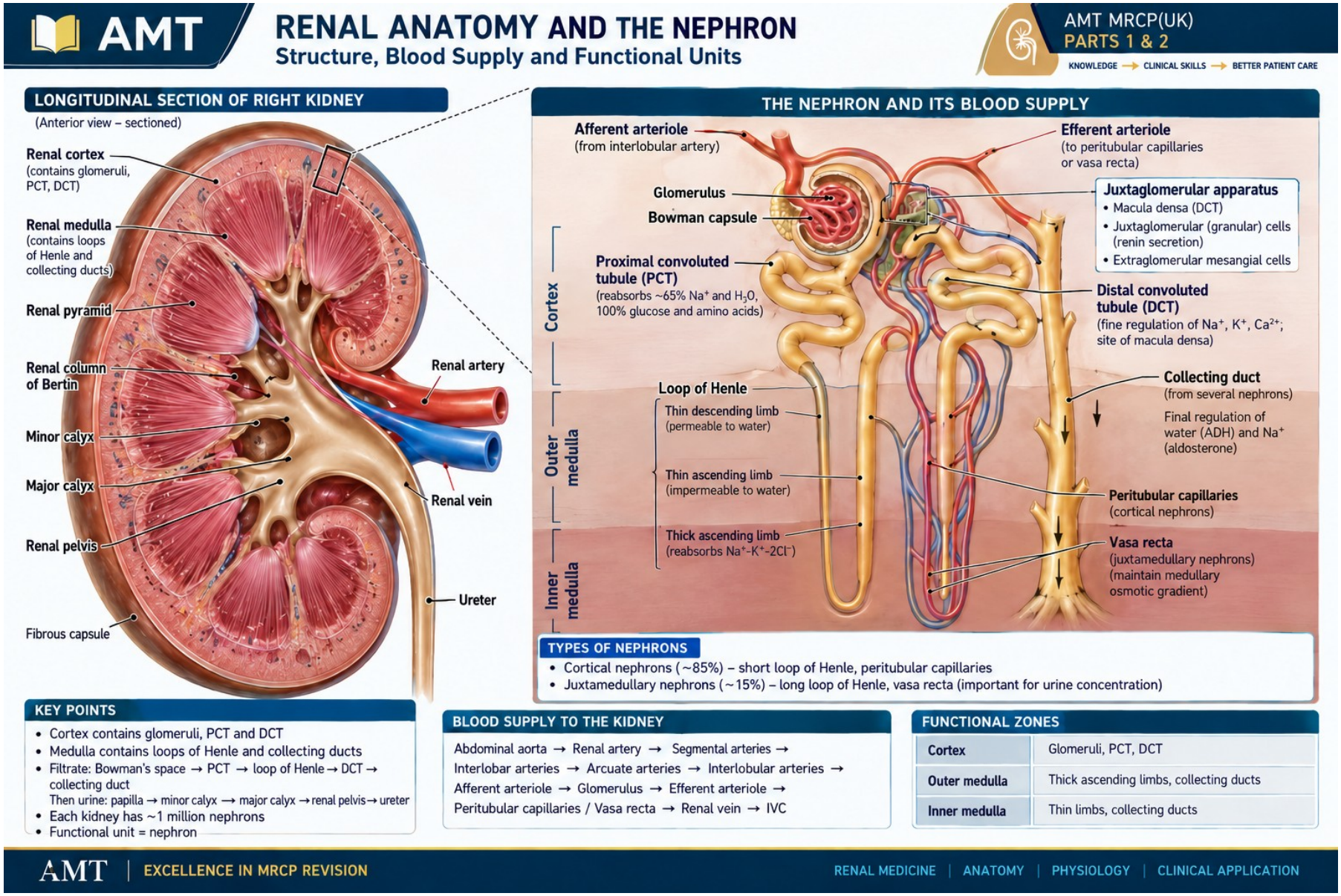


Figure REN-01. Original AMT renal anatomy plate. Filtrate starts in Bowman's space, passes through proximal tubule, loop of Henle, distal tubule and collecting duct; urine then passes via papilla, calyces and renal pelvis to the ureter. Educational illustration; not a diagnostic image.

The kidneys are retroperitoneal organs extending approximately from T12 to L3; the right kidney usually lies slightly lower because of the liver. The cortex contains glomeruli and convoluted tubules, while loops of Henle and collecting ducts traverse the medulla. Segmental arterial branches behave functionally as end arteries, making vascular anatomy clinically relevant in infarction and surgery.

Embryology and congenital correlates

- Metanephric mesenchyme and ureteric bud interact to form the permanent kidney; disruption can produce dysplasia, agenesis or collecting-system abnormalities.
- Kidneys ascend from the pelvis during development; ectopic or horseshoe kidneys reflect abnormal ascent/fusion and can predispose to obstruction, infection or stones.
- Congenital anomalies of kidney and urinary tract (CAKUT) are important causes of CKD in younger people.

CLINICAL CORRELATE: A structural abnormality can produce recurrent infection, hypertension or obstruction long before eGFR declines. Anatomy therefore belongs in the diagnostic reasoning, not in an isolated preclinical section.

AMT MEMORY AID
Filtrate: Bowman's space → proximal tubule → loop of Henle → distal tubule → collecting duct. Urine then drains via papilla → minor calyx → major calyx → renal pelvis → ureter. Blood follows afferent arteriole → glomerulus → efferent arteriole → peritubular capillaries or vasa recta.

MRCP RAPID RECALL
Localise first: glomerulus (filtration), proximal tubule (bulk reclamation), loop (gradient), distal nephron (fine control), collecting duct (ADH/aldosterone).

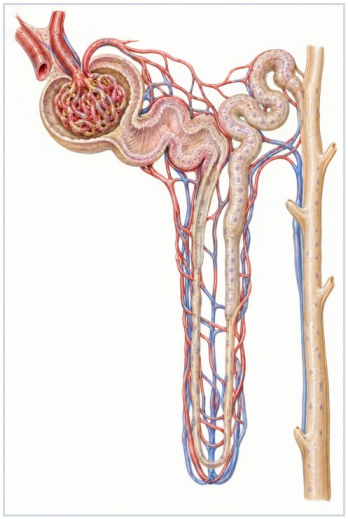
Nephron microanatomy as the memory map

Read epithelial structure, transport and drug action directly from the anatomical segment

RENAL CORPUSCLE
Afferent flow -> glomerular filtration
Bowman's space receives ultrafiltrate

PROXIMAL TUBULE
Brush border; bulk Na+, glucose, phosphate and bicarbonate handling

LOOP OF HENLE
Countercurrent architecture
NKCC2 in thick ascending limb



DISTAL NEPHRON
NCC then ENaC/ROMK regulation
Aldosterone and ADH shape output

MICROCIRCULATION
Efferent arteriole -> peritubular capillaries and vasa recta

AMT sequence: structure -> transporter -> electrolyte -> disease -> drug

AI-assisted original medical atlas artwork; synthetic educational image; human clinical review required

Figure REN-M01. Original AMT nephron memory map linking segment, transporter, physiology, inherited disorder and drug site.

CLINICAL PEARL
An ectopic or horseshoe kidney may be asymptomatic, but abnormal ascent or drainage increases the risk of obstruction, infection and stones.

MRCP TRAP
The right kidney is usually lower, but renal level varies with respiration and body habitus; avoid treating a textbook vertebral level as an imaging diagnosis.

AMT 60-SECOND RECALL
- Kidneys are retroperitoneal; the right usually lies lower.
- Glomeruli lie in the cortex; loops and collecting ducts extend into the medulla.
- The ureteric bud forms the collecting system; metanephric mesenchyme forms nephrons.
- Afferent enters and efferent leaves the glomerulus.
- Juxtamedullary nephrons and vasa recta preserve the medullary gradient.

2. Renal physiology and biochemistry

PART 1 CORE • ANATOMY → PHYSIOLOGY → PATHOLOGY → PHARMACOLOGY

2.1 GFR, renal blood flow and autoregulation

GFR reflects the filtration coefficient and net Starling forces across the glomerular capillary. Afferent tone, efferent tone, systemic perfusion and plasma oncotic pressure therefore alter filtration. Autoregulation stabilises renal blood flow across a physiological pressure range through myogenic responses and tubuloglomerular feedback.

CLINICAL CORRELATE: ACE inhibitors/ARBs reduce efferent arteriolar tone; NSAIDs reduce prostaglandin-mediated afferent dilation. During hypovolaemia, combining these with diuretics can precipitate haemodynamic AKI.

2.2 Tubular handling

Segment	Key function	Clinical correlate
Proximal tubule	Bulk Na+/water reabsorption; glucose, amino acids, bicarbonate	Fanconi syndrome; SGLT2 pharmacology; proximal RTA
Thick ascending limb	NKCC2 transport; water impermeable; medullary gradient	Loop diuretics; Bartter physiology
Distal convoluted tubule	NCC-mediated NaCl reabsorption; Ca2+ handling	Thiazides; Gitelman physiology
Collecting duct	ENaC, aldosterone, ADH-regulated water handling	Type 4 RTA; potassium disorders; diabetes insipidus

2.3 Water, sodium, potassium and acid-base

- ADH increases collecting-duct water permeability via aquaporin-2 insertion; osmolality and effective circulating volume regulate secretion.
- Aldosterone promotes distal sodium retention and potassium/hydrogen secretion; impaired effect contributes to hyperkalaemic type 4 RTA.
- Renal acid excretion depends on titratable acid, ammonium generation and bicarbonate reclamation/regeneration.

MRCP TRAP: Serum sodium is primarily a water-balance concentration, not a direct measure of total-body sodium. Oedematous patients may have excess total sodium but hyponatraemia.

2.4 Endocrine renal function

- Renin initiates RAAS signalling and links renal perfusion to vascular tone and sodium balance.
- Erythropoietin production supports erythropoiesis; deficiency contributes to CKD anaemia.
- Renal 1-alpha hydroxylation generates calcitriol; reduced activity and phosphate retention contribute to CKD-mineral bone disorder.

AMT MEMORY AID
Use the sequence filter -> reclaim -> build gradient -> fine tune -> final urine. At every nephron segment ask: which transporter, which hormone, which electrolyte and which drug?

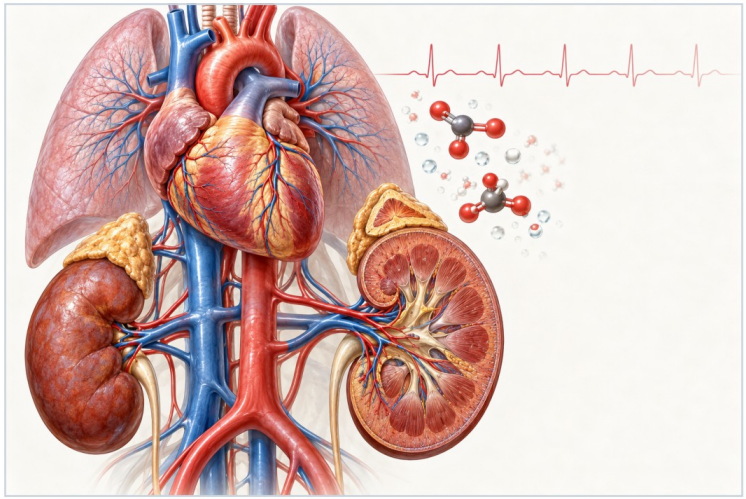
MRCP RAPID RECALL
Afferent constriction lowers renal blood flow and GFR. Moderate efferent constriction can support GFR while reducing renal blood flow; marked constriction eventually lowers GFR.

RAAS from renal trigger to systemic effect

Real organ anatomy anchors the endocrine sequence and pharmacological interruption points

RENIN TRIGGER
Reduced afferent stretch, reduced NaCl at macula densa, sympathetic drive

RENAL RESPONSE
Renin initiates angiotensin signalling and supports aldosterone release



TARGET ORGANS
Vasoconstriction, sodium retention, K+ secretion and thirst

PHARMACOLOGY
ACEi/ARB/MRA interrupt different steps; monitor creatinine and potassium

Do not memorise a floating cascade: attach each mediator to its source and target

AI-assisted original medical atlas artwork; synthetic educational image; human clinical review required

Figure REN-M02. Original AMT RAAS mechanism chain with examination-relevant pharmacology.

CLINICAL PEARL
Creatinine is convenient but imperfect: secretion, muscle mass, diet and drugs alter the relationship between creatinine and true GFR.

MRCP TRAP
ADH controls water permeability, not sodium retention directly. Aldosterone promotes sodium reabsorption with potassium and hydrogen secretion.

AMT 60-SECOND RECALL
- PCT performs bulk isosmotic reabsorption and reclaims most filtered bicarbonate.
- The thick ascending limb uses NKCC2 and is water-impermeable.
- The DCT uses NCC; thiazides increase calcium reabsorption.
- Principal cells use ENaC and secrete potassium under aldosterone influence.
- ADH inserts AQP2 into the collecting duct.
- Renal ammonium generation is central to chronic acid excretion.

3. Renal pharmacology

PART 1 CORE • ANATOMY → PHYSIOLOGY → PATHOLOGY → PHARMACOLOGY

Pharmacology is integrated with clinical use. Exact doses are version-sensitive and must be verified against the current BNF/SmPC at publication lock.

Drug/class	Mechanism & relevant PK	Main renal use	Major traps / toxicity
ACE inhibitor / ARB	Reduces angiotensin II signalling; renal elimination varies by agent	Albuminuric CKD, hypertension, cardiovascular protection	Creatinine rise, hyperkalaemia, hypotension; avoid routine dual RAAS blockade
SGLT2 inhibitor	Reduces proximal glucose/Na+ reabsorption; restores tubuloglomerular feedback	Eligible CKD with/without diabetes depending on indication	Initial eGFR dip, genital infection, volume depletion, euglycaemic DKA risk
Finerenone	Non-steroidal mineralocorticoid receptor antagonist	Selected type 2 diabetic CKD with albuminuria	Hyperkalaemia; interaction/renal criteria matter
Loop diuretic	Blocks NKCC2 in thick ascending limb	Oedema/volume overload; not routine “treatment” of AKI	Hypovolaemia, hypokalaemia, metabolic alkalosis, ototoxicity
Thiazide-like diuretic	Blocks NCC in distal convoluted tubule	Hypertension; selected oedema strategies	Hyponatraemia, hypokalaemia, gout
Sodium zirconium cyclosilicate / patiromer	GI potassium binders	Selected acute/persistent hyperkalaemia pathways	Not substitutes for IV calcium in ECG-toxic hyperkalaemia; interactions/timing differ
ESA / HIF-PHI	Stimulates erythropoiesis directly or via HIF pathway	Selected anaemia of CKD after iron assessment	Hypertension/thrombotic risk; do not normalise Hb routinely
Tacrolimus / ciclosporin	Calcineurin inhibition; CYP3A/P-gp substrate	Transplant immunosuppression	Nephrotoxicity, neurotoxicity, hyperkalaemia, major CYP interactions
Mycophenolate	IMPDH inhibition → reduced lymphocyte purine synthesis	Transplant immunosuppression	GI toxicity, cytopenias, infection, teratogenicity
Sirolimus/everolimus	mTOR inhibition	Selected transplant regimens	Dyslipidaemia, cytopenia, impaired wound healing, proteinuria

COMMON MRCP PHARMACOLOGY TRAP: A creatinine rise after RAAS blockade is not automatically “drug allergy”. Interpret magnitude, volume status, renal-artery disease, potassium and the clinical indication before stopping disease-modifying therapy.

AMT MEMORY AID
Predict renal drug effects in four steps: site of action -> intended physiological change -> electrolyte/acid-base consequence -> toxicity or interaction amplified by reduced GFR.

MRCP RAPID RECALL
Loop: NKCC2 and hypokalaemic alkalosis. Thiazide: NCC, hyponatraemia and hypokalaemia. ENaC/MR blockade: hyperkalaemia. Carbonic anhydrase inhibition: bicarbonate loss and acidosis.

CLINICAL PEARL
A drug may need dose reduction because of renal clearance even when it is not itself nephrotoxic.

MRCP TRAP
The combination of hypovolaemia, an NSAID and RAAS blockade can sharply reduce glomerular filtration by altering both afferent and efferent tone.

AMT 60-SECOND RECALL
- Match every diuretic to its nephron site and electrolyte pattern.
- Review eGFR, trend, volume state and interacting medicines before prescribing.
- NSAIDs impair prostaglandin-mediated afferent dilation.
- RAAS blockade reduces efferent arteriolar tone.
- Trimethoprim can raise creatinine and potassium without identical mechanisms.
- Verify final doses and interactions against the current BNF/SmPC.

Diuretic action on realistic nephron anatomy

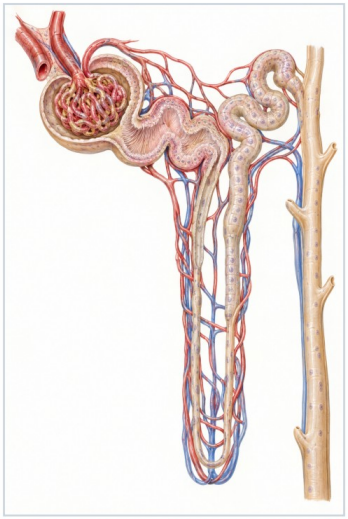
Drug class, epithelial segment and electrolyte consequence remain visible in one structure

PROXIMAL

Carbonic anhydrase inhibitors;
SGLT2 inhibitors modify proximal handling

THICK ASCENDING LIMB

Loop diuretics inhibit NKCC2;
watch volume, Na+, K+, Mg2+



DISTAL CONVOLUTED TUBULE

Thiazides inhibit NCC;
calcium handling differs from loop

COLLECTING DUCT

ENaC blockers and MR antagonists;
hyperkalaemia risk

Volume state + eGFR + interacting medicines determine the real bedside effect

AI-assisted original medical atlas artwork; synthetic educational image; human clinical review required

Figure REN-M11. Original AMT diuretic-site memory map.

4. Acute kidney injury (AKI)

PART 2 APPLICATION • PRESENTATION → DIFFERENTIAL → INVESTIGATION → INTERPRETATION → MANAGEMENT → COMPLICATION → NEXT STEP

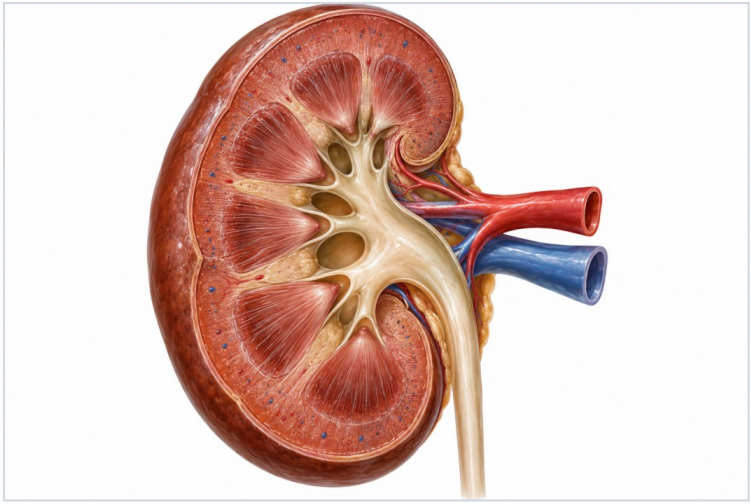
Definition: AKI is an abrupt decline in kidney function detected by creatinine change and/or reduced urine output. NICE uses a creatinine rise ≥26 micromol/L within 48 hours, ≥50% within 7 days, or urine output <0.5 mL/kg/hour for >6 hours in adults. (NICE, 2019).

AKI localisation on renal anatomy

Perfusion, vessels, parenchyma and drainage are examined around a true renal cross-section

PRE-RENAL
Hypovolaemia, sepsis, low effective arterial volume, altered glomerular tone

VASCULAR
Renal artery/vein disease, TMA, malignant hypertension, atheroembolism



INTRINSIC
Glomerular, tubular or interstitial injury; pigment, crystal and immune mechanisms

POST-RENAL
Stone, prostate, pelvic mass, catheter problem or solitary-kidney obstruction

Confirm baseline -> assess perfusion -> examine urine -> review medicines -> image drainage

AI-assisted original medical atlas artwork; synthetic educational image; human clinical review required

PART 2 APPLICATION — AKI: phenotype before label

- Confirm the trajectory: compare creatinine with baseline, quantify urine output and review timing of haemodynamic, toxic, inflammatory and obstructive insults.
- Localise: shock/volume loss and bland sediment favour pre-renal physiology; blood plus protein/casts suggests intrinsic disease; hydronephrosis or retention suggests obstruction.
- Act first on reversible threats: sepsis, hypovolaemia, nephrotoxins, hyperkalaemia, pulmonary oedema and obstruction. Do not delay decompression of an infected obstructed system.
- Escalate for refractory potassium/acidosis/overload, uraemic complications, rapidly progressive GN, suspected vasculitis/TMA or unexplained stage 3 AKI.

Figure REN-02. Original AMT AKI aetiology map: pre-renal, intrinsic, renal vascular and post-renal causes.

4.1 Aetiology and categorised causes

Category	Major causes	High-yield clues
Pre-renal	Haemorrhage/dehydration, sepsis, heart failure, cirrhosis, drug-related haemodynamic change	Volume loss, hypotension, low effective arterial volume, rapidly reversible physiology if treated early
Intrinsic - glomerular	ANCA vasculitis, anti-GBM disease, immune-complex GN, IgA disease	Haematuria, proteinuria, casts, systemic inflammatory clues
Intrinsic - tubular	Ischaemic or nephrotoxic ATN, pigment injury, crystal nephropathy	Granular casts; prolonged shock; toxins; rhabdomyolysis
Intrinsic - interstitial	Drug-induced AIN, infection, autoimmune/interstitial disease	Drug exposure, pyuria, systemic hypersensitivity clues may be absent
Renal vascular	Renal artery/vein occlusion, TMA, malignant hypertension, atheroembolism	Severe hypertension, thrombosis/embolism, livedo/eosinophilia in atheroembolism

Category	Major causes	High-yield clues
Post-renal	BPH, stones, tumour, clots/papillae, neurogenic bladder, retroperitoneal fibrosis	Bladder symptoms, hydronephrosis, bilateral disease/solitary kidney risk

4.2 Epidemiology and risk

AKI is common in acute illness and is associated with older age, CKD, diabetes, heart failure, liver disease, sepsis, hypovolaemia, nephrotoxic drugs and urological obstruction. Previous AKI increases future AKI and CKD risk.

4.3 Pathogenesis and pathology

- Pre-renal states initially reduce filtration without structural injury; prolonged hypoperfusion can progress to tubular injury.
- ATN produces tubular epithelial injury, loss of polarity, intraluminal obstruction and back-leak; recovery depends on resolution of insult and epithelial repair.
- Glomerular injury alters filtration barrier integrity, producing haematuria/proteinuria; crescents indicate severe proliferative injury in rapidly progressive GN.
- Interstitial inflammation can impair tubular function and GFR; fibrosis predicts incomplete recovery if injury persists.

4.4 Diagnosis: history, examination and investigations

Domain	Salient points
History	Fluid losses, infection, bleeding, heart/liver failure, urinary symptoms, new drugs/contrast, NSAIDs, ACEi/ARB, diuretics, antimicrobials, toxins, systemic rash/joints/haemoptysis, myeloma symptoms, transplant status.
Examination	Volume status, BP including hypotension/severe hypertension, JVP/oedema, sepsis source, rash/purpura, bladder distension, systemic vasculitic signs.
Labs	Serial creatinine/eGFR trend, urea/electrolytes, bicarbonate, FBC, CRP; CK if rhabdomyolysis; haemolysis screen if TMA; immunology/serology when phenotype supports it.
Urine	Dipstick blood/protein/leucocytes/nitrites/glucose; microscopy for RBC casts, granular casts, WBCs/crystals; culture when infection suspected.
Imaging	Ultrasound if obstruction risk or no clear cause; immediate (within 6 h) if pyonephrosis suspected; generally within 24 h if obstruction/no cause.
Special tests	ANCA, anti-GBM, complement, ANA/dsDNA, electrophoresis/free light chains, renal biopsy when intrinsic diagnosis remains uncertain or tissue diagnosis will alter therapy.

RED FLAG: AKI with haemoptysis, purpura, severe hypertension, rapidly rising creatinine, transplant status, stage 3 AKI, refractory complications or uncertain cause warrants urgent specialist involvement.

4.5 Treatment

- Treat the cause: restore perfusion when depleted, control sepsis, stop/review nephrotoxins, relieve obstruction, treat immune disease through specialist pathways.
- Monitor urine output, creatinine, potassium, acid-base status and fluid balance; avoid both persistent hypovolaemia and iatrogenic overload.
- Do not routinely use loop diuretics to “treat AKI”; reserve for clinically indicated volume overload. (NICE, 2019).
- Urgent RRT is considered for refractory hyperkalaemia, severe acidosis, pulmonary oedema/volume overload, selected toxins and uraemic complications; no creatinine value alone mandates dialysis.

PART 2 APPLICATION: A patient can be non-oliguric and still have obstruction. Persistent urine output does not exclude post-renal AKI.

4.6 Prognosis

Outcome depends on cause, severity, duration, comorbidity and recovery. AKI increases subsequent risk of recurrent AKI, CKD, cardiovascular events and death; kidney function should be reassessed after discharge.

AMT MEMORY AID
Localise AKI with three Ps: Perfusion, Parenchyma and Passage. Then check time course, urine, medicines and imaging rather than relying on a single fractional-excretion result.

MRCP RAPID RECALL
AKI is detected by creatinine change and/or urine output. Treat the cause and complications; no creatinine threshold alone mandates dialysis.

Renal imaging: choose the modality for the question

Synthetic CT, ultrasound and Doppler examples are educational rather than patient studies

NON-CONTRAST CT
First-line imaging for suspected renal colic in most adults; shows obstruction

CT / MR UROGRAPHY
Defines collecting systems and selected structural lesions; protocol matters



ULTRASOUND
Hydronephrosis, kidney size and bladder; operator and timing influence sensitivity

DOPPLER
Transplant perfusion, vessels and waveform interpretation in clinical context

Imaging supports localisation; it does not replace clinical assessment or specialist review

AI-assisted original medical atlas artwork; synthetic educational image; human clinical review required

Figure REN-M03. Synthetic educational CT, ultrasound and Doppler imaging examples; not patient studies.

CLINICAL PEARL
Urine microscopy can rapidly redirect the differential: casts and dysmorphic cells are active diagnostic data, not decorative findings.

MRCP TRAP
Diuretics can treat volume overload but do not reverse intrinsic AKI or improve recovery simply by increasing urine volume.

AMT 60-SECOND RECALL
- Compare with a reliable baseline and determine the time course.
- Assess perfusion and congestion together.
- Dip urine early; examine sediment when intrinsic disease is possible.
- Review nephrotoxins, recent contrast and new medicines.
- Relieve obstruction urgently when infection or threatened renal function is present.
- Escalate refractory potassium, acidosis, overload, uraemia or dialysable toxin exposure.

Renal replacement therapy in clinical context

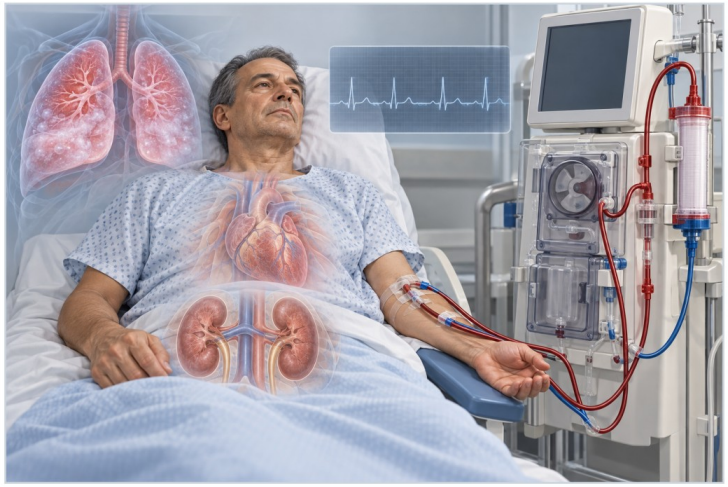
The indication, patient physiology and modality are considered together

REFRACTORY PHYSIOLOGY

Acidosis, hyperkalaemia and pulmonary oedema are treatment failures, not numbers

TOXIN / URAEMIA

Toxin properties and clinical syndrome determine extracorporeal benefit



MODALITY

Intermittent, continuous or peritoneal therapy depends on urgency and stability

REASSESS

Clinical response, biochemical trend, access and complications

Creatinine alone is not an indication; treat the immediate threat

AI-assisted original medical atlas artwork; synthetic educational image; human clinical review required

Figure REN-M14. Original AMT complication-led urgent kidney replacement therapy map.

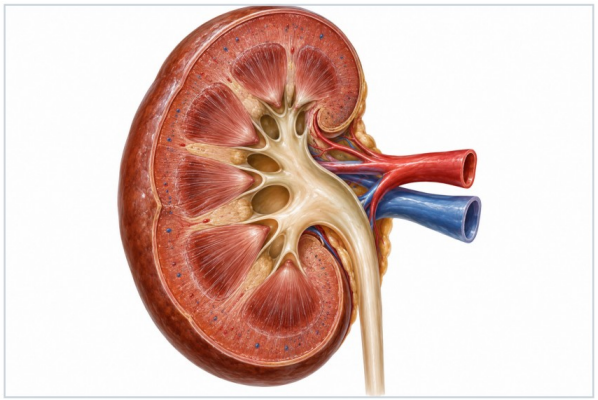
5. Chronic kidney disease (CKD)

PART 2 APPLICATION • PRESENTATION → DIFFERENTIAL → INVESTIGATION → INTERPRETATION → MANAGEMENT → COMPLICATION → NEXT STEP

Definition: abnormalities of kidney structure or function present for more than 3 months with implications for health. NICE includes markers of kidney damage or GFR <60 mL/min/1.73 m² on at least two occasions separated by at least 90 days.

CKD risk: renal structure plus function and albumin leak

Use eGFR, ACR and trajectory together; anatomy reminds you what the numbers represent



	A1 <3	A2 3-30	A3 >30
G1 >=90			
G2 60-89			
G3a 45-59			
G3b 30-44			
G4 15-29			
G5 <15			

Cause + G category + A category + trend = risk and referral context

AI-assisted original medical atlas artwork; synthetic educational image; human clinical review required

PART 2 APPLICATION — CKD: treat risk, not creatinine alone

- State cause + G category + ACR category; repeat unexpected deterioration and separate chronic progression from superimposed AKI.
- Layer disease modification: blood-pressure control, maximally tolerated ACE inhibitor/ARB when indicated, SGLT2 inhibition where licensed, diabetes and cardiovascular risk management.
- Use KFRE and clinical context for referral/planning. A low eGFR, heavy albuminuria, haematuria, resistant hypertension or rapid decline should trigger specialist assessment.
- Monitor potassium and renal function after haemodynamically active therapy; an early eGFR dip may be expected, but progressive deterioration requires review of volume, drugs and renovascular/obstructive causes.

Figure REN-03. Original AMT conceptual CKD G/A risk grid. Exact risk interpretation should use current NICE/KDIGO tools and patient context.

5.1 Causes

Category	Examples
Metabolic/endocrine	Diabetic kidney disease, hyperuricaemic/oxalate disorders
Vascular	Hypertensive nephrosclerosis, renovascular disease, ischaemic nephropathy
Glomerular/autoimmune	IgA nephropathy, membranous nephropathy, lupus nephritis, vasculitis
Tubulointerstitial/drug	Reflux nephropathy, chronic interstitial nephritis, lithium-related disease, analgesic injury
Congenital/genetic	ADPKD, Alport syndrome, CAKUT
Obstructive	Prostatic disease, stones, strictures, malignancy

Category	Examples
Other	Recurrent AKI, chronic infection, myeloma/monoclonal gammopathy-related disease

5.2 Classification and progression

- Classify by cause, GFR category G1-G5 and albuminuria category A1-A3. (NICE, 2021; KDIGO, 2024).
- Use trends rather than a single creatinine. ACR adds prognostic information even when eGFR is stable.
- NICE supports 4-variable KFRE for 5-year kidney-failure risk; >5% is one adult referral criterion. (NICE, 2021).

5.3 Evidence-based treatment

- Control blood pressure and cardiovascular risk; use ACEi/ARB where indicated by diabetes/albuminuria, with potassium and renal-function monitoring.
- Use SGLT2 inhibitors in eligible CKD according to current NICE technology-appraisal and licensing criteria. (NICE, 2021).
- Consider finerenone for selected stage 3-4 CKD with type 2 diabetes and albuminuria in line with NICE criteria. (NICE, 2021).
- Address smoking, weight, glycaemic control, vaccination, medication safety, avoidance of nephrotoxins and sick-day risk where locally recommended.
- Treat anaemia, CKD-MBD, acidosis, hyperkalaemia and fluid overload using stage-appropriate pathways.
- Plan kidney replacement therapy or conservative kidney management early when progression risk is substantial, incorporating patient goals.

5.4 Anaemia of CKD

Evaluate iron status and alternative causes. Optimise iron before or alongside ESA therapy. NICE advises against routine erythropoietin measurement and against normalising haemoglobin with ESA therapy; treatment aims are individualised and version-sensitive.

5.5 CKD-mineral bone disorder

Phosphate retention, reduced calcitriol and secondary hyperparathyroidism interact with calcium balance and bone turnover. Interpret serial phosphate, calcium and PTH in the context of CKD stage, dialysis status and treatment rather than reacting to a single result.

5.6 Prognosis

Prognosis depends on cause, eGFR, albuminuria, slope of decline, cardiovascular disease, diabetes, age and acute insults. Cardiovascular events are a major competing risk, so kidney protection and cardiovascular protection are inseparable.

AMT MEMORY AID

Classify CKD by Cause + GFR category + Albuminuria category, then estimate progression risk. Remember complications as Blood, Bone, Buffer, Balance and cardiovascular Burden.

MRCP RAPID RECALL

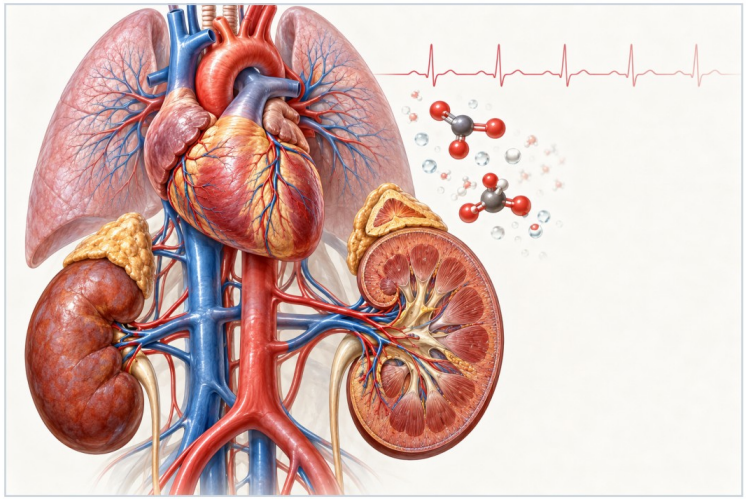
A stable eGFR does not neutralise albuminuria risk. Trends, ACR and validated risk prediction outperform a single creatinine value.

CKD complications on real organ anatomy

Cardiorenal, pulmonary, endocrine and metabolic effects are anatomically anchored

BLOOD / IMMUNITY
EPO deficiency, iron restriction, inflammation and infection risk

BONE / MINERAL
Phosphate retention, low calcitriol, secondary hyperparathyroidism



CARDIOVASCULAR
Hypertension, volume burden, vascular disease and heart failure

ACID-BASE / POTASSIUM
Reduced excretion and reserve; medicines may expose vulnerability

Treat the patient and trajectory, not eGFR in isolation

AI-assisted original medical atlas artwork; synthetic educational image; human clinical review required

Figure REN-M07. Original AMT CKD complication memory map.

CLINICAL PEARL
Kidney-protective therapy is layered: blood pressure and RAAS management, SGLT2 inhibition where indicated, diabetes/cardiovascular care, smoking cessation and avoidance of nephrotoxins.

MRCP TRAP
Do not label chronicity from one abnormal blood test. CKD requires persistence or other evidence of chronic structural damage.

AMT 60-SECOND RECALL
- CKD requires >3 months of structural or functional abnormality.
- Use cause, G category and A category.
- ACR adds risk information at every eGFR level.
- KFRE supports referral and kidney-replacement planning in appropriate adults.
- Monitor potassium and creatinine after relevant RAAS changes.
- Treat anaemia only after assessing iron and alternative causes.
- Plan replacement therapy from symptoms, trajectory and shared decisions, not eGFR alone.

6. Glomerular disease: pattern first

PART 2 APPLICATION • PRESENTATION → DIFFERENTIAL → INVESTIGATION → INTERPRETATION → MANAGEMENT → COMPLICATION → NEXT STEP

Pattern	Typical features	Examples
Nephritic	Haematuria, casts, hypertension, impaired GFR, usually sub-nephrotic proteinuria	IgA nephropathy, post-infectious GN, ANCA GN, anti-GBM disease

Pattern	Typical features	Examples
Nephrotic	Heavy proteinuria, hypoalbuminaemia, oedema; thrombosis/infection risk	Minimal change, FSGS, membranous nephropathy, diabetic kidney disease, amyloid
Pulmonary-renal	GN plus pulmonary haemorrhage/infiltrates	Anti-GBM disease, ANCA-associated vasculitis
Monoclonal	Proteinuria/AKI with plasma-cell or paraprotein clues	Myeloma cast nephropathy, AL amyloidosis, MGRS

FREQUENTLY CONFUSED: IgA nephropathy often causes haematuria close to a mucosal infection; post-infectious GN typically follows infection after a latent period and may have low complement.

AMT MEMORY AID

Read glomerular disease in three layers: urine phenotype -> systemic/complement clues -> biopsy through LM, IF and EM. Each view answers a different question.

MRCP RAPID RECALL

Nephritic means inflammatory haematuria with impaired filtration; nephrotic means severe barrier protein leak. Real patients may show mixed patterns.

Glomerular disease: phenotype to tissue pattern

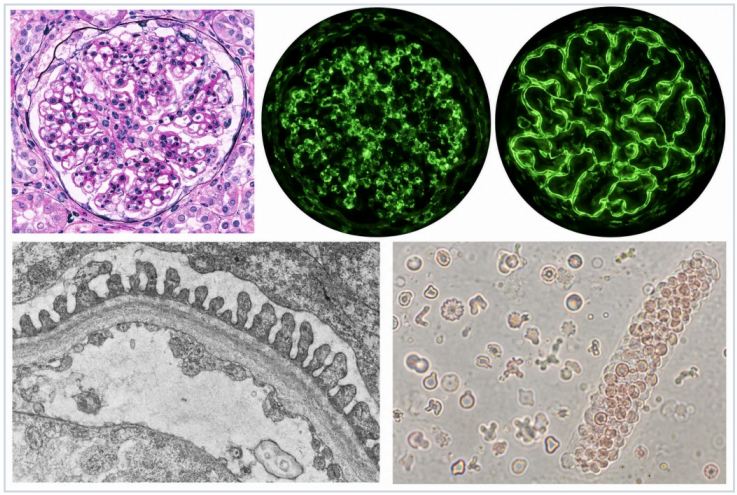
Authentic-style LM, IF, EM and urine microscopy replace abstract symbols

LIGHT MICROSCOPY

Cellularity, sclerosis, necrosis, crescents and capillary architecture

IMMUNOFLUORESCENCE

Granular, linear or pauci-immune patterns refine mechanism



URINE MICROSCOPY

Dysmorphic red cells and casts localise bleeding to the glomerulus

ELECTRON MICROSCOPY

Deposit site, GBM contour and podocyte foot-process change

Integrate presentation, complement, serology and all three biopsy modalities

AI-assisted original medical atlas artwork; synthetic educational image; human clinical review required

PART 2 APPLICATION — glomerular disease: syndrome → serology → biopsy

- Nephritic phenotype: haematuria, dysmorphic red cells/casts, hypertension and AKI. Nephrotic phenotype: heavy proteinuria, hypoalbuminaemia, oedema and thrombotic/infective risk.
- Send targeted serology before immunosuppression: complements, ANA/dsDNA, ANCA, anti-GBM, hepatitis/HIV and monoclonal studies guided by phenotype.
- Pulmonary haemorrhage plus AKI is a time-critical pulmonary–renal syndrome: involve nephrology urgently; treatment may precede tissue confirmation when immediately life-threatening.
- Biopsy interpretation must integrate LM, IF and EM. A pattern is not a complete diagnosis; age, drugs, infection, malignancy and systemic disease change the differential.

Figure REN-M06. Original AMT nephritic-nephrotic and LM-IF-EM pattern map.

CLINICAL PEARL
Pulmonary haemorrhage plus active urinary sediment is a time-critical pulmonary-renal syndrome until proved otherwise.

MRCP TRAP
A negative dipstick for blood or protein at one time point does not exclude evolving glomerulonephritis; repeat and quantify in the correct clinical context.

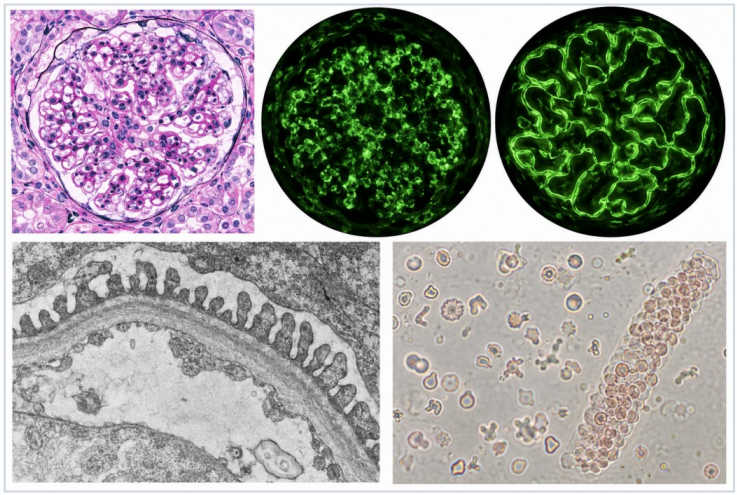
AMT 60-SECOND RECALL
- Dysmorphic red cells and red-cell casts support glomerular bleeding.
- Quantify proteinuria; do not rely on dipstick grading alone.
- Low complement narrows the immune-complex differential but is not diagnostic alone.
- Linear IF suggests anti-GBM disease; pauci-immune patterns support ANCA-associated disease.
- Granular IF requires immunoglobulin/complement and deposit-location interpretation.
- LM, IF and EM must be integrated with presentation and serology.

Complement pattern recognition beside real IF tissue

Complement narrows the differential; it never replaces urine microscopy, serology or biopsy

LOW C3
Post-infectious GN, C3 glomerulopathy, MPGN patterns and selected lupus cases

LOW C3 + C4
Immune-complex disease including lupus and cryoglobulinaemia



USUALLY NORMAL
ANCA vasculitis, anti-GBM disease, IgA nephropathy and minimal change

DO NOT STOP AT COMPLEMENT
Biopsy pattern and serology remain essential to final diagnosis

Pattern -> mechanism -> confirmatory test; avoid treating complement as a diagnosis

AI-assisted original medical atlas artwork; synthetic educational image; human clinical review required

Figure REN-M13. Original AMT complement-pattern comparison.

6.1 Nephrotic syndrome: diagnostic work-up and complications

Nephrotic syndrome combines heavy proteinuria (often urine protein:creatinine ratio >300 mg/mmol or approximately >3.5 g/day), hypoalbuminaemia and oedema; hyperlipidaemia is common but is not required for diagnosis. Quantify proteinuria, inspect urine sediment and review GFR trend. Proteinuria alone is not synonymous with the complete syndrome. Albumin thresholds and nephrotic-range definitions vary by laboratory and guideline. (KDIGO GD 2021, general principles).

At presentation determine whether oedema reflects salt retention and intravascular depletion, whether a rapidly rising creatinine or active sediment requires urgent biopsy, and whether a secondary driver (diabetes, systemic autoimmunity, infection, malignancy, drugs or monoclonal gammopathy) is present. History includes onset, weight, drug exposure (especially NSAIDs), diabetes, systemic symptoms, thromboembolism and infection risks. Examine blood pressure, volume status, retinopathy, skin/joints and thrombosis signs.

Investigation	What it resolves / caution
Urinalysis; UPCR or ACR; microscopy	Confirm and quantify protein; red-cell casts/dysmorphic erythrocytes suggest inflammatory overlap.
Creatinine/eGFR, albumin, lipids, blood count	Baseline disease and complications; CKD can distort apparent protein excretion.
C3/C4, ANA/dsDNA, ANCA, anti-GBM as indicated	Choose tests from phenotype: these are not a universal unselected screening panel.
HBV/HCV/HIV; immunoglobulins, SPEP/free light chains	Search for infection or monoclonal process when history, age or biopsy warrants.
Anti-PLA2R; kidney ultrasound; kidney biopsy	Anti-PLA2R supports primary membranous disease in the right context; biopsy often defines diagnosis, activity and chronicity, and can reveal concurrent disease.

Supportive management: individualise sodium restriction and loop diuresis to clinical volume state; avoid rapid intravascular depletion and AKI. Control blood pressure and proteinuria with ACE inhibitor or ARB when safe; assess potassium/creatinine after initiation or dose change. Consider SGLT2 inhibitor in eligible CKD according to current kidney guidance, not as a replacement for lesion-specific therapy. Vaccinate and assess infection risk before immunosuppression; address dyslipidaemia and cardiovascular risk. Decide venous-thromboembolism prophylaxis individually (histology, albumin, bleeding risk, planned biopsy, mobility); membranous nephropathy has relatively high risk. Treat thrombosis when present. (KDIGO GD 2021; CKD 2024).

MRCP RAPID RECALL • Persistent nephrotic syndrome plus new dyspnoea, unilateral leg swelling or flank pain raises venous thromboembolism; escalating oedema with hypotension can coexist with low effective circulating volume.

6.2 Primary nephrotic lesions: biopsy pattern, mechanism and treatment

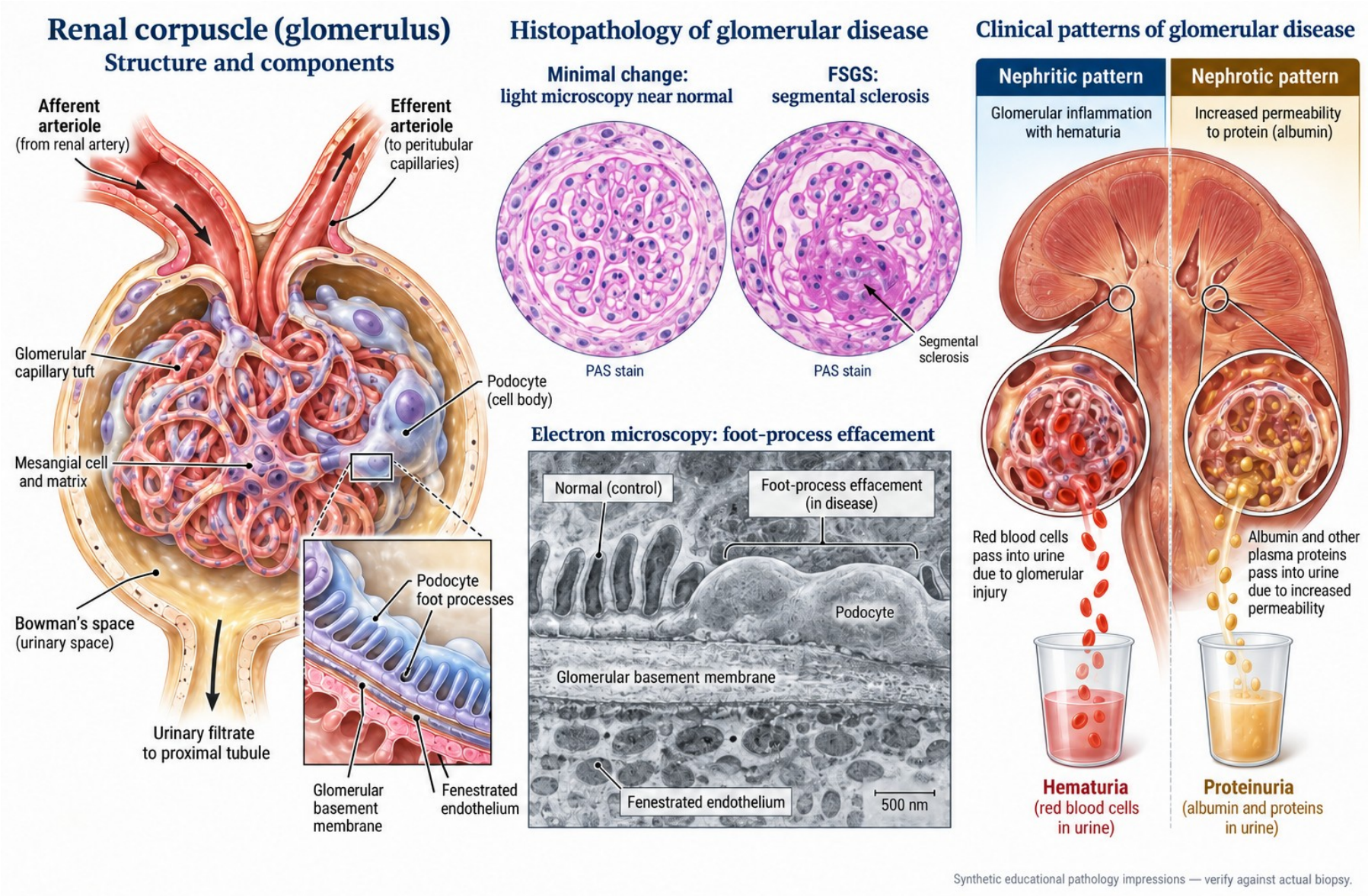


Figure REN-M17. Synthetic educational renal corpuscle, podocyte and glomerular pathology impressions. Histology panels are illustrations, not biopsy specimens; inspect labels against actual LM/IF/EM and clinical context.

Lesion / phenotype	LM • IF • EM / key discriminator	Treatment pathway and special cautions
Minimal change disease (adult)	LM often nearly normal; IF usually negative; EM diffuse foot-process effacement. May present with abrupt marked oedema and AKI.	Biopsy adults in usual practice; oral glucocorticoids are initial disease-specific therapy after secondary causes reviewed. Frequently relapsing, steroid-dependent or contraindicated cases: specialist steroid-sparing agent (rituximab, calcineurin inhibitor, cyclophosphamide or mycophenolate) chosen by risk. Check for NSAIDs/lymphoma.
Primary FSGS	Segmental scarring in a subset of glomeruli; IF may show nonspecific IgM/C3 trapping; EM often extensive podocyte effacement in primary nephrotic presentation.	Separate primary nephrotic FSGS from secondary/adaptive, genetic and FSGS of undetermined cause. Steroids for selected primary disease; calcineurin inhibitor when steroid resistant/intolerant under renal care. Treat secondary drivers with supportive care; do not give empiric immunosuppression merely because biopsy says FSGS.
Membranous nephropathy	GBM thickening/spikes; granular capillary-wall immune deposits; subepithelial deposits on EM. Anti-PLA2R positive in many primary cases.	Risk stratify using proteinuria trajectory, eGFR, albumin and anti-PLA2R, then choose observation/supportive care or immunosuppression (rituximab, cyclophosphamide with glucocorticoids, or calcineurin inhibitor strategies). Screen for secondary causes; thrombosis risk merits individual assessment.
Secondary nephrotic disease	Diabetic nodular/diffuse sclerosis, amyloid deposits, lupus class V or infection/drug-related pattern depend on biopsy.	Treat underlying disease and kidney risk; amyloid requires typing before clone-directed therapy; lupus V with nephrotic syndrome may require immunosuppression. Biopsy interpretation must be matched to diabetes duration, retinopathy and serology.
Diabetic kidney disease	Often persistent albuminuria with diabetic vascular/glomerular lesions; abrupt nephrotic onset, active sediment or rapid GFR loss suggests an additional lesion.	Optimise glucose, blood pressure and cardiovascular risk; RAAS blockade and eligible kidney-protective agents. Biopsy if an atypical phenotype changes management.
AL/AA amyloidosis and MGRS	Congo-red-positive amyloid or monoclonal deposit pattern requires typing; assess light chains, immunofixation and systemic disease.	AL or other MGRS needs haematology-led clone-directed therapy after tissue diagnosis; AA needs control of its inflammatory or infective driver.
Lupus membranous nephritis (class V)	Immune-complex deposits and lupus serology; check for concurrent proliferative III/IV disease, which changes urgency and regimen.	Supportive care; nephrotic proteinuria may need glucocorticoid plus mycophenolate and/or a calcineurin-based regimen under KDIGO LN 2024.

Follow response with proteinuria, serum albumin, creatinine, weight and adverse effects; protein reduction may lag immunological response (especially anti-PLA2R in membranous disease). Specify remission/relapse criteria for the particular lesion. A biopsy pattern is a description, not always an aetiological diagnosis. (KDIGO GD 2021, current 2024–2025 chapter updates).

AMT MEMORY AID • Read the biopsy in three views: LM = architecture and activity; IF = immune-deposit location/pattern; EM = deposit compartment and podocyte injury. Pair these with urine sediment and clinical tempo before selecting treatment.

6.3 Nephritic and rapidly progressive glomerulonephritis

Nephritic presentation is glomerular haematuria, variable proteinuria, hypertension, reduced GFR and sometimes oedema; red-cell casts support a glomerular inflammatory process. A rapidly rising creatinine over days to weeks with active sediment is an emergency: send directed serology and arrange urgent nephrology review/biopsy, without delaying time-critical treatment of pulmonary haemorrhage. Consider anti-GBM, ANCA vasculitis, lupus, infection-related GN and immune-complex disease; microangiopathy can mimic the presentation.

Disease / clue	Key tests and biopsy	Disease-specific action / trap
IgA nephropathy / IgA vasculitis	Synpharyngitic macroscopic haematuria, proteinuria; biopsy mesangial IgA. Assess risk by eGFR/proteinuria, exclude secondary causes.	Optimised supportive kidney care and risk-specific therapies under current KDIGO 2025 IgAN guidance; immunosuppression is not routine for all mesangial IgA. Systemic vasculitis adds skin/GI/joint assessment.
ANCA-associated pauci-immune GN	PR3/MPO-ANCA and systemic features; necrotising/crescentic biopsy with little/no immune staining.	Induction with glucocorticoids plus rituximab or cyclophosphamide; avacopan may reduce glucocorticoid exposure in selected regimens. Assess infection and extra-renal disease; maintenance after remission. (KDIGO AAV 2024).

Anti-GBM disease	Anti-GBM antibody; linear IgG on IF; pulmonary haemorrhage may occur.	Urgent specialist-led plasma exchange plus glucocorticoid and cyclophosphamide when appropriate; prognosis depends strongly on presentation and biopsy. Do not wait for final pathology when highly suspected. (KDIGO GD 2021).
Infection-related GN	During/after infection, often low C3; biopsy immune deposits; cultures and focus search.	Treat infection/source first. Immunosuppression usually avoided unless a carefully established separate indication.

6.4 Systemic disease and the kidney: SLE, infections and monoclonal disease

Lupus nephritis can present with haematuria, proteinuria, nephrotic syndrome or silent urinary abnormalities. In SLE assess urine ACR/UPCR and sediment, eGFR, complement, anti-dsDNA, blood pressure and extra-renal activity; biopsy typically when clinically significant proteinuria or unexplained GFR decline warrants classification. Class III/IV proliferative lesions generally require glucocorticoids plus mycophenolic acid analogues or low-dose IV cyclophosphamide; selected patients may receive add-on belimumab or a calcineurin inhibitor. Class V with nephrotic syndrome needs an individualised immunosuppressive approach; class VI reflects irreversible sclerosis rather than active immunosuppression target. Medication selection depends on eGFR, chronicity, fertility/pregnancy, infection risk and access. (KDIGO LN 2024).

Hepatitis B can be linked to membranous and other immune-complex lesions; hepatitis C to cryoglobulinaemic MPGN; HIV to collapsing FSGS or immune-complex lesions. Infection-related GN can arise with endocarditis, staphylococcal infection or other persistent foci. Send organism-directed tests, cultures and cardiac assessment when phenotype supports them, then treat the infection; consider antiviral or immunosuppressive strategies only with disease-specific specialist guidance. Diabetes, systemic amyloidosis and monoclonal gammopathy of renal significance can cause proteinuric or rapidly progressive disease; renal biopsy and deposit typing can change therapy even with a small circulating clone.

Pattern clue	Targeted renal/systemic interpretation
Low C3 alone or C3 dominant IF	Infection-related GN and complement-mediated disease; correlate with course and serial complement recovery.
Low C3 and C4 with systemic features	Think immune-complex disease such as lupus or cryoglobulinaemia; use ANA/dsDNA, hepatitis studies, rheumatoid factor/cryoglobulin protocol as indicated.
Pulmonary haemorrhage + active sediment	Anti-GBM or ANCA vasculitis until assessed urgently; clinical stability changes biopsy and treatment timing.
Monoclonal protein with renal lesion	Do not assume incidental MGUS: biopsy typing and haematology-nephrology discussion can identify MGRS.

AMT 60-SECOND RECALL • Quantify protein and inspect sediment; nephrotic is a syndrome, FSGS a lesion; MCD often normal LM with diffuse foot-process effacement; membranous has subepithelial deposits; primary versus secondary FSGS alters immunosuppression; nephritic rapid GFR loss needs urgent biopsy/serology; low complement narrows immune-complex/complement causes; lupus classification guides therapy; infection-related GN needs source treatment; assess thrombosis, infection and drug toxicity in nephrotic disease.

7. Tubular, interstitial and electrolyte disorders

7.1 Confirm, classify and treat renal tubular acidosis

First confirm persistent metabolic acidosis with pH, bicarbonate, chloride, potassium and albumin-adjusted anion gap; review medicines, diarrhoea, kidney function, ketones and lactate. A normal anion gap raises gastrointestinal bicarbonate loss or impaired renal acid excretion. Urinary ammonium is best estimated using an available direct assay or an appropriately interpreted urine osmolal gap; urine anion gap is unreliable in several settings, including unmeasured urinary anions. A single urine pH does not diagnose RTA and is confounded by UTI, volume depletion and alkali therapy.

Disorder and mechanism	Evaluation	Management and monitoring
Distal type 1: impaired distal H+ secretion; often autoimmune, inherited or drug related; hypokalaemia, calcium phosphate stones/nephrocalcinosis	Persistently inappropriate urine alkalinity in systemic acidosis; exclude urease UTI; assess urine citrate, calcium, imaging and autoimmune/drug context.	Oral alkali, often potassium citrate if hypokalaemic and stones present; replace K+ with close monitoring. Treat underlying cause; monitor bicarbonate, K+, stone risk, nephrocalcinosis and bone health.
Proximal type 2: impaired HCO3− reclamation; isolated or Fanconi syndrome	Urine pH may fall once plasma HCO3− drops. Check glycosuria with normal plasma glucose, phosphate and urate losses, aminoaciduria, medicines/toxins, paraprotein and inherited disease.	Treat cause; alkali requirement may be higher and can worsen urinary K+ loss. Replace K+, phosphate and vitamin D as individually indicated; monitor volume, electrolytes and bone effects.
Type 4: hypoaldosteronism or resistance; hyperkalaemia impairs ammonium generation	Assess diabetes, CKD, obstruction and ACEi/ARB, NSAID, heparin, trimethoprim or CNI exposure; consider renin/aldosterone only when clinically useful.	Correct hyperkalaemia and reversible drivers; adjust offending medicines with risk assessment; diuretic and/or bicarbonate when clinically indicated. Fludrocortisone is a specialist option for selected proven deficiency, limited by fluid retention and hypertension.
MRCP TRAP A high urine pH during acidosis supports distal disease only in the right context. Proximal RTA can acidify urine at low plasma bicarbonate; type 4 often has urine pH below 5.5 despite impaired ammonium		

excretion.

AMT 60-SECOND RECALL Confirm pH and anion gap; exclude gut loss and CKD. Distal: stones plus hypokalaemia; proximal: Fanconi features and greater alkali need; type 4: hyperkalaemia. Treat cause, replace deficits and monitor response.

PART 2 APPLICATION • PRESENTATION → DIFFERENTIAL → INVESTIGATION → INTERPRETATION → MANAGEMENT → COMPLICATION → NEXT STEP

Disorder	Mechanism	Key clue
Type 1 distal RTA	Failure of distal H+ secretion	Normal-gap acidosis, hypokalaemia, urine pH inappropriately high, stones
Type 2 proximal RTA	Reduced proximal bicarbonate reabsorption	Normal-gap acidosis, often Fanconi features
Type 4 RTA	Reduced aldosterone effect	Normal-gap acidosis with hyperkalaemia
Acute interstitial nephritis	Immune-mediated interstitial inflammation, often drug related	AKI + pyuria; rash/eosinophilia may be absent
Fanconi syndrome	Generalised proximal tubular dysfunction	Glycosuria without hyperglycaemia, phosphate/amino-acid/bicarbonate wasting

AMT MEMORY AID

For normal-gap metabolic acidosis, ask gut loss or kidney loss; then use potassium and urine acidification to localise the renal tubular defect.

MRCP RAPID RECALL

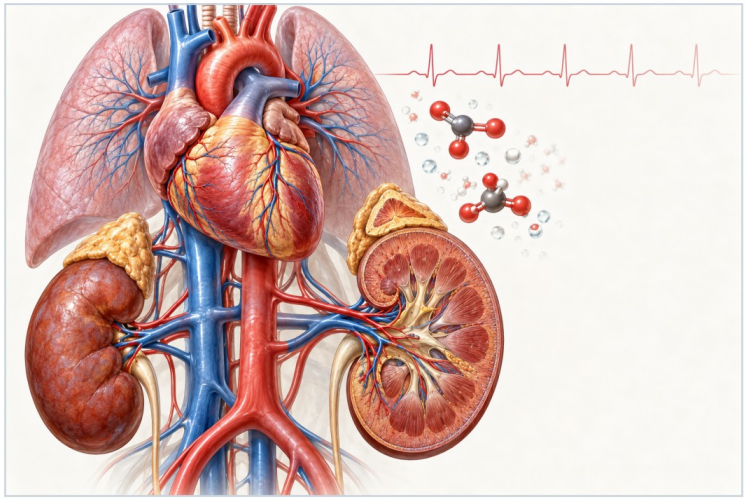
Type 1 and type 2 RTA usually lower potassium. Type 4 RTA raises potassium through low aldosterone effect and impaired ammonium excretion.

Acid-base physiology across lung, heart and kidney

Real organ relationships support a disciplined interpretation ladder

RESPIRATORY
PaCO2 direction and expected compensation

METABOLIC
Bicarbonate generation, reclamation and net acid excretion



INTEGRATE
pH -> primary process -> compensation -> anion gap -> delta relationship

CLINICAL CONTEXT
Ventilation, perfusion, renal function, ketones, lactate and toxins

Metabolic acidosis check: expected PaCO2 about 1.5 x HCO3- + 8 +/- 2

AI-assisted original medical atlas artwork; synthetic educational image; human clinical review required

PART 2 APPLICATION — acid–base: calculate, compensate, then explain

- Confirm pH and primary process; calculate anion gap and correct for albumin when relevant. A normal pH can conceal a mixed disorder.
- Test compensation rather than assuming it: respiratory compensation in metabolic acidosis is predictable; excess or inadequate change identifies a second process.
- Raised-gap acidosis: lactate, ketones, renal failure and toxins dominate. Normal-gap acidosis: gastrointestinal bicarbonate loss, RTA, saline load and early renal failure.
- Treat the cause and circulation; bicarbonate is not a reflex treatment for every acidaemia. Severe refractory acidosis with AKI may require critical care and renal replacement therapy.

Figure REN-M04. Original AMT acid-base diagnostic ladder.

CLINICAL PEARL
Correct hypomagnesaemia when hypokalaemia remains refractory; potassium replacement alone may repeatedly fail.

MRCP TRAP
Urine pH is context dependent. Type 2 RTA can produce acidic urine after plasma bicarbonate has fallen below the reabsorptive threshold.

AMT 60-SECOND RECALL
- Confirm the disturbance and exclude sampling artefact.
- Use serum and urine context to distinguish redistribution from true gain or loss.
- Normal-gap acidosis suggests bicarbonate loss or impaired renal acid handling.
- Type 1 RTA: distal H+ secretion failure, stones and hypokalaemia.
- Type 2 RTA: proximal bicarbonate wasting, often within Fanconi syndrome.
- Type 4 RTA: hyperkalaemia with aldosterone deficiency or resistance.

8. Hyperkalaemia: emergency framework

PART 2 APPLICATION • PRESENTATION → DIFFERENTIAL → INVESTIGATION → INTERPRETATION → MANAGEMENT → COMPLICATION → NEXT STEP

Treat the patient and ECG, not merely the number. Confirm pseudohyperkalaemia when safe, but do not delay emergency treatment when severe hyperkalaemia or ECG toxicity is present. (UK Kidney Association, 2026).

Goal	Typical approach	What it does
Stabilise myocardium	IV calcium in ECG-toxic hyperkalaemia	Reduces arrhythmic risk; does not lower K+
Shift K+ into cells	Insulin + glucose; beta-agonist adjunct in selected patients	Temporarily lowers serum K+
Remove K+	Diuresis if appropriate, potassium binders, dialysis when needed	Reduces total-body potassium
Prevent recurrence	Review RAAS drugs, diet, acidosis, constipation, diabetes, kidney function	Addresses drivers and enables disease-modifying therapy when possible

EVIDENCE UPDATE: UK Kidney Association acute hyperkalaemia guidance was updated in July 2026. NICE published updated sodium zirconium cyclosilicate guidance (TA1148) in April 2026. Dose-level emergency content requires pharmacy verification at publication lock.

AMT MEMORY AID

Manage dangerous hyperkalaemia in sequence: verify and monitor -> protect the myocardium when indicated -> shift potassium into cells -> remove potassium -> find and reverse the cause.

MRCP RAPID RECALL

Calcium protects the heart but does not lower serum potassium. Insulin/glucose shifts potassium but does not remove it. Definitive removal may require binders, diuresis or dialysis depending on context.

Renal tubular acidosis mapped to real nephron microanatomy

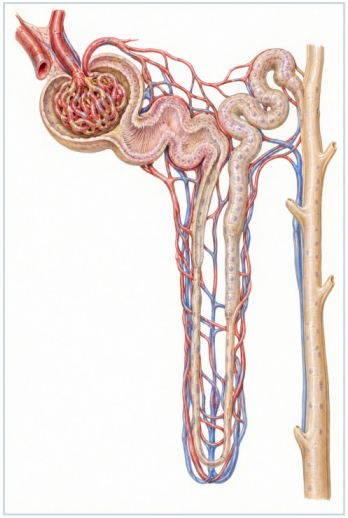
Epithelial structure and segment location separate proximal, distal and aldosterone-related defects

TYPE 2 PROXIMAL

Reduced bicarbonate reclamation;
Fanconi associations

TYPE 1 DISTAL

Impaired distal acid secretion;
high urine pH, stones, hypokalaemia



TYPE 4

Hypoaldosteronism or resistance;
hyperkalaemia and impaired NH4+

LOCALISE FIRST

Normal-gap acidosis -> potassium ->
urine response -> nephron segment

Do not diagnose RTA before excluding diarrhoea, low GFR and drug effects

AI-assisted original medical atlas artwork; synthetic educational image; human clinical review required

Figure REN-M05. Original AMT RTA and potassium pattern map.

CLINICAL PEARL
Rebound occurs because temporising therapies redistribute potassium. Repeat potassium and glucose checks are part of treatment, not optional follow-up.

MRCP TRAP
A normal ECG does not exclude life-threatening hyperkalaemia; interpret potassium level, rate of rise, symptoms, renal function and ECG together.

AMT 60-SECOND RECALL

- Repeat an unexpected result promptly without delaying treatment when the patient is unstable.
- Obtain ECG monitoring in severe or clinically concerning hyperkalaemia.
- Use IV calcium for ECG toxicity according to the emergency guideline.
- Insulin/glucose is effective but requires prolonged glucose surveillance.
- Nebulised beta-agonist is an adjunct and response varies.
- Stop drivers and plan potassium removal; dialyse when refractory or otherwise indicated.

9. Stones, inherited disease and obstruction

PART 2 APPLICATION • PRESENTATION → DIFFERENTIAL → INVESTIGATION → INTERPRETATION → MANAGEMENT → COMPLICATION → NEXT STEP

Condition	High-yield features	Management principle
Calcium oxalate stone	Most common; hypercalciuria, enteric hyperoxaluria, low urine volume	Hydration; investigate recurrent/high-risk stone disease
Uric acid stone	Acidic urine; radiolucent on plain film but visible on non-contrast CT	Urine alkalinisation and metabolic risk management where appropriate

Condition	High-yield features	Management principle
Struvite stone	Urease-positive infection; alkaline urine; staghorn calculus	Treat infection and address stone burden
Cystinuria	Inherited dibasic amino-acid transport defect	High fluids, urine alkalinisation, specialist therapy
ADPKD	Bilateral cystic kidneys, hypertension, liver cysts, selected aneurysm risk	BP/CKD care; selected tolvaptan eligibility; family/genetic counselling

AMT MEMORY AID
Stone questions become easier when you connect crystal shape -> urine pH -> radiological behaviour -> cause -> prevention.

MRCP RAPID RECALL
An infected obstructed collecting system is a drainage emergency. Antibiotics alone cannot reliably sterilise an obstructed system.

Renal stone recognition by anatomy and microscopy

True crystal morphology, urinary-tract position and clinical context replace outline icons

CALCIUM OXALATE
Envelope/dumbbell forms; common; consider hypercalciuria or enteric causes

URIC ACID / STRUVITE
Rhomboids in acidic urine; coffin lids with urease-positive infection



CYSTINE
Hexagonal crystals suggest inherited dibasic amino-acid transport defect

OBSTRUCTED INFECTION
Analgesia, sepsis assessment, CT when indicated and urgent drainage

Match composition and urine chemistry to prevention after the acute episode

AI-assisted original medical atlas artwork; synthetic educational image; human clinical review required

PART 2 APPLICATION — stone patient: pain control is only step one

- Non-contrast CT KUB is the usual adult diagnostic test; ultrasound is preferred when radiation avoidance matters. Check renal function, urinalysis and pregnancy status where relevant.
- Fever/sepsis, anuria, solitary kidney, bilateral obstruction, uncontrolled pain/vomiting or deteriorating renal function requires urgent urological discussion.
- An infected obstructed kidney needs prompt drainage plus antimicrobials—not medical expulsive therapy alone.

- Recurrent/high-risk stones merit metabolic assessment: stone analysis, serum calcium/urate/electrolytes and appropriately collected urine studies; prevention follows composition and mechanism.

Figure REN-M08. Original AMT renal-stone recognition grid.

CLINICAL PEARL
Non-contrast CT detects most stones, including uric-acid stones that may be radiolucent on plain radiography.

MRCP TRAP
Visible haematuria and loin pain are not specific to stones. Consider vascular, malignant and glomerular alternatives when the story is atypical.

AMT 60-SECOND RECALL
- Calcium oxalate crystals are classically envelope or dumbbell shaped.
- Uric-acid stones favour acidic urine and can be radiolucent on plain film.
- Struvite stones link to urease-positive infection and may become staghorn calculi.
- Cystine crystals are hexagonal and suggest cystinuria.
- Infected obstruction requires urgent urological drainage.
- Analyse recurrent/high-risk stones and target prevention to composition and urine chemistry.

9.1 Autosomal dominant polycystic kidney disease (ADPKD)

ADPKD is an inherited systemic cystic disorder, commonly associated with PKD1 or PKD2 variation; family history may be absent because of new variants or unrecognised disease. Assess affected relatives, blood pressure, eGFR slope, albuminuria, haematuria, stones, cyst infection and liver cyst burden. Ultrasound is first-line in many adults with a known family history; age-dependent cyst-count criteria and a negative scan in a younger adult require careful interpretation. MRI can clarify equivocal ultrasound and quantify height-adjusted total kidney volume (Mayo imaging classification) for progression risk; genetics may aid atypical disease, donor evaluation or reproductive counselling. (KDIGO ADPKD 2025).

Management: optimise blood pressure and cardiovascular risk, manage CKD complications, treat stone/haematuria/infection episodes, and consider tolvaptan for eligible adults at risk of rapid progression after formal risk assessment and shared decision-making. Tolvaptan causes aquaresis and needs clear sick-day rules plus liver-function monitoring under its prescribing programme; counsel on hydration, pregnancy and feasibility. Cyst infection may require prolonged cyst-penetrating antibiotics, specialist imaging and occasionally drainage; haemorrhagic cysts often need supportive care after excluding obstruction, infection and malignancy. Plan kidney replacement and potential transplant as CKD advances.

Family screening: counsel adult first-degree relatives about inheritance, implications and shared decisions on genetic testing or age-adjusted imaging. For intracranial aneurysm, obtain family/personal history of aneurysm, subarachnoid haemorrhage or unexplained sudden death; offer screening in the higher-risk groups described by KDIGO, usually non-contrast time-of-flight MRA. Other people requesting screening can enter shared decision-making. Consider repeat imaging individually in high-risk people with a negative first screen. Do not screen all relatives for an aneurysm solely because they are related to a person with ADPKD. (KDIGO ADPKD 2025, ch. 6).

MRCP RAPID RECALL • A normal young-adult ultrasound does not always exclude familial ADPKD; a Mayo imaging class guides risk only when disease morphology is typical.

9.2 Benign renal lesions and renal cancer

Finding	Imaging and risk assessment	Management principle
Simple cyst	Thin smooth wall, no enhancing septa/solid component; apply Bosniak classification for complex cystic lesions.	Simple cyst usually requires no intervention unless symptomatic; complex cyst needs appropriate radiology/urology pathway.
Angiomyolipoma (AML)	Macroscopic fat often visible on CT; fat-poor AML can mimic malignancy. Ask about tuberous sclerosis and size/growth/bleeding risk.	Observe many asymptomatic lesions; intervene for bleeding or selected high-risk lesions (embolisation or nephron-sparing approach); management depends on symptoms/vascular features, not size alone.
Oncocytoma	Can mimic RCC on imaging; biopsy may help but is not always definitive.	Surveillance or intervention after multidisciplinary risk review; do not label a solid mass benign from appearance alone.

Renal cell carcinoma / solid enhancing mass	Multiphasic contrast CT or MRI to assess enhancement, local stage, renal vein and contralateral kidney; characterise incidental masses by radiology and consider biopsy when it changes management.	For localised T1 lesions, nephron-sparing partial nephrectomy when feasible. Selected small masses: surveillance or ablation with explicit risks. Stage advanced disease and refer uro-oncology for systemic options. Preserve baseline and residual renal function. (EAU RCC 2026).

Presentations include incidental mass, visible haematuria, flank pain or constitutional/paraneoplastic symptoms. A new suspicious renal mass requires referral through the appropriate local cancer pathway; investigate unexplained visible haematuria according to NICE NG12. Consider renal cystic neoplasm and urothelial disease in the differential. Establish performance status, CKD, contralateral renal function and hereditary-cancer clues before treatment. Avoid unnecessary biopsy of an obvious surgical lesion when tissue will not alter management; a benign result from a nondiagnostic sample does not dismiss an enhancing mass.

9.2.1 Renal cell carcinoma: biology, staging and treatment

RCC is a family of tumours: clear-cell (commonly VHL/HIF pathway), papillary, chromophobe and rarer hereditary or aggressive entities. Risks include smoking, obesity, hypertension, acquired cystic kidney disease after prolonged dialysis, and familial syndromes (for example VHL, hereditary papillary RCC, Birt–Hogg–Dubé and hereditary leiomyomatosis/RCC). The classic haematuria–flank pain–mass triad is uncommon; incidental detection is frequent. Look for anaemia or polycythaemia, hypercalcaemia, hepatic dysfunction without metastasis, constitutional symptoms and metastatic pain.

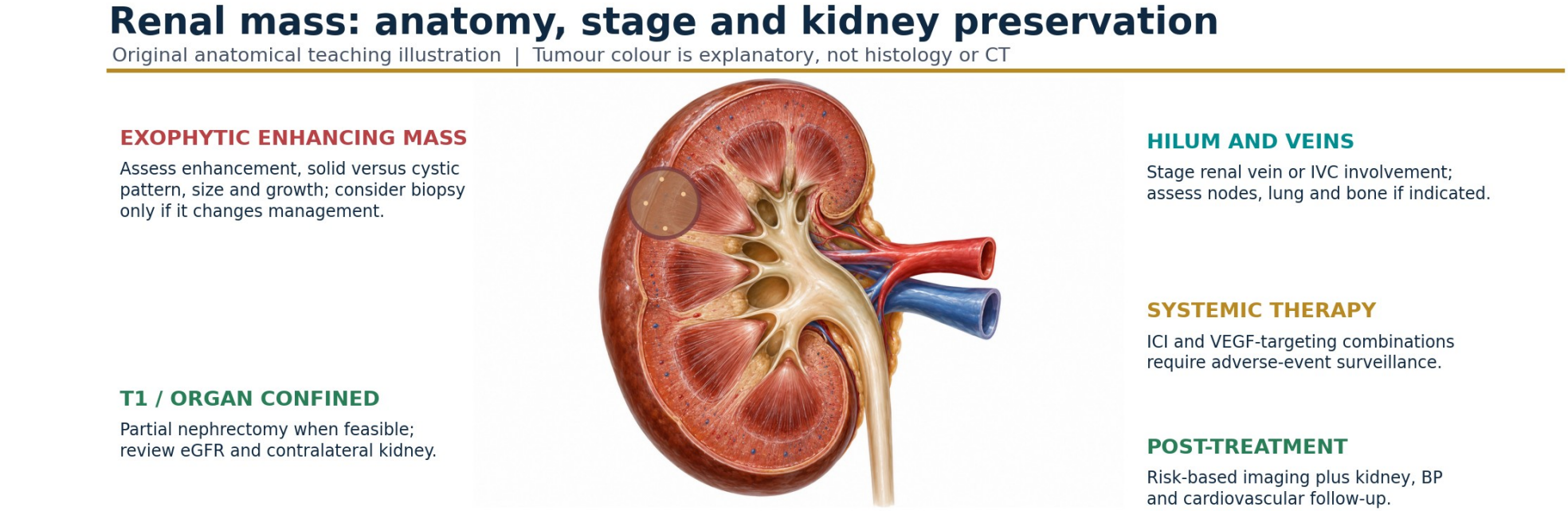


Figure REN-V6-02. Original annotated RCC anatomical illustration; explanatory, not a patient scan.

Characterise a solid enhancing lesion with multiphasic renal-protocol CT or MRI; assess renal vein/IVC thrombus, nodes, contralateral kidney, eGFR and functional reserve. Stage chest appropriately, use selective additional imaging for symptoms or risk, and review Bosniak category for cystic lesions. Percutaneous core biopsy is useful if histology would change surveillance, ablation or systemic therapy; an indeterminate/nondiagnostic sample does not establish benignity. Discuss in a uro-oncology multidisciplinary team. (EAU RCC 2026).

Disease state / modality	Selection and principal benefit	Complications and follow-up
Localised T1: partial nephrectomy	Preferred when feasible to preserve nephrons; open, laparoscopic or robotic route depends on tumour complexity and team expertise.	Bleeding, urinary leak, vascular injury, ischaemia and residual CKD; pathological margin and recurrence risk guide surveillance.
Localised larger/complex: radical nephrectomy	When partial resection is not technically/oncologically appropriate;	CKD progression, hypertension and cardiovascular risk; contralateral

	weigh solitary kidney, baseline CKD and contralateral function.	kidney and long-term eGFR review.
Selected small mass: active surveillance or thermal ablation	Frailty, small indolent mass or competing mortality; biopsy before ablation when feasible. Document thresholds for intervention.	Growth/local progression; ablation risks bleeding, thermal injury and higher local recurrence than resection, so imaging follow-up matters.
High-risk resected clear-cell RCC: adjuvant immunotherapy	Pembrolizumab may be offered after restaging to patients meeting trial-defined high-risk criteria; assess benefit versus toxicity and availability.	Immune-mediated colitis, hepatitis, pneumonitis, endocrinopathies and nephritis; permanent endocrine replacement may be needed.
Metastatic clear-cell RCC: systemic options	IMDC risk guides nivolumab/ipilimumab or ICI–VEGF-TKI (pembrolizumab/axitinib or lenvatinib; nivolumab/cabozantinib). Eligibility, UK access and later-line sequence require MDT review.	ICI colitis, hepatitis, nephritis, endocrinopathy or pneumonitis; VEGF-TKI hypertension, proteinuria, hand–foot syndrome, diarrhoea, hypothyroidism and vascular events; monitor BP, urine and labs.
Non-clear-cell or advanced symptomatic disease	Subtyping and genomic/hereditary context guide specialist trials, targeted combinations and palliative radiotherapy/supportive care.	Variable efficacy and toxicity; coordinate symptom control and renal dosing, especially in CKD.
AMT MEMORY AID Mass → enhancement → stage → kidney reserve → histology when useful → MDT modality → toxicity and surveillance.		
MRCP TRAP Immune checkpoint inhibitor nephritis is not equivalent to calcineurin inhibitor nephrotoxicity; assess creatinine rise with the drug timeline, urinalysis and oncology/nephrology advice.		
AMT 60-SECOND RECALL An enhancing mass needs characterisation and stage. Partial nephrectomy preserves renal function when feasible. Surveillance/ablation suit selected small lesions. Adjuvant pembrolizumab is risk selected; metastatic therapy is histology and IMDC based. Monitor renal and immune adverse effects.		

9.3 Urinary tract infections: cystitis, pyelonephritis and complicated infection

9.3.1 Microbiology, pathogenesis and susceptibility

Most community episodes arise by ascent of enteric flora: uropathogenic Escherichia coli predominates; Staphylococcus saprophyticus is a recognised cause in younger women. Klebsiella, Proteus mirabilis (urease, alkaline urine and struvite stones), Enterococcus and Pseudomonas gain prominence with instrumentation, healthcare exposure, obstruction or prior antibiotics. ESBL-producing Enterobacterales and other resistant organisms demand previous-culture review. Candida in urine often reflects colonisation but may require action in selected high-risk or symptomatic patients. Haematogenous Staphylococcus aureus in urine can signal invasive infection; assess context and blood cultures rather than automatically treating it as simple cystitis. (EAU Urological Infections 2026).

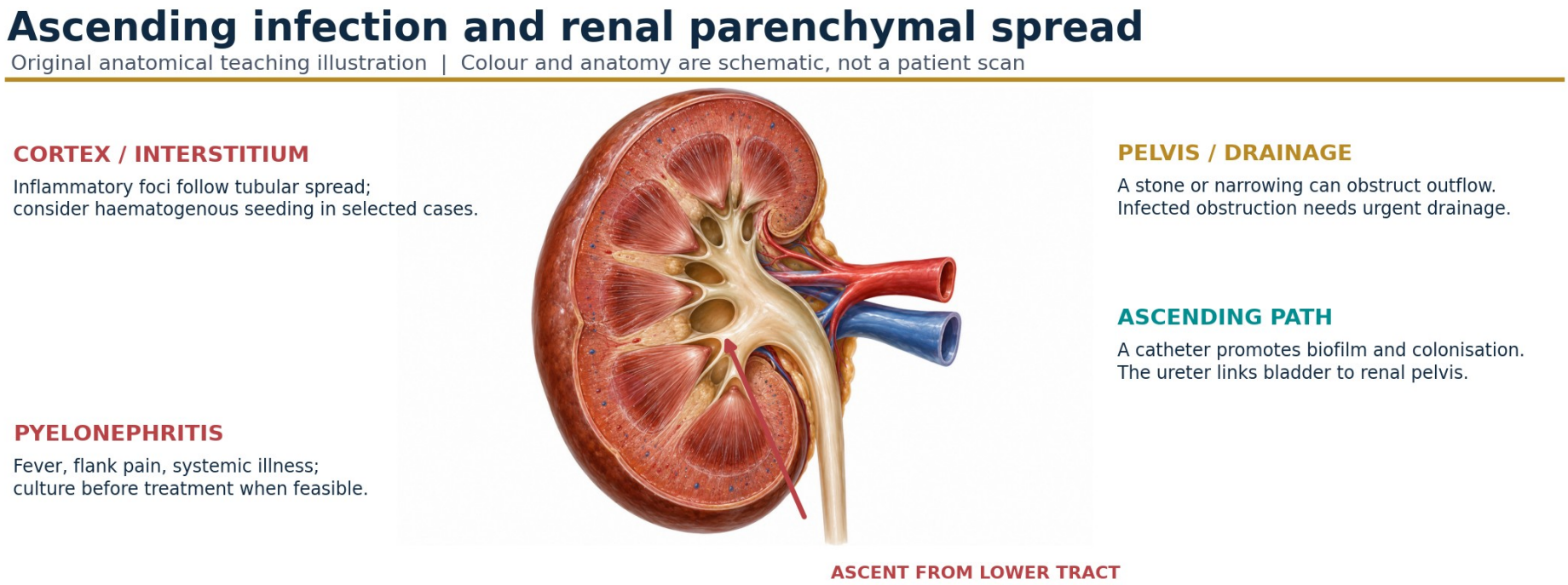


Figure REN-V6-01. Original kidney and urinary tract illustration of ascent, cortical infection and obstructed drainage.

Predisposition	Clinical implication / targeted assessment
Female anatomy, sex, pregnancy, menopause, recurrent episodes	Altered ascent and host protection; pregnancy changes culture and antibiotic pathway; consider vaginal atrophy and prevention choices.
Urinary stasis: retention, BPH, prolapse, neurogenic bladder, obstruction, stones	Measure residual if suspected; image obstruction and check for infection plus impaired drainage.
Diabetes, CKD, transplant or immunosuppression	Higher severe infection or atypical presentation risk; glucose, renal function, graft assessment and interacting drugs matter.
Catheter, stent, nephrostomy, recent procedure; prior antibiotics/resistant isolates	Biofilm/colonisation, source control and culture-directed therapy; avoid treating asymptomatic bacteriuria by default.

9.3.2 Clinical syndromes and pyelonephritis variants

Pattern	Presentation, investigation and complication
Localised cystitis / recurrent UTI	Dysuria, urgency, frequency, suprapubic pain without systemic signs. Recurrent episodes need documented cultures, trigger review and assessment for retention or structural risk when indicated; separate new infection from relapse.
Acute uncomplicated pyelonephritis	Fever, flank pain/tenderness, nausea or rigors; urine culture and susceptibility, blood tests and blood cultures when severe. A negative nitrite test does not exclude infection.
Obstructive pyelonephritis / pyonephrosis	Fever or sepsis with hydronephrosis, stone or reduced drainage; urgent ultrasound and usually cross-sectional imaging; immediate antibiotics plus decompression via stent/nephrostomy.
Emphysematous pyelonephritis / pyelitis	Gas in parenchyma/collecting system, often diabetes or obstruction. CT defines extent; urgent antibiotics, haemodynamic support and drainage; selected uncontrolled necrosis may require nephrectomy.
Renal or perinephric abscess / focal bacterial nephritis	Persistent fever, local pain or failure to improve; CT/MRI delineates collection or focal inflammatory mass; targeted antibiotics and drainage when indicated.
Xanthogranulomatous pyelonephritis	Chronic destructive inflammation, often obstruction and staghorn calculus; CT may show enlarged poorly functioning kidney; manage infection and plan surgery with urology.
Papillary necrosis or fungal ball	Consider diabetes, sickle cell disease, analgesic injury or immunosuppression; imaging and culture guide drainage and pathogen-specific therapy.

Investigation sequence: observations, sepsis screen and pregnancy status first; obtain a correctly collected urine specimen for culture in pyelonephritis, men with suspected UTI, pregnancy, catheter-associated symptomatic infection, recurrence, atypical infection or treatment failure. Check FBC, CRP, renal profile and blood cultures for systemic illness; measure lactate when shocked. Ultrasound detects hydronephrosis; CT with appropriate contrast strategy delineates abscess, gas and complicated obstruction, while MRI is an alternative in selected pregnancy or contrast-limited cases. Review imaging urgently for severe disease, suspected obstruction or failure to improve after 48–72 hours of effective therapy. (EAU 2026; NICE NG111).

Complications include bacteraemia, urosepsis/shock, AKI, abscess, pyonephrosis, papillary necrosis, recurrent infection, scarring and, in pregnancy, maternal or fetal complications. Source control is essential when there is an infected obstructed system, abscess requiring drainage or infected device. In adults with recurrent cystitis discuss behavioural measures and, when eligible, vaginal oestrogen or prevention strategies following the current NICE recurrent-UTI pathway; avoid reflex long courses for every recurrence.

9.3.3 Population-specific assessment and prevention

Pregnancy: document culture and susceptibility for suspected infection, assess maternal observations and fetal context, avoid medicines unsuitable for gestation, and involve obstetrics promptly for fever, flank pain or sepsis. Acute pyelonephritis in pregnancy warrants a lower threshold for admission, IV treatment and imaging that accounts for fetal safety. Asymptomatic bacteriuria in pregnancy is an important exception to the usual no-treatment rule. In men consider prostatic involvement, retention and obstruction rather than assuming a simple short-course cystitis pathway. A kidney transplant recipient with fever or graft pain needs transplant input, assessment for rejection and graft obstruction, and an antibiotic interaction check, especially with tacrolimus/ciclosporin.

For recurrent lower UTI, first confirm episodes with cultures when appropriate and separate relapse (same organism soon after treatment, suggesting a persistent focus) from reinfection. Review prior susceptibilities, post-void residual, stones, atrophic vaginitis, sexual and contraceptive triggers, fluid intake and catheter need. Consider postmenopausal vaginal oestrogen when eligible, then NICE-directed options such as single-dose prophylaxis after a trigger or methenamine hippurate in suitable non-pregnant adults after other measures, with specialist advice for complex upper-tract disease. Continuous antibiotic prophylaxis requires shared decisions about resistance, adverse effects and review timing. Persistent haematuria after resolution needs separate evaluation; never attribute all red urine to recurrent infection. (NICE NG112; EAU Urological Infections 2026).

Prescribing examples for adult teaching: nitrofurantoin or trimethoprim may suit lower UTI in eligible adults, subject to kidney function, pregnancy, resistance and local policy; fosfomycin can be an option in defined lower-UTI pathways. Upper UTI requires an agent with renal tissue activity, such as a guideline-suitable oral cephalosporin in stable cases or intravenous therapy when severe or unable to take oral treatment; de-escalate after susceptibility and step down IV when stable. Restrict fluoroquinolones according to current MHRA safety advice. Check present NICE NG109/NG111/NG113 tables, local antibiogram and BNF for dose, interval and course length; this chapter does not prescribe a regimen for an individual patient.

9.3.4 Catheter-associated UTI: bedside management

A catheterised patient often has bacteriuria and pyuria without symptomatic infection. Cloudy or malodorous urine, a positive dipstick or delirium in isolation should prompt assessment for other causes rather than automatic antibiotics. New fever, rigors, flank/suprapubic pain, haemodynamic instability or other compatible symptoms require evaluation, including alternative infection sources. Obtain a fresh urine specimen aseptically from the sampling port, never the bag; if replacing the catheter, culture urine from the new catheter when possible before antibiotics. (NICE NG113; EAU 2026).

Remove the catheter if it is no longer needed. If it has been in place for more than 7 days, NICE recommends removal or change as soon as possible without delaying antibiotics; ensure no difficulty or contraindication to replacement. Treat symptomatic infection according to localised versus systemic severity, prior cultures, local resistance, allergy, pregnancy, kidney function and interactions. Start empiric therapy promptly for systemic illness, then review susceptibility and narrow therapy. Monitor response within 48 hours and assess retention or obstruction if still unwell. Do not give routine prophylactic antibiotics or treat catheter-associated asymptomatic bacteriuria except specific preprocedure pathways. Prevent recurrence by limiting catheter duration, maintaining a closed system and avoiding unnecessary manipulation. (NICE NG113; EAU 2026).

AMT MEMORY AID Symptoms → culture correctly → remove or replace device → choose tissue-appropriate antibiotic → reassess → drain obstruction if present.
MRCP RAPID RECALL Nitrofurantoin treats eligible lower-tract infection, not renal parenchymal infection. Catheter biofilm often colonises; treat the patient, not the bag culture.
AMT 60-SECOND RECALL E. coli dominates community UTI; Proteus suggests stones; previous isolates shape resistance risk. Localised versus systemic symptoms set urgency. Culture pyelonephritis. CT delineates gas and abscess. Infected obstruction needs drainage. Replace/remove a symptomatic long-standing catheter and avoid treating colonisation.

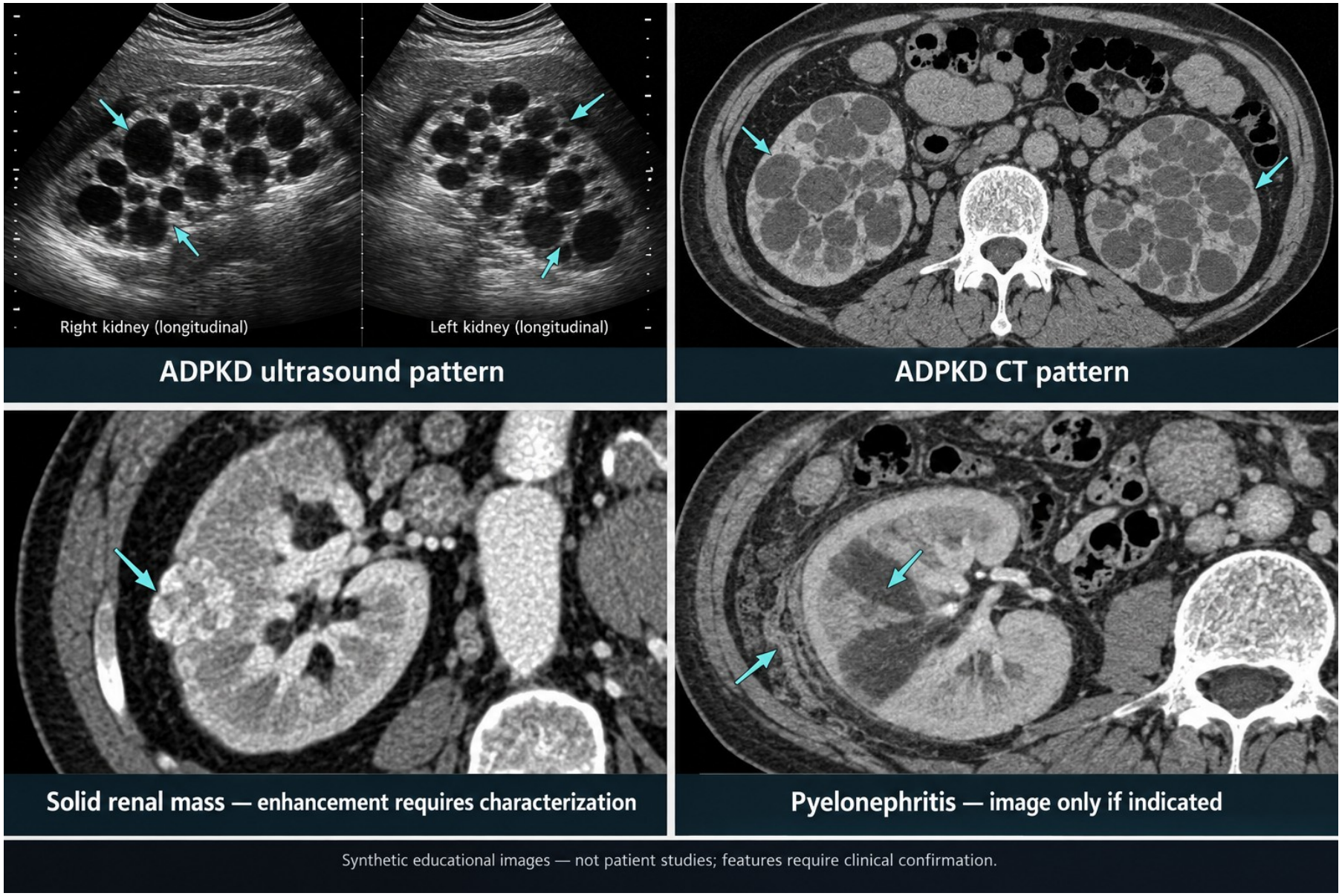


Figure REN-M18. Synthetic educational renal ultrasound and CT impressions of ADPKD, a solid renal lesion and pyelonephritis. These are not patient scans; investigate and verify imaging findings in context.

10. Renal hypertension: mechanisms, diagnosis and treatment

PART 1 CORE • RENAL PERFUSION → SODIUM BALANCE → RAAS → VASCULAR TONE

Kidney disease can cause hypertension through sodium and volume retention, RAAS activation, sympathetic signalling and vascular stiffness; hypertension can in turn accelerate albuminuria, glomerulosclerosis and cardiovascular disease. Establish the BP phenotype with standardised clinic measurements and home or ambulatory confirmation when suitable, and evaluate orthostasis, adherence, salt intake, volume state, medicines (NSAIDs, steroids, calcineurin inhibitors, erythropoiesis-stimulating agents), sleep apnoea and endocrine causes. Assess eGFR trajectory, urine ACR, haematuria/sediment, electrolytes and ECG; ultrasound is indicated for structural suspicion rather than indiscriminate testing. (NICE NG136; NG203; KDIGO CKD 2024).

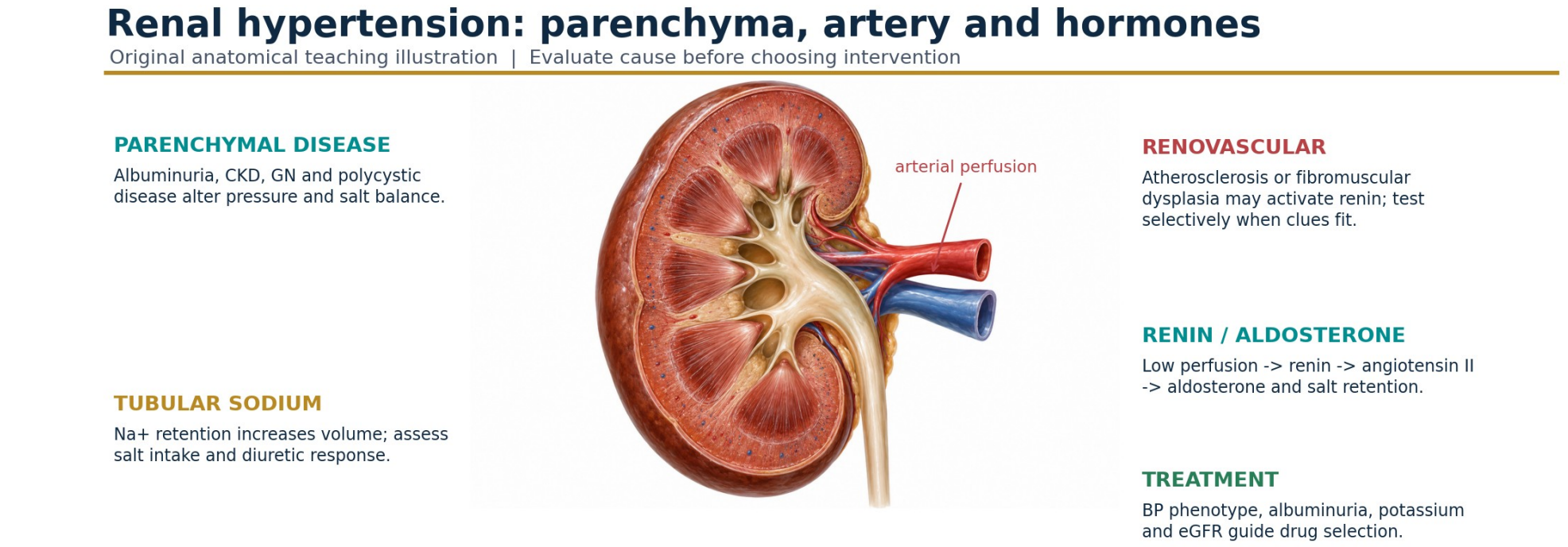


Figure REN-V6-03. Original kidney and renal artery anatomy anchoring hypertensive mechanisms.

Renal phenotype / clue	Tests and interpretive caution	Treatment direction
Albuminuric CKD / diabetes	Repeated ACR and eGFR, potassium, medication review. A fall in GFR after RAAS blockade requires context and monitoring.	ACEi or ARB when guideline indicated and tolerated; monitor renal function/K+, titrate BP therapy and consider SGLT2 inhibitor under CKD criteria. Avoid dual ACEi+ARB.
Volume-dependent hypertension / advanced CKD	Examine oedema, weight and salt balance; plasma renin alone does not diagnose the cause.	Moderate salt intake, appropriate diuretic (loop as GFR falls or volume expands), monitor sodium/K+, avoid overdiuresis.
Renovascular disease: abrupt/worsening resistant HTN, flash pulmonary oedema or asymmetric kidneys	Duplex Doppler, CT or MR angiography selectively; consider contrast risk and image quality. FMD in a younger patient; atherosclerosis in older vascular disease.	Optimal medical therapy and vascular risk control for most atherosclerotic disease; discuss revascularisation in selected recurrent flash oedema, refractory BP or threatened renal function; FMD often responds to angioplasty.
Resistant hypertension, low K+ or disproportionate aldosterone	Exclude pseudoresistance with ambulatory/home BP, adherence and correct cuff; consider aldosterone–renin ratio with medication effects in mind.	Optimise combination, volume and salt; consider MRA if K+/eGFR permit with close monitoring, or alternatives and specialist referral.
Severe BP with acute organ injury	Neurologic signs, retinal changes, pulmonary oedema, AKI, TMA: urgent ECG, bloods and targeted imaging; distinguish emergency from severe asymptomatic BP.	Hospital treatment tailored to organ injury and careful controlled reduction; do not abruptly normalise BP without specialist plan.

Targets depend on albuminuria, diabetes, frailty, treatment tolerance and how BP is measured. For adults with CKD, NICE NG203 uses clinic targets below 140/90 mmHg when ACR is below 70 mg/mmol and below 130/80 mmHg when ACR is at least 70 mg/mmol; apply current guideline details and individualise. In a patient receiving RAAS blockade check baseline and follow-up creatinine/eGFR and potassium after initiation or dose increase, and reassess a marked creatinine rise, severe hyperkalaemia, dehydration, NSAID exposure or bilateral renal artery stenosis. Avoid withdrawing kidney-protective therapy reflexively for a small expected haemodynamic change; assess symptoms and trends. (NICE NG203; KDIGO CKD 2024).

AMT MEMORY AID Measure → identify kidney phenotype → check albumin and volume → choose renoprotective therapy → monitor K+ and GFR → seek secondary cause when pattern fits.
MRCP RAPID RECALL Flash pulmonary oedema plus worsening kidney function during RAAS blockade can raise suspicion for bilateral renovascular disease, but the diagnosis requires clinical and imaging assessment.
AMT 60-SECOND RECALL Confirm BP outside clinic when appropriate. CKD risk is GFR plus ACR. Salt/volume, RAAS and vascular stiffness link kidney disease to BP. ACEi/ARB supports albuminuric CKD with K+/eGFR checks. Revascularisation is selective. Resistant BP needs adherence, volume and secondary-cause review. Hypertensive emergency requires organ-injury assessment.

11. Kidney replacement therapy and transplantation

PART 2 APPLICATION • PRESENTATION → DIFFERENTIAL → INVESTIGATION → INTERPRETATION → MANAGEMENT → COMPLICATION → NEXT STEP

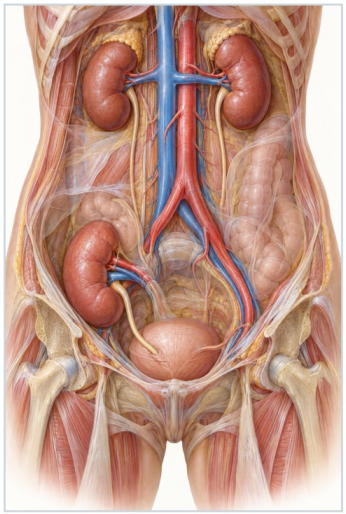
- RRT modalities include intermittent haemodialysis, continuous therapies and peritoneal dialysis; modality depends on haemodynamics, urgency, access, goals and local expertise. (NICE, 2018).
- Long-term care includes vascular/peritoneal access planning, transplantation assessment, conservative kidney management and shared decision-making.

Transplant AKI on realistic pelvic anatomy

Native kidneys, iliac vessels, graft anastomoses, ureter and bladder remain visible

PERFUSION / VASCULAR
Dehydration, sepsis, thrombosis or arterial/venous stenosis

PARENCHYMA
Rejection, recurrent disease, BK/CMV or pyelonephritis



PLUMBING
Ureteric obstruction, lymphocele or catheter problem

PHARMACOLOGY
Calcineurin toxicity, interaction, missed doses and nephrotoxins

Compare baseline -> assess volume/sepsis -> urine/culture -> Doppler -> levels -> biopsy if needed

AI-assisted original medical atlas artwork; synthetic educational image; human clinical review required

PART 2 APPLICATION — transplant AKI: protect the graft while defining cause

- Check haemodynamics, fluid balance, sepsis, drug history, trough immunosuppressant levels, urine protein/sediment and obstruction/vascular flow with graft ultrasound as indicated.
- Time course informs probability but never excludes a diagnosis: acute tubular injury, rejection, CNI toxicity, BK/CMV disease, recurrent primary disease and obstruction overlap.
- Do not empirically stop baseline immunosuppression without transplant-team input unless toxicity or life-threatening infection mandates an urgent coordinated change.
- Biopsy is often decisive after structural/vascular causes and readily reversible factors are addressed; dose and interaction review is integral, especially with azoles, macrolides and CYP3A modifiers.

Figure REN-04. Original AMT transplant-AKI diagnostic pathway.

11.1 Dialysis modalities: indications, selection and complications

Acute kidney replacement is considered for complications refractory to appropriate medical treatment: life-threatening hyperkalaemia, severe metabolic acidosis, pulmonary oedema/volume overload, uraemic complications (pericarditis, encephalopathy, bleeding) and selected dialysable toxins. No single creatinine or urea threshold determines timing in isolation. For advanced CKD, shared planning considers symptom burden, biochemical and fluid control, trajectory, patient goals, access and transplant suitability; NICE NG107 advises considering dialysis initiation for uraemic symptoms, biochemical measures or uncontrollable fluid overload, or around eGFR 5–7 mL/min/1.73m² if asymptomatic. Do not apply the elective threshold to an emergency. (NICE NG107).

Modality / usual setting	Mechanism, access and practical fit	Principal complications and monitoring
Intermittent haemodialysis (IHD)	Extracorporeal diffusion/ultrafiltration; usually 3 sessions weekly for maintenance, with prescription individualized. AV fistula/graft preferred for durable access when feasible; tunnelled catheter when necessary.	Intradialytic hypotension/cramps, arrhythmia, access stenosis/thrombosis or infection, blood loss and inadequate clearance; check dry weight, Kt/V or local adequacy marker, access and blood cultures when febrile.
Continuous kidney replacement therapy (CKRT)	Continuous venovenous haemofiltration, haemodialysis or haemodiafiltration in critical care; often favoured with haemodynamic instability or cerebral oedema risk. Uses large-bore vascular catheter and anticoagulation protocol.	Filter clotting, bleeding or citrate-associated metabolic disturbances, hypothermia, electrolyte/phosphate depletion, drug underdosing; track effluent dose delivered and medication clearance.
Prolonged intermittent dialysis / haemodiafiltration	Sustained low-efficiency dialysis may bridge IHD and CKRT in critical care; haemodiafiltration combines diffusion and convection with dedicated equipment.	Monitor haemodynamics, solute and volume targets, anticoagulation, circuit clotting and drug clearance; protocols vary by unit.
Peritoneal dialysis (CAPD/APD)	Peritoneum is the exchange membrane; manual daytime exchanges or cycler overnight. Home modality needs training, catheter function and suitability assessment.	Peritonitis, exit-site/tunnel infection, leak, hernia, catheter dysfunction, ultrafiltration failure and glucose burden; inspect effluent/exit site and assess volume/nutrition/residual kidney function.
Conservative kidney management	Symptom control, CKD complication management, advance care planning and psychosocial support without dialysis; valid positive choice.	Monitor symptom burden, fluid and potassium issues and goals; revisit preferences if circumstances change.

11.1.1 Modality choice, prescription and surveillance

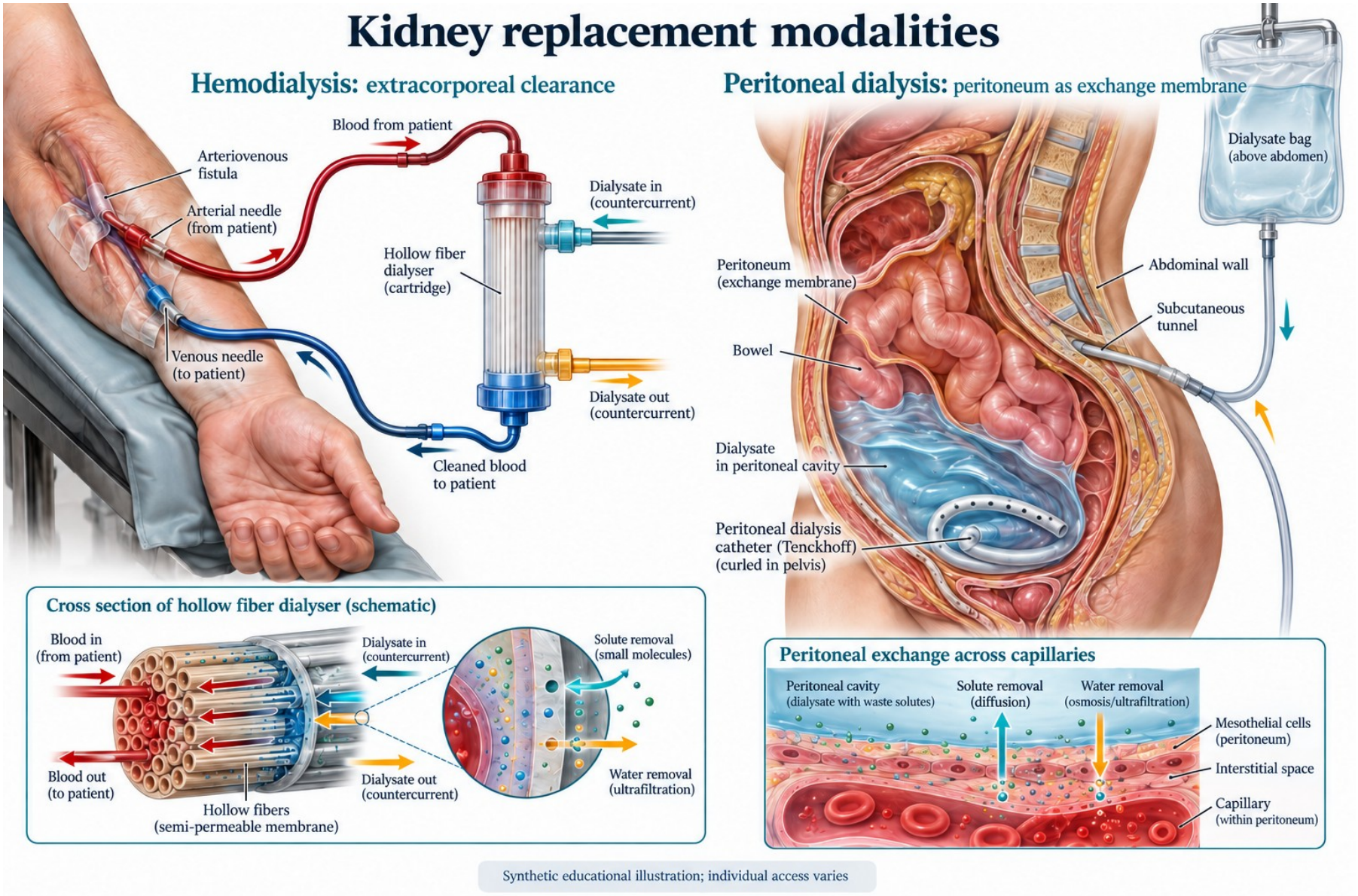
Intermittent haemodialysis (IHD) offers rapid solute and potassium clearance through a vascular circuit; assess anticoagulation, vascular access, haemodynamic tolerance and dialysis disequilibrium risk when initiating in severe uraemia. Continuous kidney replacement therapy (CKRT) delivers slower clearance and fluid removal in haemodynamically unstable critical illness; it depends on circuit patency, prescribed versus delivered dose, fluid balance, anticoagulation and close electrolyte/phosphate monitoring. Prolonged intermittent therapies combine aspects of both. Peritoneal dialysis (PD) uses the peritoneum as an exchange membrane; it offers home-based flexibility but requires catheter competence, membrane adequacy and attention to glucose exposure, hernia, leak and infectious risk. (NICE NG107; ISPD 2022).

Access is a treatment in its own right. For HD, plan an arteriovenous fistula or appropriate graft and preserve future access sites; a central venous dialysis catheter is useful when urgent but raises bloodstream infection, thrombosis and stenosis risk. For PD, insertion technique, exit-site hygiene and early assessment of cloudy effluent matter. Suspected PD peritonitis requires prompt dialysate cell count/differential, Gram stain and culture using the correct specimen process, and protocol-directed intraperitoneal antibiotics where suitable; do not wait for culture before treating a convincing presentation. Rule out surgical abdomen or refractory/fungal infection, where catheter removal may be required. (UKKA Vascular Access 2025; ISPD Peritonitis 2022).

Long-term HD complications include intradialytic hypotension, cramps, access infection or stenosis, anaemia, mineral-bone disorder, pruritus and accelerated cardiovascular disease. PD complications include bacterial/fungal peritonitis, exit-site/tunnel infection, mechanical catheter failure, hernia, ultrafiltration failure, glucose burden and, rarely, encapsulating peritoneal sclerosis. Monitor symptoms, nutrition, haemoglobin/iron, bone-mineral parameters, BP, volume and adequacy; adjust modality to residual kidney function, access, capacity and patient goals. Shared planning includes transplant assessment and an active conservative kidney management option; modality changes should follow changing physiology, access and preference.

MRCP TRAP A dialysed patient with abdominal pain and cloudy PD effluent needs a PD peritonitis pathway; an HD catheter with fever needs bloodstream infection assessment. The access type changes the work-up.

AMT 60-SECOND RECALL IHD is faster but less well tolerated in some unstable patients; CKRT supports gradual clearance and volume control; PD is home based with peritonitis risk. Assess access, delivered clearance, residual renal function and complications before changing prescription.



Synthetic educational illustration; individual access varies

PART 2 APPLICATION — dialysis: prescription plus access plus complication

- Choose modality through shared decision-making: physiology, lifestyle, vascular/peritoneal access, cognition, support, infection risk and residual kidney function all matter.
- Haemodialysis emergencies include access infection/thrombosis, severe hyperkalaemia, pulmonary oedema, haemolysis, air embolism and disequilibrium. Stop and assess immediately when circuit or neurological danger is suspected.
- Peritoneal dialysis peritonitis: abdominal pain/cloudy effluent demands prompt effluent cell count, differential and culture, followed by protocol-directed intraperitoneal antibiotics where

appropriate.

- Interpret adequacy with the patient—not a single clearance number: symptoms, volume/BP, nutrition, electrolytes, anaemia, bone-mineral indices and access performance complete the assessment.

Figure REN-M19. Original synthetic educational haemodialysis access and dialyser, and peritoneal dialysis catheter and exchange membrane; device depiction is illustrative.

Access planning starts before maintenance dialysis when feasible; preserve suitable arm veins and choose fistula, graft or catheter by prognosis, anatomy and patient choice. Suspected AV-access infection or impaired flow needs prompt assessment. A haemodialysis catheter carries bloodstream-infection and central venous stenosis risk. Peritoneal dialysis cloudy effluent or abdominal pain requires immediate effluent cell count/differential and culture, with empiric intraperitoneal antibiotics per local/ISPD protocol after specimen collection; do not wait for culture. Peritonitis is diagnosed using symptoms/cloudy effluent, effluent leukocytes with neutrophil predominance, and/or positive culture in combination, not on appearance alone. (UKKA vascular access 2025; ISPD peritonitis 2022).

Dialysis choice is revisited when ultrafiltration fails, access complications recur, solute clearance is inadequate or preferences change. In AKI, compare daily fluid balance, electrolyte/acid-base response and haemodynamic tolerance; reassess renal recovery and the continuing indication each day. In end-stage disease, assess transplant candidacy and conservative pathways alongside modality options. Drug dosing varies between intermittent and continuous therapies, and should account for residual kidney function and local pharmacology advice.

MRCP RAPID RECALL • Haemodialysis clears through an extracorporeal dialyser; peritoneal dialysis uses the peritoneal membrane; CKRT is often suited to unstable ICU patients. Urgent dialysis is driven by refractory complications, not creatinine alone.

AMT 60-SECOND RECALL • Acute indications are refractory K+, acidosis, oedema, uraemic organ injury or toxins; chronic timing is symptom/biochemistry/volume plus preference; plan access early; IHD has rapid extracorporeal exchange; CKRT requires delivered-dose and electrolyte checks; PD uses living peritoneum and can be home-based; cloudy PD effluent is an urgent culture-and-treat issue; monitor access infection, volume and clearance; adapt medication doses to modality; revisit conservative care and transplant.

11.2 Transplant pharmacology

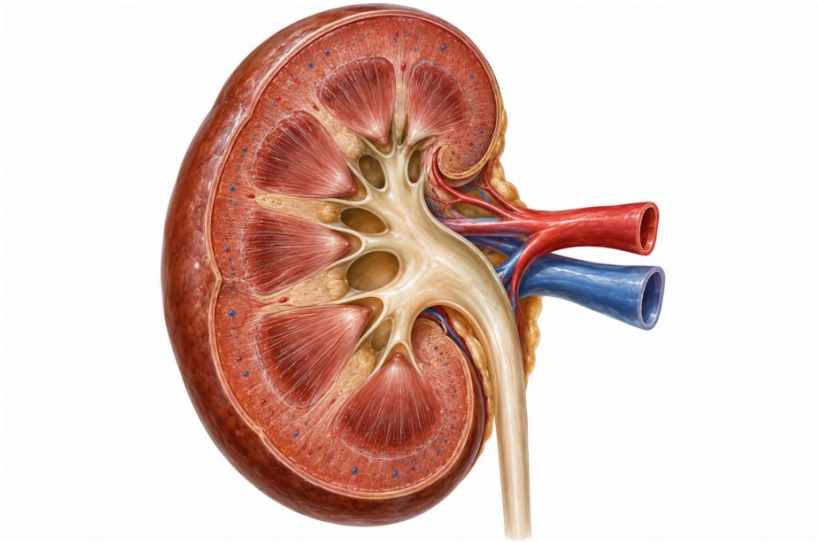
11.2.1 Complications by immunosuppressant and time after transplantation

Immunosuppression balances rejection prevention against infection, malignancy and drug toxicity. Early infection often reflects surgery, devices and hospital exposure; subsequent opportunistic infection relates to net immunosuppression and prophylaxis; late risks include community infection, chronic viral complications and cancer. Investigate fever or graft dysfunction even when symptoms are muted. Compare baseline creatinine, urinalysis, cultures, drug troughs (draw at the specified time), imaging and adherence; coordinate immunosuppression changes with the transplant centre. (KDIGO Kidney Transplant Recipient guideline; NICE TA481).

Class / agent	Mechanism-linked complications	Important interactions and surveillance
Calcineurin inhibitors: tacrolimus, ciclosporin	Acute/chronic nephrotoxicity, hypertension, hyperkalaemia, hypomagnesaemia, neurotoxicity and post-transplant diabetes (especially tacrolimus); ciclosporin can cause gingival hyperplasia/hirsutism.	CYP3A/P-gp inhibitors (for example clarithromycin, azoles, some calcium-channel blockers, grapefruit) raise levels; inducers (rifampicin, some anticonvulsants) lower levels. Trough, eGFR, K+, Mg2+, BP, glucose.
Mycophenolate; azathioprine	Mycophenolate: diarrhoea, leukopenia and teratogenicity; azathioprine: marrow suppression, hepatotoxicity and pancreatitis; both increase infection/cancer risk with overall exposure.	Allopurinol/febuxostat can cause profound azathioprine toxicity; specialist regimen required. Monitor CBC/LFT and infection; pregnancy counselling and contraception with mycophenolate.
mTOR inhibitors: sirolimus, everolimus	Poor wound healing, stomatitis, dyslipidaemia, oedema, cytopenias, proteinuria, pneumonitis and thrombotic microangiopathy; may have role in selected cancer-risk strategies.	CYP3A interactions; measure trough when indicated, urine protein, lipids, CBC and respiratory symptoms.
Steroids and lymphocyte-depleting induction	Steroids: hyperglycaemia, osteoporosis, infection, mood effects, cataract and avascular necrosis. Induction/depletion: cytopenias, infusion reactions and opportunistic infections.	Glucose, bone health, BP, vaccination and tailored infection prophylaxis; monitor CBC and viral risk according to protocol.

Transplant immunosuppression: target tissue and tumour surveillance

Original anatomical teaching illustration | Skin and lymphoid patterns are representative, not diagnostic histology



ALLOGRAFT

CNI arteriolopathy / nephrotoxicity;
BK nephropathy; rejection and recurrence.

DRUG TOXICITY

Cytopenias, infection, diabetes and metabolic or vascular effects.

SKIN / MUCOSA

Cutaneous squamous cell carcinoma; basal cell carcinoma, melanoma and HPV-related malignancy risk.

LYMPHOID / VIRAL

PTLD often EBV-associated; Kaposi sarcoma (HHV-8) in at-risk hosts.

SCREEN AND INVESTIGATE

Sun protection, dermatology review, age-appropriate screening and biopsy.

Figure REN-V6-04. Original anatomical illustration linking transplant drug effects with skin and lymphoid cancer surveillance.

11.2.2 Malignancy associated with long-term immunosuppression

Name the cancers rather than grouping them as “malignancy”: cutaneous squamous cell carcinoma (particularly increased), basal cell carcinoma and melanoma; post-transplant lymphoproliferative disorder (PTLD), often EBV-associated and ranging from polyclonal proliferation to lymphoma; Kaposi sarcoma associated with HHV-8; HPV-associated cervical, anogenital and oropharyngeal cancers; native-kidney RCC, especially with acquired cystic disease after dialysis; and liver cancer in relevant chronic viral hepatitis. Relative risk varies by region, virus exposure, age, transplant duration, prior cancer and immunosuppressive burden. A graft recipient can also develop ordinary population cancers such as colorectal, breast or lung cancer; do not attribute every lesion to immunosuppression.

Prevention and response: sun avoidance/protection and skin surveillance; age- and risk-appropriate cervical/HPV, colorectal and other screening; vaccination when appropriate; investigate constitutional symptoms, adenopathy, unexplained cytopenias, haematuria or suspicious skin lesions promptly and obtain tissue diagnosis. PTLD work-up commonly includes imaging, biopsy and EBV assessment, but blood EBV alone does not establish or exclude disease. Manage confirmed malignancy jointly with oncology, dermatology and transplant teams; any reduction or switch of immunosuppression balances cancer response against rejection. Do not stop a calcineurin inhibitor casually. (KDIGO transplant recipient guideline).

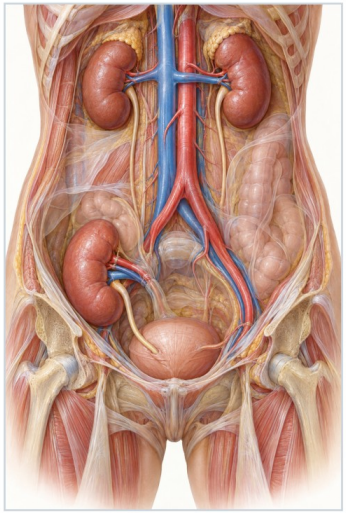
MRCP TRAP A raised tacrolimus trough with AKI after a macrolide or azole suggests exposure toxicity; persistent creatinine rise may still reflect rejection, obstruction, infection or recurrent disease.
AMT 60-SECOND RECALL CNI: kidney, potassium, magnesium, BP and glucose. Antimetabolites: marrow/GI; azathioprine–xanthine oxidase interaction. mTOR: wounds, protein and lipids. Steroid: metabolic/bone. Cancer: skin SCC, PTLD, Kaposi, HPV-related and native-kidney RCC. Screen and biopsy; preserve graft protection through multidisciplinary changes.

Immunosuppression adverse effects on patient and graft

A realistic transplant field replaces disconnected drug icons

EXPOSURE
CYP3A/P-gp interactions, diarrhoea, adherence and sampling time matter

ORGAN TOXICITY
Kidney, marrow, liver, metabolic, lung, skin and bone effects



INFECTION / MALIGNANCY
Net immunosuppression evolves with regimen, dose and time

REJECTION RISK
Underdosing, enzyme induction or missed doses may threaten the graft

Macrolides/azoles may raise CNI exposure; enzyme inducers may lower it

AI-assisted original medical atlas artwork; synthetic educational image; human clinical review required

Figure REN-M16. Original AMT immunosuppressant toxicity and interaction map.

12. Complex Part 2 application

PART 2 APPLICATION • PRESENTATION → DIFFERENTIAL → INVESTIGATION → INTERPRETATION → MANAGEMENT → COMPLICATION → NEXT STEP

12.1 Myocardial infarction / ACS in CKD

- Do not dismiss chest pain because troponin is chronically elevated. Diagnose acute MI from the clinical syndrome, ECG and a dynamic biomarker pattern where appropriate.
- Renal impairment increases both ischaemic and bleeding risk. Antithrombotic choice/dose must reflect kidney function; NICE advises considering monitored unfractionated heparin as an alternative to fondaparinux in significant renal impairment. (NICE, 2020).
- Do not delay clinically essential emergency contrast imaging or PCI solely because of AKI risk. Mitigate risk, review nephrotoxins and monitor renal function.
- Secondary prevention remains important, with renal-function/electrolyte monitoring around RAAS blockade and other renally relevant drugs.

MRCP TRAP: CKD modifies ACS management; it does not make evidence-based ACS care optional. The correct answer is usually "treat the ACS while adapting drug choice, dose, contrast strategy and monitoring," not therapeutic nihilism.

12.2 DKA in advanced renal failure / dialysis

JBDS guidance specifically recognises DKA in end-stage renal failure as a special situation with limited evidence. Diagnostic criteria remain broadly similar, but physiology changes management. (Joint British Diabetes Societies for Inpatient Care, 2023).

Issue	Preserved renal function	Advanced renal failure/dialysis
Fluid deficit	Often substantial due to osmotic diuresis	May be small/absent; volume overload may coexist
Fluids	Protocol-driven replacement with reassessment	Individualise; if hypovolaemic, small aliquots with frequent reassessment

Issue	Preserved renal function	Advanced renal failure/dialysis
Insulin	Fixed-rate IV insulin with glucose added as required	Mainstay; reduced renal clearance increases hypoglycaemia risk and rate may need reduction
Potassium	Total-body depletion common despite initial serum K+	Routine replacement often unnecessary; hyperkalaemia more common
Dialysis	Usually not required solely for DKA	Consider for severe hyperkalaemia, overload, refractory acidosis or usual dialysis indications

RED FLAG: Do not automatically apply a several-litre DKA fluid prescription or routine potassium replacement to an anuric dialysis patient.

12.3 Transplant recipient with AKI or adverse drug effects

CASE APPLICATION: a creatinine rise in a graft recipient requires urgent review of baseline, perfusion, infection, obstruction, vascular integrity, rejection and medication exposure. Use the transplant pathway in section 10.2 for detailed work-up; interpret timing and biopsy need with the transplant team. This case tests prioritisation rather than repeating the general transplant work-up.

- Biopsy: consider when rejection or intrinsic graft pathology remains suspected after reversible causes and structural/vascular problems are assessed.
- Management: correct reversible causes, avoid nephrotoxins, adjust interacting drugs with transplant specialists, and do not stop immunosuppression reflexively.

COMMON MRCP TRAP: Macrolides and azole antifungals can inhibit CYP3A and raise tacrolimus/ciclosporin levels; rifampicin and other inducers can lower levels and increase rejection risk.

AMT MEMORY AID

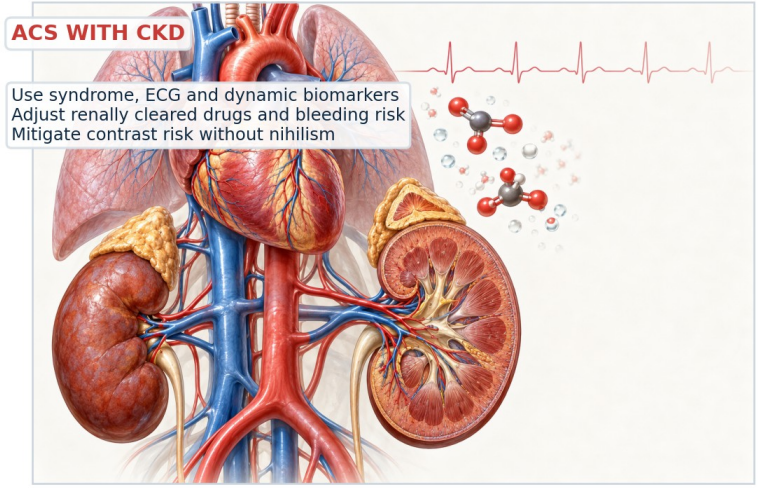
Preserve the emergency diagnosis, then modify four domains for renal failure: volume, potassium, drug clearance and procedure risk.

MRCP RAPID RECALL

In dialysis DKA, insulin remains essential, but routine large-volume fluid and potassium protocols may be dangerous. In CKD ACS, adjust drugs and mitigate risk without reflexively withholding indicated reperfusion or invasive assessment.

Complex renal decisions: ACS and DKA in advanced CKD

Preserve emergency treatment, then modify fluids, potassium, clearance, contrast strategy and monitoring





Renal failure changes treatment delivery and monitoring; it does not remove the emergency

AI-assisted original medical atlas artwork; synthetic educational image; human clinical review required

PART 2 APPLICATION — ACS or DKA in advanced CKD/dialysis

- ACS: interpret troponin dynamically with symptoms and ECG; CKD raises baseline values but does not explain a convincing rise/fall. Balance antithrombotic benefit, bleeding risk, renal dosing and an invasive strategy with cardiology/nephrology.
- DKA: insulin remains essential. Dialysis patients may be volume replete or overloaded; avoid automatic multi-litre fluid protocols and reassess after small boluses only when hypovolaemic.
- Potassium replacement is not routine in kidney failure. Monitor frequently; hyperkalaemia, pulmonary oedema or refractory acidosis may require urgent dialysis.
- A normalised glucose is not resolution: continue treatment until ketosis and acidosis resolve, while preventing hypoglycaemia because insulin clearance is reduced.

Figure REN-M10. Original AMT renal modification map for ACS and DKA.

CLINICAL PEARL
Troponin may be chronically elevated in CKD; the clinical syndrome, ECG and dynamic change remain central.

MRCP TRAP
Do not assume every breathless dialysis patient is fluid overloaded or every acidosis is DKA. Confirm the physiology before applying a modified protocol.

AMT 60-SECOND RECALL
- ACS in CKD: assess ischaemic and bleeding risk together.
- Adjust renally cleared antithrombotic therapy and check interactions.
- Use contrast-risk mitigation without delaying necessary emergency care.
- Dialysis DKA: define volume phenotype before giving fluid.
- Insulin clearance is reduced; monitor glucose and potassium closely.
- Do not add potassium routinely when serum potassium is high or normal.
- Consider dialysis for refractory hyperkalaemia, acidosis or overload.

13. Rapid review: must know

PART 2 APPLICATION • PRESENTATION → DIFFERENTIAL → INVESTIGATION → INTERPRETATION → MANAGEMENT → COMPLICATION → NEXT STEP

- AKI is defined by change from baseline, not by whether creatinine sits inside a laboratory reference interval.
- Classify AKI as pre-renal, intrinsic renal, renal vascular or post-renal; intrinsic disease is then subdivided into glomerular, tubular and interstitial patterns.
- Urinalysis and urine microscopy are diagnostic tools, not administrative add-ons.
- CKD risk is cause + GFR + albuminuria; albuminuria and KFRE influence referral and prognosis.
- Hyperkalaemia management has three separate aims: stabilise myocardium, shift K+ intracellularly, remove K+.
- Dialysis decisions are driven by refractory complications and clinical context, not a solitary creatinine threshold.
- In dialysis DKA, fluid and potassium assumptions change because osmotic diuresis may be absent.
- In transplant AKI, look early for obstruction/vascular issues and drug interactions while involving the transplant team.

AMT MEMORY AID
Use the final page as a retrieval test. Cover the answer column and reconstruct the mechanism or next step before checking it.

MRCP RAPID RECALL
If a recall statement cannot be connected to anatomy, physiology or a clinical decision, return to the relevant figure rather than memorising it in isolation.

CLINICAL PEARL
Fast recall is safest when it compresses a fully understood pathway.

MRCP TRAP
A memorable rule is a prompt, not permission to ignore contraindications, uncertainty or patient-specific data.

AMT 60-SECOND RECALL
- Localise the lesion before naming the disease.
- Connect nephron site with transporter, electrolyte pattern and drug.
- Use time course and urine phenotype to frame AKI and glomerular disease.
- Treat hyperkalaemia as protect, shift, remove and monitor.
- Use CKD cause, G category, A category and progression risk.
- Modify fluids, potassium and drug clearance in advanced renal failure.

Appendix A Evidence and guideline version register

Organisation	Source	Version/date	Controlled content
Federation MRCP(UK)	Part 1 format and blueprint	Current page checked 23 Sep 2026	Renal: 14 indicative questions
Federation MRCP(UK)	Part 2 format and blueprint	Current page checked 23 Sep 2026	Renal: 20 indicative questions; BOF clinical scenarios
NICE NG148	Acute kidney injury: prevention, detection and management	Current recommendations; 2024 contrast update	AKI criteria, urinalysis, ultrasound, obstruction, referral
NICE NG203	Chronic kidney disease: assessment and management	Current recommendations checked 23 Sep 2026	CKD definition, ACR/eGFR, KFRE, RAAS therapy, anaemia
UK Kidney Association	Management of acute hyperkalaemia in adults	Updated July 2026	Acute hyperkalaemia pathway; dose verification required
NICE TA1148	Sodium zirconium cyclosilicate for treating hyperkalaemia	Published 29 Apr 2026	Persistent/acute hyperkalaemia technology appraisal

Organisation	Source	Version/date	Controlled content
KDIGO	2024 CKD Evaluation and Management guideline	2024; summary resources updated 2026	International CKD framework/contrast
JBDS-IP	The Management of DKA in Adults	March 2023	Special section for end-stage renal failure/dialysis
JBDS-IP / UKKA	Management of adults with diabetes on dialysis	March 2023	DKA/dialysis context
NICE NG185	Acute coronary syndromes	2020; surveillance reviewed 27 Mar 2026	ACS in renal impairment; antithrombin principles
BNF	BNF online	Current licensed resource; final dose check required	Medicine doses, contraindications, interactions

Appendix B Physician approval and specialist advisory register

Physician approval was recorded on 27 October 2026. The specialist streams below remain advisory for future updates, local formulary alignment and discipline-specific refinement; they do not prevent publication of this edition.

Advisory stream	Scope	Status
Consultant nephrologist	All renal science, AKI/CKD, GN, RRT, transplant and prognosis	Advisory
Renal pharmacist / prescribing clinician	All drug doses, renal adjustment, interactions, hyperkalaemia and transplant pharmacology	Advisory
Diabetologist	DKA in advanced renal failure/dialysis	Advisory
Transplant nephrologist/pharmacist	Transplant AKI, rejection differential, immunosuppression toxicity	Advisory
Cardiology reviewer	ACS/MI in CKD crossover section	Advisory
MRCP educator	P1/P2 balance, distractor quality, difficulty	Advisory
Medical copyeditor	British English, consistency, readability, references	Advisory
Glomerular disease	MCD/FSGS/membranous response and lesion-specific immunosuppression; SLE and infection-related disease	Advisory
Dialysis and UTI	Acute dialysis thresholds, modality complications and local antimicrobial susceptibility/dosing	Advisory
ADPKD and renal masses	Aneurysm screening, tolvaptan eligibility and Bosniak/solid-mass pathway	Advisory
Synthetic imaging	Every synthetic scan and pathology-style illustration versus real clinical radiology and biopsy	Advisory

Appendix C Image and licence register

Asset ID	Illustration and provenance	Licence / safety label
REN-V6-01	Composite of existing original AMT kidney cutaway REN-01 with programmatic infection annotations	AMT project artwork; AI-assisted anatomy, explanatory overlay, not a patient scan.
REN-V6-02	Composite of existing original AMT kidney cutaway REN-01 with programmatic tumour annotations	AMT project artwork; AI-assisted anatomy, explanatory overlay, not clinical imaging.
REN-V6-03	Composite of existing original AMT kidney cutaway REN-01 with programmatic BP annotations	AMT project artwork; AI-assisted anatomy, explanatory overlay.
REN-V6-04	Composite of existing original AMT kidney cutaway REN-01 with programmatic transplant annotations	AMT project artwork; AI-assisted anatomy, representative drawing, not diagnostic histology.

ID	Asset	Method	Licence/safety label
REN-01	Renal anatomy and nephron plate	AI-assisted original educational illustration	AMT educational illustration; anatomically oriented; not a diagnostic image
REN-02	AKI categories	AI-assisted atlas art	Original AMT educational artwork; educational only
REN-03	CKD G/A grid	AI-assisted atlas art	Original AMT educational artwork; conceptual risk display
REN-04	Transplant AKI pathway	AI-assisted atlas art	Original AMT educational artwork; educational only
REN-M01	Nephron transporter and drug-site memory map	AI-assisted medical atlas art with AMT annotations	Original AMT artwork; synthetic imaging; physician reviewed for educational use
REN-M02	RAAS mechanism and pharmacology chain	AI-assisted medical atlas art with AMT annotations	Original AMT artwork; synthetic imaging; physician reviewed for educational use
REN-M03	Renal imaging modality atlas	AI-assisted medical atlas art with AMT annotations	Original AMT artwork; synthetic imaging; physician reviewed for educational use
REN-M04	Acid-base diagnostic ladder	AI-assisted medical atlas art with AMT annotations	Original AMT artwork; synthetic imaging; physician reviewed for educational use
REN-M05	RTA and potassium pattern map	AI-assisted medical atlas art with AMT annotations	Original AMT artwork; synthetic imaging; physician reviewed for educational use
REN-M06	Glomerular LM/IF/EM pattern map	AI-assisted medical atlas art with AMT annotations	Original AMT artwork; synthetic imaging; physician reviewed for educational use
REN-M07	CKD complication memory map	AI-assisted medical atlas art with AMT annotations	Original AMT artwork; synthetic imaging; physician reviewed for educational use
REN-M08	Renal-stone recognition grid	AI-assisted medical atlas art with AMT annotations	Original AMT artwork; synthetic imaging; physician reviewed for educational use
REN-M10	ACS and DKA renal modification	AI-assisted medical atlas art with	Original AMT artwork; synthetic

	map	AMT annotations	imaging; physician reviewed for educational use
REN-M11	Diuretic sites of action map	AI-assisted medical atlas art with AMT annotations	Original AMT artwork; synthetic imaging; physician reviewed for educational use
REN-M12	Normal versus raised anion-gap metabolic acidosis map	AI-assisted medical atlas art with AMT annotations	Original AMT artwork; synthetic imaging; physician reviewed for educational use
REN-M13	Complement-pattern comparison	AI-assisted medical atlas art with AMT annotations	Original AMT artwork; synthetic imaging; physician reviewed for educational use
REN-M14	Urgent kidney replacement therapy indications	AI-assisted medical atlas art with AMT annotations	Original AMT artwork; synthetic imaging; physician reviewed for educational use
REN-M16	Immunosuppressant adverse-effect and interaction map	AI-assisted medical atlas art with AMT annotations	Original AMT artwork; synthetic imaging; physician reviewed for educational use
REN-M17	Renal corpuscle and nephrotic pathology teaching plate	AI-assisted original AMT synthetic educational artwork	Author-owned new figure; not a patient study; physician reviewed for educational use
REN-M18	Synthetic renal ultrasound and CT comparison	AI-assisted original AMT synthetic educational artwork	Author-owned new figure; not a patient study; physician reviewed for educational use
REN-M19	Dialysis and peritoneal access illustration	AI-assisted original AMT synthetic educational artwork	Author-owned new figure; not a patient study; physician reviewed for educational use

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KDIGO ADPKD 2025, incl. aneurysm screening — https://kdigo.org/guidelines/autosomal-dominant-polycystic-kidney-disease-adpkd/

NICE NG109 lower UTI, current online recommendations — https://www.nice.org.uk/guidance/ng109/chapter/Recommendations

NICE NG111 acute pyelonephritis, current online recommendations — https://www.nice.org.uk/guidance/ng111/chapter/Recommendations

NICE NG107 kidney replacement and conservative management — https://www.nice.org.uk/guidance/ng107/chapter/Recommendations

EAU Renal Cell Carcinoma 2026 — https://uroweb.org/guidelines/renal-cell-carcinoma

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ISPD Peritonitis Guideline 2022 — https://journals.sagepub.com/doi/10.1177/08968608221080586

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