

2: KIDNEY

Q1. RENAL TUBERCULOSIS

Definition & Route of Infection

- Renal TB = *secondary haematogenous spread*
- Primary focus: *distant TB infection*
- Progressive renal parenchymal destruction → lower urinary tract involvement

Pathological Progression (Sequential Lesions)

Stage	Morphology
Papillary ulcer	Cortical granulomas coalesce → ulcerate into papilla
Cavernous TB	Progressive necrosis → pus-filled cavities
Putty / cement kidney	Autonephrectomy; shrunken non-functioning kidney with caseous or calcific material
Secondary sequelae	Hydronephrosis, pyonephrosis, capsular breach → perinephric abscess

Lower Tract & Systemic Involvement

- **Ghost calyx:** moth-eaten calyceal outline
- **Ureteric TB:**
 - Fibrosis + scarring
 - Beaded ureter
 - Kerr's kink at PUJ
- **Bladder TB:**
 - Chronic fibrosis
 - **Thimble (systolic) bladder**
 - Severely reduced capacity
- **Golf-hole ureter:**
 - Rigid, permanently open ureteric orifice
- **Genital spread (males):**
 - Epididymo-orchitis
 - Beaded vas deferens

Clinical Features

- **Sterile pyuria:** hallmark
- **Persistent loin pain**
- **Haematuria**
- **Palpable renal mass:** late; hydronephrosis / abscess
- **Constitutional symptoms:**

- Weight loss
- Fatigue
- Evening rise of temperature
- Lower urinary tract symptoms:
 - Frequency
 - Dysuria
 - Reduced bladder capacity

Investigations

Modality	Purpose
CT urography	Investigation of choice; disease extent
Urine microscopy	Sterile pyuria
Microbiology	≥ early-morning urine samples → ZN stain for AFB + TB culture
Plain X-ray	Renal calcifications

Management Principles

- Combined medical + surgical
- Organ preservation whenever feasible
- Surgery only after ATT optimisation

Treatment

- Medical therapy:
 - Standard anti-tubercular therapy (ATT)
 - Initiated weeks before surgery
- Surgical interventions (indication-based):
 - Pigtail drainage → perinephric abscess
 - Double-J stenting → ureteric stricture / kinking
 - Hanley's cavernostomy → isolated pyocalyx
 - Augmentation cystoplasty (ileal segment) → thimble bladder
 - Nephroureterectomy:
 - Non-functioning kidney
 - Extensive renal destruction
 - RCC association

Q2. HYDRONEPHROSIS

Definition & Overview

- Hydronephrosis = aseptic dilatation of pelvi-calyceal system (PCS)
- Mechanism: partial / intermittent urinary outflow obstruction

- Laterality: unilateral or bilateral
- Nature: congenital (primary) or acquired (secondary)

Pathological Progression

- Obstruction → ↑ intrapelvic pressure
- Initial pelvic dilatation → calyceal involvement → renal parenchymal transmission
- Chronic pressure → parenchymal thinning + destruction
- Functional threshold: parenchymal thickness <2 mm → negligible renal function
- Calyceal morphological sequence
 - Normal cup-shaped
 - Blunted
 - Clubbed
 - Broad, ballooned
- Pelvic anatomy influence
 - Extrarenal pelvis (80%): pelvic dilatation precedes parenchymal damage
 - Intrarenal pelvis (20%): early parenchymal destruction

Etiology — Anatomical Classification

Unilateral Causes

Level	Causes
Intraluminal	Renal calculi (MC acquired), sloughed papillae, blood clots
Intramural	PUJ obstruction (MC congenital; adynamic), ureterocele, ureteric TCC, post-traumatic / post-surgical / TB strictures
Extramural	Aberrant renal vessels (L > R), retroperitoneal fibrosis (Ormond's), retrocaval ureter, pelvic malignancies (cervix, rectum)

Bilateral Causes

Site	Causes
Bladder outlet	BPH, carcinoma prostate, bladder neck tumours
Urethra	Posterior urethral valves (children), strictures, phimosis, meatal stenosis
Functional / Others	Pregnancy (progesterone-induced atony), neurogenic bladder, bilateral occurrence of unilateral causes

Clinical Features

- Unilateral hydronephrosis
 - Dull aching loin pain
 - Dragging heaviness
 - Smooth, mobile, ballotable loin mass

- **Dietl's crisis**
 - Loin pain + swelling
 - Sudden disappearance after passage of large urine volume
- **Bilateral hydronephrosis**
 - LUTS: frequency, poor stream
 - Progressive renal failure
 - Oliguria, anaemia, oedema

Management Principles

- Primary aim: renal preservation
- Nephrectomy: non-functional kidney (<10–20% function)

Definitive Etiology-Based Treatment

- Renal calculi → lithotomy
- BPH → TURP
- Obstructing tumours → excision

Reconstructive Surgery

- PUJ obstruction → Anderson–Hynes dismembered pyeloplasty
 - Excision of adynamic segment
 - Redundant pelvis removal
 - Dependent re-anastomosis

Urinary Diversion

- Acute / severe obstruction → nephrostomy
- Internal drainage → Double-J (DJ) stenting

Bilateral Disease with Renal Failure

- Initial: bilateral nephrostomy + haemodialysis
- Definitive surgery: better kidney first
- Second side after ~3 months

Minimally Invasive Approaches

- Laparoscopic pyeloplasty
- Retroperitoneoscopic pyeloplasty

Q3. POLYCYSTIC KIDNEY DISEASE

- Genetic renal disorder → multiple parenchymal cysts
- Cysts form at distal tubule–collecting duct junction → parenchymal destruction

Type	Inheritance	Gene	Notes

Infantile	AR	PKHD1 (Chr 6)	Often lethal; hepatic fibrosis
Adult	AD	PKD1 (Chr 16), PKD2 (Chr 4)	Life compatible; common

- Adult PKD: asymptomatic until 30–40s
- Hypertension (early, common)
- Bilateral, lobular, firm, ballotable kidneys
- Pain: dull (capsular stretch) or sharp (cyst haemorrhage)
- Haematuria ~25%; late renal failure → uraemia

Extra-renal features	Notes
Liver cysts	18–30%
Berry aneurysm	Circle of Willis
Others	Mitral valve prolapse, colonic diverticulosis

- Investigations: US (multiple cysts), IVU (“spider leg” pattern), blood/urine tests (↑ urea/Cr, low urine SG)
- Management:
 - Asymptomatic → conservative, vasopressin antagonists, MTOR inhibitors
 - Symptomatic cysts → Rovsing operation, US-guided aspiration, laparoscopic de-roofing
 - End-stage → haemodialysis → bilateral nephrectomy + renal transplant

Q4. RENAL CALCULUS

Basic Concepts & Epidemiology

- Renal calculi: crystalline concretions within renal collecting system
- Male predominance
- ~90% radio-opaque on X-ray (contrast with gallstones)

Etiopathogenesis (Multifactorial)

- Dietary–metabolic factors
 - Vitamin A deficiency → epithelial desquamation → nidus formation
 - ↓ Urinary citrate → loss of calcium salt solubility
- Environmental factors
 - Hot climate → ↑ sweating → concentrated urine → ↑ solute, ↓ colloids
- Infective factors
 - Urea-splitting organisms: *Proteus*, *Staphylococcus*, *E. coli*
 - Alkaline urine → phosphate stone formation
- Stasis & obstruction
 - Medullary sponge kidney
 - Anatomical obstruction → urinary stasis

- Systemic causes
 - Hyperparathyroidism → hypercalciuria → bilateral/multiple stones
 - Prolonged immobilisation → bone decalcification → hypercalciuria
- Theories of formation
 - Randall's plaque theory → papillary erosions
 - Carr's postulates → lymphatic obstruction → microliths

Types of Renal Calculi (Composition & Features)

Type	Key Characteristics
Oxalate (75%)	Brown, spiculated "mulberry"; very hard; acidic urine
Phosphate (10–15%)	Smooth, white; alkaline/infected urine; staghorn calculus
Uric acid	Radiolucent; gout, high purine metabolism
Cystine (2%)	Hexagonal, soft, yellow; radio-opaque (sulphur)
Rare	Xanthine (brick red), Indinavir

Clinical Features

- Pain
 - Renal pain: renal angle, loin, lumbar region
 - Colic: waxing–waning; loin → groin → genital tip
 - Vomiting due to reflex pylorospasm
- Haematuria: microscopic or gross
- Loin mass: hydronephrosis; smooth, soft, bimanually palpable
- Dietl's crisis: loin swelling during pain → sudden diuresis → relief
- Associated features: pyuria, fever, UTI signs

Q5. DIETL'S CRISIS

Definition

- Intermittent obstructive uropathy in hydronephrosis / renal calculi
- Sudden relief of obstruction → characteristic symptom sequence

Stage	Clinical event
1	Acute severe renal colic, loin pain
2	Palpable lumbar mass from aseptic PCS dilatation

Resolution phase	Finding
Mass	Sudden disappearance
Urine	Polyuria with dilute urine

- Classical “Flush tank sign”: pain + mass → sudden polyuria
- Diagnostic of intermittent ureteropelvic obstruction

Investigations

Modality / Test	Purpose
NCCT KUB	Gold standard; size, site, density
X-ray KUB	90% stones visible; follow-up
Ultrasound	Radiolucent stones; hydronephrosis
IVU	Renal function; anatomy
Blood	Ca ²⁺ , PO ₄ ³⁻ , uric acid, PTH
Urine	pH; crystals—envelope (oxalate), coffin-lid (triple phosphate)

Management

- Conservative
 - Stones <5 mm → spontaneous passage
 - Hydration (“flush therapy”)
 - Tamsulosin to facilitate expulsion
- ESWL
 - Stones <2.5 cm
 - Contraindicated: pregnancy, cystine stones
- PCNL
 - >2.5 cm stones; staghorn calculi
- RIRS
 - Flexible ureteroscope + Holmium laser; lower pole stones
- Open surgery (rare)
 - Pyelolithotomy, nephrolithotomy

Complications

- Infection → pyonephrosis, perinephric abscess
- Obstructive uropathy → renal failure (esp. bilateral)
- Ureteric strictures post-stone or intervention

Q6. Staghorn Calculus (Struvite / Triple Phosphate Stone)

- Renal calculus occupying pelvis + calyces; branching configuration
- Composition: Ca-Mg-NH₄ phosphate ± carbonate
- Forms in alkaline urine; chronic UTI background
- Urease-producing organisms, esp. *Proteus* → ↑ ammonia
- Smooth surface; massive size; radiopaque

- Microscopy: “coffin-lid” crystals
- X-ray KUB: branching pelvicalyceal cast

Key characteristics	Details
Urine pH	Alkaline
Infection	Chronic UTI
Radiology	Radio-opaque
Microscopy	Coffin-lid crystals

- PCNL: preferred for uni/bilateral stones
- Open nephropylolithotomy: large burden
- Anatomic nephrolithotomy: giant stones; Brodel line
- Nephrectomy: <20% function / pyonephrosis

Special situations	Management
Bilateral stones	Operate better kidney first
Pyonephrosis + RF	Bilateral nephrostomy
Infection control	Antibiotics; acetohydroxamic acid (urease inhibitor)

Q6. HEMATURIA

Definition & Classification

- Presence of blood in urine
- Gross haematuria: visible discoloration
- Microscopic haematuria: >5 RBCs/HPF on microscopy

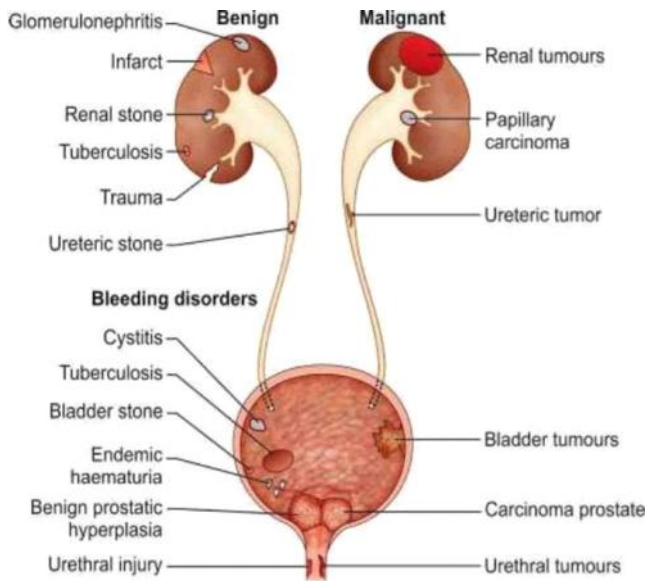
Timing of Haematuria & Source Localization

Timing during micturition	Likely anatomical source
Initial (early)	Urethra distal to external sphincter
Terminal	Bladder neck, prostate
Total (diffuse)	Bladder, upper urinary tract

Etiological Classification by Site

Site	Causes
Kidney	RCC (commonest symptom), Wilms' tumour, renal trauma, renal calculi (Ca-oxalate “mulberry”), renal TB, renal infarct, PCKD, glomerulonephritis
Ureter	Ureteric calculi, ureteric tumours

Bladder	Vesical calculus, TCC bladder (gross painless haematuria), cystitis, urinary bilharziasis (<i>Schistosoma haematobium</i>)
Prostate	BPH ("vesical piles"), carcinoma prostate
Urethra	Urethral injury, urethral tumours
Systemic	Blood dyscrasias, thrombocytopenia, anticoagulant use



Important Diagnostic Concepts

- Silent haematuria: tumour kidney/bladder until proven otherwise
- False haematuria: food pigments, myoglobinuria
- Haemoglobinuria: free Hb, no intact RBCs
- Decoy prostate: premature attribution to BPH

Investigations

- Urine: routine microscopy (RBCs), cytology (malignant cells), culture & sensitivity
- Blood: Hb%, PCV, blood urea, serum creatinine, BT, CT, PT, platelet count
- Imaging: USG-KUB; IVU—functional anatomy, filling defects; CECT—tumour extent, nodes, small ureteric stones
- Cystourethroscopy: direct visualization, biopsy confirmation

Q7. RENAL CELL CARCINOMA (GTAWITZ TUMOR, Hypernephroma)

Definition & Epidemiology

- RCC = primary renal adenocarcinoma
- Synonyms: Grawitz tumour, Hypernephroma
- Origin: renal tubular epithelium, mainly proximal renal tubule

- ~3% of adult malignancies
- Male predominance; peak incidence: 5th–6th decade

Etiopathogenesis

- Environmental / lifestyle risk factors
 - Cigarette smoking
 - Hypertension
 - Obesity
 - High animal-protein diet
 - Thorotrast exposure
 - Heavy metals: cadmium, lead
 - Asbestos exposure
- Genetic / familial associations
 - Sporadic majority
 - Von Hippel–Lindau (VHL) syndrome → chromosome 3p deletion → clear cell RCC
 - Birt–Hogg–Dubé syndrome → chromophobe RCC
 - Hereditary papillary RCC → MET gene mutation

Histological Variants

Variant	Origin / Features
Clear cell (75%)	PCT origin; most common
Papillary (15%)	Often multifocal; MET mutation
Chromophobe (5%)	Collecting duct origin; plant-like cells; perinuclear halo
Collecting duct of Bellini (1%)	Medullary ducts; worst prognosis

Clinical Features (Internist's Tumour)

- Haematuria: most common symptom (30–60%)
- Classic triad: haematuria + loin pain + flank mass (15–20% → advanced disease)
- Loin pain: capsular stretch
- Palpable renal mass: late sign
- Left-sided irreducible varicocele
 - Mechanism: tumour thrombus → left renal vein → left testicular vein obstruction
- Paraneoplastic syndromes (~20%)
 - Anaemia (most common)
 - Erythrocytosis → ↑ erythropoietin
 - Hypercalcaemia
 - Hypertension

- Stauffer's syndrome
 - Reversible hepatic dysfunction
 - ↑ LFTs, bilirubin
 - No liver metastasis
 - Resolution after nephrectomy

Patterns of Spread

- Haematogenous metastasis predominant
- Common sites: lungs (cannonball secondaries), bones, liver, brain
- Venous tumour thrombus
 - Renal vein → IVC → right atrium

Investigations

Modality	Role
CECT abdomen–pelvis	IOC; tumour size, extent, nodes
MRI	IVC / atrial tumour thrombus
CT angiography	Vascular anatomy; venous invasion
Chest X-ray / CT	Pulmonary metastases
Biopsy	Avoid if resectable; use in doubtful or metastatic disease

Staging (TNM – Prognostic Core)

- Stage I: ≤7 cm, kidney-confined
- Stage II: >7 cm, kidney-confined
- Stage III: major veins / regional nodes
- Stage IV: beyond Gerota's fascia or distant metastasis

Treatment

- RCC resistant to chemotherapy & radiotherapy
- Surgery = mainstay
 - Partial nephrectomy
 - T1 tumours ≤7 cm
 - Polar lesions
 - Solitary kidney / bilateral RCC
 - Radical nephrectomy
 - Kidney + Gerota's fascia
 - Ipsilateral adrenal
 - Proximal 2/3 ureter
- Ablative options
 - Cryoablation

- Radiofrequency ablation
- Small T1a tumours; elderly / high-risk patients
- Metastatic RCC
 - Sorafenib, Sunitinib (TKIs)
 - Interleukin-2, Nivolumab (immunotherapy)

Q8. Intravenous Pyelogram (IVP / IVU)

- IV contrast X-ray study of kidneys, ureters, bladder
- Requires normal renal function, fasting, bowel prep
- Preliminary plain KUB mandatory

Contrast protocol	Details
Test dose	1 ml iodine dye
Full dose	1 ml/kg (Infusion IVU 2 ml/kg)

Key IVP signs	Condition
Cobra / adder head	Ureterocele
Spider-leg	PCKD / RCC
Clubbed calyces	Hydronephrosis
Reverse-J	Retrocaval ureter

- Contraindications: iodine allergy, myeloma, toxic thyroid, poor renal function, bilirubin > 3 mg%

Q9. CYSTOSCOPY

- Endoscopic visualisation of urethra & bladder
- Types: Rigid, flexible
- Indications: Tumours, infections, fistulas, TURP, urethrotomy, lithotripsy, valve fulguration
- Procedure: Lithotomy position, GA/spinal anaesthesia, continuous irrigation (glycine)
- Contraindications: Acute cystitis/prostatitis
- Complications: Urethral injury, bleeding, water intoxication

Q10. HORSE-SHOE KIDNEY

Anatomy	Fusion at lower poles, isthmus anterior to aorta (L4-L5), ascent blocked by IMA
Clinical	Often asymptomatic; may have stones, infection, palpable mass, hydronephrosis

Radiology	IVU → “flower vase”/“handshake” sign; CT confirms fusion
Management	Treat complications (pyeloplasty); avoid cutting isthmus unless necessary

- Focus: Complication management; preserve renal vascularity.

Q11. URETEROCELE

- Congenital cystic dilatation of intramural ureter; often female, sometimes bilateral
- **Presentation:** Recurrent UTIs, obstructive uropathy, progressive renal dysfunction
- **Investigations:** IVU → “Cobra/Adder head” sign; US → dilated ureteric ends; Cystoscopy → thin-walled fluctuating cyst
- **Management:** Endoscopic ureteric meatotomy; surgical excision + ureteric reimplantation if complex

Q12. URETERIC CALCULI

Basic Concept & Composition

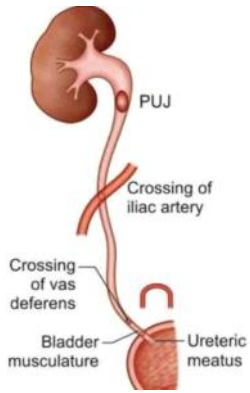
- Renal-origin calculus descending into ureter
- Shape elongated, adapting to ureteric lumen
- Chemical composition identical to renal stones
- Common constituents: Ca-oxalate (most common), phosphate, uric acid, cystine

Clinical Presentation

- Acute ureteric colic: sudden, severe, colicky pain
- Radiation: loin → groin → genital tip
- Neural basis: genitofemoral nerve referral
- Autonomic features: nausea, vomiting, sweating (reflex pylorospasm)
- Urinary symptoms: haematuria, dysuria, ↑ frequency
- Strangury: intramural ureteric impaction → intense urge, scanty bloody urine
- Examination: iliac fossa + renal angle tenderness; rebound tenderness absent

Anatomical Sites of Impaction

Site	Anatomical Narrowing
PUJ	Pelvi-ureteric junction
Pelvic brim	Crossing iliac vessels
Pelvic crossing	Vas deferens / broad ligament
Intramural ureter	Entry through bladder wall
Terminal ureter	Ureteric orifice (narrowest)



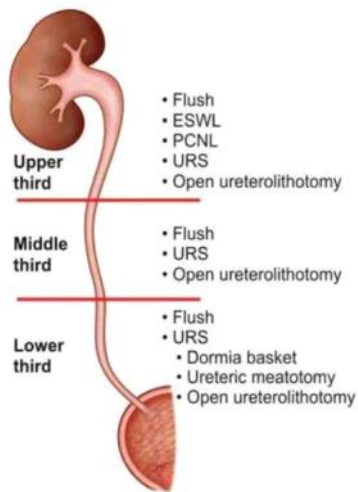
Investigations

- NCCT-KUB: investigation of choice
- X-ray KUB: ~90% radio-opaque stones; ureteric line landmarks
- Ultrasonography: radiolucent stones, hydronephrosis
- IVU: renal function, obstruction level

Management Principles

- Decision factors: size, site, complications

Situation	Modality
<5–8 mm	Observation, fluids, analgesia
Lower ureter	Flush therapy + frusemide 60–80 mg; tamsulosin
Upper ureter	ESWL → PCNL if failure
Mid ureter	URS + Holmium-YAG / pneumatic lithotripsy
Lower ureter	Dormia basket (<10 mm); ureteric meatotomy
Failure/complex	Open ureterolithotomy



Complications

- Hydroureter
- Pyonephrosis
- Ureteric stricture

Q13. WILMS TUMOR (NEPHROBLASTOMA)

Definition & Epidemiology

- Wilms tumour = nephroblastoma
- Most common paediatric renal malignancy
- 2nd most common abdominal malignancy in children
- Peak age: 2–5 years
- Origin: embryonic nephrogenic tissue
- Histology: mixed epithelial + mesenchymal (connective tissue) elements

Pathobiology & Spread

- Arises from primitive renal blastema
- Growth pattern: expansile renal mass
- Vascular invasion → renal vein tumour thrombus
- Metastatic route: haematogenous
 - Lungs (MC)
 - Liver
 - Rare: bone

Clinical Presentation

Feature	Description
Abdominal mass	Painless loin mass; smooth, firm/hard, lobulated
Palpation	Bimanually palpable, ballotable
Respiration	Moves with respiration
Midline	Rarely crosses midline (vs neuroblastoma)
Percussion	Resonant colonic band anteriorly
Haematuria	Grave sign; tumour rupture into pelvis
Hypertension	~25% cases
Fever	Tumour necrosis
Syndromic associations	WAGR, Beckwith-Wiedemann, Denys-Drash

Syndromic Associations

- WAGR

- Wilms tumour
- Aniridia
- Genitourinary anomalies
- Mental retardation
- Beckwith-Wiedemann syndrome
- Denys-Drash syndrome

Management Principles

- Tumour is chemo-sensitive + radiosensitive
- Treatment guided by stage + histology
- Multimodal therapy essential

Treatment Protocols

Component	Details
International strategies	NWTSG: upfront surgery → chemo ± RT; SIOP: pre-op chemo → surgery → adjuvant therapy
Surgery	Radical nephrectomy for unilateral disease
Bilateral disease (~5%)	Nephron-sparing surgery; bilateral partial nephrectomy OR total one side + partial other
Chemotherapy	Vincristine, Actinomycin-D (Dactinomycin), Doxorubicin
Radiotherapy	Stage III onwards; pre-op use if locally inoperable
Vascular extension	Nephrectomy with renal vein thrombus clearance

Staging & Prognosis

- Prognosis correlates strongly with:
 - Tumour stage
 - Histological subtype
- Anaplastic histology → poor prognosis
- Early-stage, favourable histology → excellent survival

Key Exam Pearls

- Painless paediatric loin mass → Wilms until proven otherwise
- Does not cross midline
- Lung metastasis = most common distant spread
- Bilateral disease → preserve nephrons
- Haematuria = pelvic breach → adverse sign

Q14. RETROGRADE PYELOGRAPHY

- Diagnostic radiology for upper urinary tract when IVU fails
- Indications: Non-visualisation on IVU, urinary TB, renal pelvis tumours

- **Procedure:** Under GA, cystoscope → ureteric catheter → sodium diatrizoate injection, 15° head-down tilt → X-rays
- **Advantages:** Higher dye concentration, selective ureteric urine sampling, brush biops

Q15. Renal Function Tests (RFTs)

- **Biochemical:** Serum creatinine, BUN (10–20 mg/dL), creatinine clearance (M:140–150, F:105–130 mL/min), urine SG 1.010–1.025, protein/casts
- **Isotope Renography:** Tc-99m DTPA → GFR; Tc-99m DMSA → parenchymal scarring; differential GFR <10% → non-functional

Q16. Percutaneous Nephrolithotomy

Indications	Stones >2.5 cm, staghorn, lower calyx stones unsuitable for ESWL, failed ESWL/RIRS
Procedure	Prone, cystoscopic ureteric stent, calyceal puncture under US/fluoroscopy, track dilation, nephroscope, fragment stones (Holmium-YAG/pneumatic/ultrasonic)
Complications	Haemorrhage, infection, pneumothorax, colon injury

Q17. Extracorporeal Shock Wave Lithotripsy

(ESWL)	Notes
Indications	Stones 5–25 mm, upper ureter, suitable composition
Principle	Focused ultrasonic shock waves, 1,000–4,000 shocks/session, usually outpatient
Contraindications	Pregnancy, obesity, bleeding disorders, pacemaker, hard stones (cystine/CaOx monohydrate), lower calyx stones, children
Complications	Pain, haematuria, steinstrasse (DJ stent prophylaxis), renal hematoma, UTI
Alternatives	RIRS <2 cm, PCNL >2 cm, staghorn, failed ESWL