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Clinical Cases & Pearls in MEDICINE





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Section 4

Nephrology

Outlines

Case 1 Nephrotic Syndrome
Case 2 Uremia (Chronic Renal Failure)
Case 3 Polycystic Kidney Disease
Case 4 Unilateral Palpable Kidney
Case 5 Acute Renal Failure
Case 6 Acute Tubular Necrosis
Case 7 Hypernephroma (Renal Adenocarcinoma)

BASIC CONSIDERATIONS

Kidneys are retroperitoneal organs and each measures 11–14 cm in size. Nephron is a functional unit of kidney. It has rich supply of blood (25% of cardiac output at rest is utilized by kidneys). The glomerulus consists of bunch of capillaries placed in **Bowman's capsule**.

Juxtaglomerular apparatus (JGA), which secrets renin is a collection of specialized cells lying near the junction of afferent arteriole and convoluted tubule. Blood during its passage through glomerular capillaries, filters out (by process of diffusion) water and solutes across the glomerular capillary membrane. In healthy persons, glomerular filtrate is like plasma and has very little proteins. In glomerular nephritis, there is proteinuria. In healthy persons, glomerular filtration rate (GFR) remains constant. Renal perfusion pressure and renal blood flow are regulated by alterations in the relative tone of the afferent and efferent arterioles via release of renin by JGA in response to the changes in the delivery of solutes to the macula densa.

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CASE 1: NEPHROTIC SYNDROME

INTRODUCTION

Nephrotic syndrome presents with generalized edema, pallor, puffy face **(Fig. 4.1)**, low serum albumin (< 3 g/dL), albuminuria (> 3.5 g/day) and elevated cholesterol levels (> 300 mg/dL).



Fig. 4.1 A case of nephrotic syndrome showing pallor, puffy face

ETIOPATHOLOGICAL FACTORS

1. **Glomerular disease: Majority (75% due to glomerular pathology)**
   - Minimal change (glomerulonephritis), this is the most common form (90% in children)
   - Membranous glomerulonephritis
   - Membranoproliferative glomerulonephritis
   - Mesangioproliferative glomerulonephritis
   - Focal glomerulosclerosis
2. **Systemic diseases causing nephropathy**
   - *Metabolic:* Diabetes mellitus (DM)
   - *Infections:* Malaria, infective endocarditis, syphilis, HIV
   - *Collagen disorders:* Systemic lupus erythematosus (SLE), vasculitis, amyloidosis
3. **Drug toxic effects:** Nonsteroidal anti-inflammatory drugs (NSAIDs), gold, penicillamine, rifampin, captopril, lithium, tolbutamide
4. **Hypersensitivity reactions**
5. **Malignant diseases:** Lymphomas, Hodgkin's, multiple myeloma
6. **Heredofamilial: Alport syndrome**, sickle cell disease
7. **Parasitic diseases:** Malaria, toxoplasmosis, schistosomiasis
8. **Idiopathic**.

SALIENT CLINICAL FEATURES

- Minimal change disease is the commonest etiopathological cause and nephrotic syndrome is more commonly seen in children and adolescents
- It has insidious onset and presents with generalized anasarca, puffy face, pallors, ascites, swollen abdomen, scrotum and legs. Blood pressure is normal
- Nails show white ridges due to hypoalbuminemia
- Proteinuria (24 hours urinary albumin > 3.5 g), hypoalbuminemia (serum albumin < 3 g/dL), Hb low, cholesterol high because of increased production by the liver
- Renal biopsy gives the diagnosis of type of glomerular disease

Diabetic nephropathy occurs in patients with long standing uncontrolled DM. It is due to microangiopathy and is usually associated with retinopathy and neuropathy. Patient has proteinuria, hypoproteinemia, hypertension, and nephrotic syndrome resulting in kidney failure in few years.

INVESTIGATIONS TO ASCERTAIN ETIOLOGY OF THE DISEASE

- Complete blood count (CBC), erythrocyte sedimentation rate (ESR)

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- Complement levels, DNA antibody, antinuclear factors
- Presence of DM (fasting and 2 hours PPBS and glycosylated Hb)
- Presence of collagen disorder, e.g. SLE
- Type of glomerular lesion on kidney biopsy
- Exclusion of renal vein thrombosis.

ASSESSMENT OF NEPHROTIC SYNDROME FOR SEVERITY OF DISEASE

- Serum albumin level
- 24 hours urinary albumin level (> 3.5 g)
- Creatinine clearance
- Level of hypercholesterolemia.

PROGNOSIS

Minimal lesion has best prognosis of all other glomerular lesions.

COMPLICATIONS

- Infections (peritonitis, lung infections)
- Atherosclerosis and xanthomas (hypercholesterolemia plays an important part)
- Deep vein thrombosis resulting in pulmonary embolism and other thromboembolic events, accelerated atherosclerosis. Thromboembolic events occur due to loss of antithrombin III and other proteins involved in the coagulations and fibrinolytic cascade
- *Renal failure*
- Protein malnutrition.

MANAGEMENT

- Ensure that patient takes bed rest and avoids heavy exertion
- *Diet:* Proteins-quantity allowed according to severity of glomerular damage and GFR values
  - If GFR is normal (> 60 mL/min), do not restrict proteins
  - If GFR is less than 60 mL/min, protein intake restricted to 0.8 g/kg/day plus 1 g protein/g for proteinuria
- Diuretics for edema (furosemide)
- *Prevention of thromboembolism:* Anticoagulants, heparin and elastic stockings
- Salt free albumin infusions to be administered in generalized anasarca
- Control of DM and hypertension with angiotensin converting enzyme (ACE) inhibitors along with careful monitoring of serum K-levels. ACE inhibitors or angiotensin receptor blockers also helpful with normal blood pressure to reduce proteinuria
- Plasma ultrafiltration in severe cases
- Pneumococcal vaccine
- Prednisolone (8 weeks course) in minimal change nephropathy is helpful
- *Interferon alfa* is helpful in hepatitis B associated nephrotic syndrome; hepatitis C associated membranous proliferative glomerulonephritis and cryoglobulinemia.

VIVA VOCE

Q1. Describe orthostatic proteinuria.
It occurs usually in adolescents and on standing for a long period of time and resolves on lying down. Prognosis is good.

Q2. Describe importance of Tamm-Horsfall protein in the urine in nephrotic syndrome.
Healthy person excretes less than 150 mg/day proteins, of which 5–15 mg is albumin and the rest consists of different plasma proteins and glycoproteins derived from kidney cells. **Tamm-Horsfall mucoprotein** is the maximum protein that is not derived from the plasma but from the renal cells of the ascending limb of the loop of Henle. It is excreted at the rate of 50–75 mg/day. Pathological proteinuria occurs when daily excretion is more than 150 mg protein/day.

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CASE 2: UREMIA (CHRONIC RENAL FAILURE)

INTRODUCTION

This is the stage when there is significant and permanent reduction of GFR. Here, end products of protein metabolism are elevated in the blood. It invariably results in death unless hemodialysis and later renal transplant are carried out.

CAUSES

- Glomerulonephritis (proliferative, membranoproliferative and crescentic glomerulonephritis)
- Vascular (renovascular vasculitis, polyarteritis nodosa, SLE)
- Obstructive uropathy (enlarged prostate, renal stones, kidney tumors, e.g. hypernephroma, retroperitoneal fibrosis)
- Metabolic diseases (DM, gout, amyloidosis)
- *Interstitial:* Chronic interstitial nephritis, chronic pyelonephritis, analgesic nephropathy, tuberculous pyelonephritis
- *Congenital:* Polycystic kidneys, **Alport's syndrome**, cystic medullary necrosis.

SALIENT CLINICAL MANIFESTATIONS

- *Central nervous system (CNS):* Confusion, coma, peripheral neuropathy, irritability, flapping tremors (asterixis), convulsions, fasciculations, restless legs
- *General:* Lethargy, weakness, weight loss, pallor, muscle weakness, epistaxis, bruising, pruritus, amenorrhea, impotence, edema of legs, nocturia, polyuria, dyspnea **(Kussmaul's breathing)** and hiccoughs
- *Cardiovascular (CVS):* Hypertension, pericarditis, cardiomegaly, heart failure, uremic pericardial rub should be looked for.
- *Gastrointestinal tract (GIT):* Nausea, vomiting, diarrhea, furred tongue
- *Skin:* Scratch marks, bruising, uremic frost, calcium deposits in skin particularly in patients on dialysis, dry skin, hyperpigmentation, tetany, nails show **Lindsay's "half-and-half" nails**, where proximal part is whitish and distal part is brownish red
- *Eyes:* Periorbital edema, band keratopathy, retinopathy (HT, DM)
- *Kidneys:* Bilateral or unilateral palpable. Polycystic kidneys, hydronephrosis, hypernephroma
- Stunted growth in children
- Bone tenderness (hyperparathyroidism), bone pain.

Stages of Chronic Renal Failure

Stage I: Normal GFR more than 90 mL/min.
Stage II: GFR 60–90 mL/min.
Stage III: GFR 30–59 mL/min.
Stage IV: GFR 15–29 mL/min.
Stage V: GFR less than 15 mL/min.

Note:

- **Albuminuria is of increasing severity as the stage increases and the GFR decreases**
- **Serum creatinine is widely used as a marker for GFR, which is related to the urine creatinine excretion and inversely to the serum creatinine. Creatinine clearance and GFR are calculated as per formula given below:**

$$\text{Creatinine clearance (mL/minute)} = \frac{(140 - \text{age}) \times \text{lean body weight (kg)}}{\text{Plasma creatinine (mg/dL)} \times 72}$$

For women, multiply the calculated value by 0.85 (*Cockcroft-Gault equation*).

***GFR* (mL/min per 1.73 m^2) = 186.3 x Pcr ($e^{-1.154}$) x age ($e^{-0.203}$) (*MDRD equation*)**

- **In general symptoms of uremia do not occur unless GFR is less than 15 mL/min.**

INVESTIGATIONS

- *Blood:* Serum creatinine, blood urea
- *Ultrasonography (USG) of abdomen:* To assess size of kidney. In uremia, usually both

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kidneys are small and contracted (< 8 cm in length). If the kidneys are enlarged suspect polycystic kidneys, amyloidosis, unilateral or bilateral hydronephrosis, tumors of kidney.

ETIOLOGICAL DIAGNOSIS

The cause of uremia can be ascertained from the findings seen on clinical examinations.

- Skin rash (drug hypersensitivity)
- *Splinter hemorrhages, splenomegaly:* Suspect infective endocarditis
- *Presence of hemoptysis:* Suspect vasculitis
- *Jaundice:* Consider **Weil's disease**
- *Tophi:* Consider gout
- Nail patella syndrome
- *Distended bladder:* Bladder neck obstruction
- *Bruit over abdomen:* Rule out renal artery stenosis.

Note: Look for arteriovenous fistula used for hemodialysis.

MANAGEMENT

- In obstructive uropathy, treat the obstruction, e.g. stone, enlarged prostate, stricture urethra and bladder neck obstruction should be surgically corrected
- Erythropoietin for anemia
- Appropriate antihypertensive drug according to the case
- *Diet:* Important is the protein which is given as per the GFR
  - *Normal GFR:* No restriction
  - *Low GFR:* Restrict to 0.6 g/kg/day with 30% of protein of high biological value
- *Potassium:* Low potassium. Potassium rich foods and drinks, e.g. coconut water, fruit juices to be avoided
- *Sodium:* It should be restricted to 4–6 g according to edema and blood pressure
- To treat acidosis (serum bicarbonate < 20 mmol/L) give sodium bicarbonate IV
- *Water:* Fluids are restricted < 1 L/day) so long as edema is present
- Diuretics (furosemide) up to 120–250 mg/day IV depending upon edema
- For treatment of renal bone disease give calcium carbonate (300–1200 mg thrice a day) and alfacalcidol (0.25–1 mcg/day) orally
- Hiccups are treated with chlorpromazine in adjusted dosages according to serum creatinine value.
- Give statin and aspirin and no tobacco/smoking to reduce CVS complications
- Hemodialysis, if medical treatment fails
- Renal transplant.

VIVA VOCE

Q1. Explain azotemia and uremia.

- In azotemia there is an elevated level of urea and creatinine but no symptoms. GFR about 20–50% of normal
- In uremia there is worsening of renal function with symptoms (GFR < 20% of normal).

Q2. When will you start hemodialysis?

- Creatinine concentration more than 500 µmol/L
- Bicarbonate concentration less than 12 mmol/L
- Potassium levels more than 7 mmol/L.

Q3. Describe bone changes in chronic renal failure.

- Osteomalacia (renal rickets)
- *Osteosclerosis:* Enhanced density of bones in upper and lower margins of vertebrae called **rugger-jersey spine**
- Osteitis fibrosa cystica (secondary hyperparathyroidism)
  Subperiosteal erosions particularly in skull **(pepper pot skull)**, **(Fig. 4.2)** phalanges, distal end of clavicles, long bones.

Q4. Describe beneficial effects of ACE inhibitors in renal disease.

They are beneficial due to antihypertensive and antiproteinuria effects. Trials show that ACE inhibitors result in less renal failure than other antihypertensive drugs in renal disease.

Q5. Describe Bricker's trade-off hypothesis.

In early stages of renal failure, the kidney fails to excrete phosphorus resulting in rise in phosphorus levels. To correct the rise in serum phosphorus and calcium, parathormone

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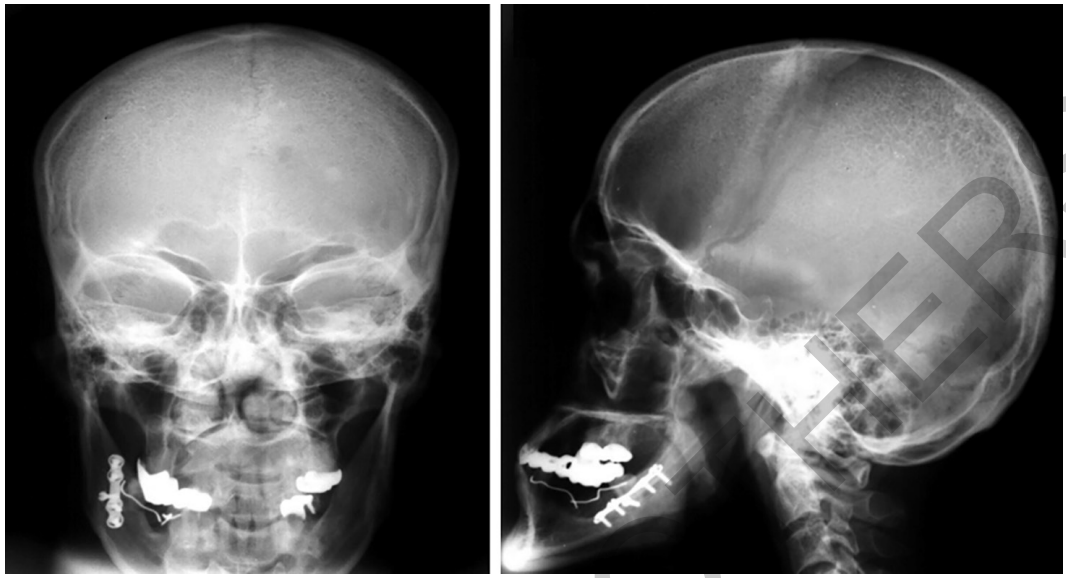

Fig. 4.2 X-ray skull (anteroposterior and lateral view) showing subperiosteal erosion in skull (pepper pot skull)

hormone is released; which results in bone disease **(osteitis fibrosa cystica)**. That is how secondary hyperparathyroidism sets in renal failure.

Q6. Which drugs are contraindicated in uremia?

Aminoglycosides, potassium salts, tetracyclines.

Q7. How will you assess efficacy of dialysis?

Efficacy of dialysis is better assessed by blood urea nitrogen (BUN). Serum creatinine is not a good measure of efficacy. Each sitting of dialysis is adjusted to achieve a urea reduction ratio (URR) of at least 65% with treatment duration lasting for 3–4 hours.

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CASE 3: POLYCYSTIC KIDNEY DISEASE

INTRODUCTION

It is a genetic disorder in which along with polycystic kidneys, there is associated polycystic liver disease. There are two types:

1. **Adult polycystic kidneys:** It is inherited as an autosomal dominant trait with nearly 100% penetrance and equal gender prevalence. The cysts, which appear in infancy, are small, later they increase in size as age advances and kidneys enlarge asymmetrically.
2. **Infantile polycystic kidneys:** It is rare and inherited as autosomal recessive. It is fatal in first year of life.

SALIENT CLINICAL FEATURES OF ADULT POLYCYSTIC KIDNEYS

It usually remains without symptoms. It presents with insidious onset with hypertension in a young adult. Hypertension may be the only feature for a long period with no other symptoms. Other features related to enlarged kidneys like heaviness, pain, vague discomfort in loins appear late. Some patients may present with hematuria, burning in urine due to urinary tract infection or even renal failure. Kidney stones are twice as prevalent as the general population.

Physical examination reveals palpable and tender kidneys on both sides and high blood pressure. In 30% of cases, enlarged liver due to cysts is palpable. One should look for presence of berry aneurysms in cerebral blood vessels, which in few cases result in subarachnoid hemorrhage. Overall prognosis is fair but as time progresses, renal failure sets in.

Note: Polycystic kidney disease is the most common genetic disease. It affects approximately 12.5 million people worldwide.

INVESTIGATIONS

- Routine urine examination
- *Renal function tests:* Serum creatinine, blood urea, etc.
- Ultrasound of abdomen which confirms the diagnosis **(Fig. 4.3)**
- If USG does not give clear picture, CT scan abdomen is done for confirmation
- Electrocardiogram (ECG) for left ventricular hypertrophy.

MANAGEMENT

- Treat hypertension
- If urinary tract infection is present, treat according to culture and sensitivity report
- Treat renal failure when it sets in.

Note: Since gene defect is known, one should screen first-degree relatives of patients at young age with USG abdomen and counseling is done before they start their family.

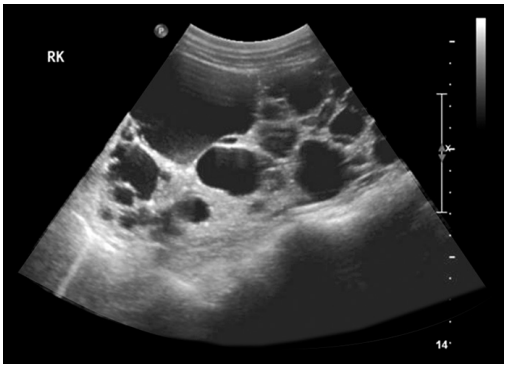

Fig. 4.3 Ultrasonography (USG) of abdomen showing polycystic kidney

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VIVA VOCE

Q1. Describe the organs in which cysts are seen in polycystic kidney disease.

Liver in 30% of cases, spleen, pancreas, lungs, ovaries, testes, thyroid and uterus. Women may have massive cystic enlargement of the liver due to estrogens promoting growth of cysts. Exogenous estrogens are also risk factors; hence oral contraceptives and hormone replacing therapy (HRT) should be avoided.

Q2. Describe neurological manifestations of polycystic kidney disease.

- Subarachnoid hemorrhage from rupture of berry aneurysm causing neurological defect or death in 90% of cases
- About 80% have asymptomatic berry aneurysms
- In some, aneurysms present as a *"sentinel"* or *"thunderclap" headache.*

Q3. Name CVS manifestations in polycystic kidney disease.

- Mitral valve prolapse in about 25%
- Other lesions reported are mitral, aortic and tricuspid valve regurgitation.

Q4. Describe causes of death in polycystic kidney disease.

One-third of adult patients die from kidney failure, another one-third from hypertension and its complications such as heart disease, intracerebral hemorrhage and rupture of berry aneurysm, the remaining 1/3rd die from unrelated disease.

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CASE 4: UNILATERAL PALPABLE KIDNEY

CAUSES OF PALPABLE KIDNEY ON ONE SIDE

- Hydronephrosis
- Hypernephroma (Renal carcinoma)
- Large cyst in kidney
- Hypertrophy of the solitary functioning kidney when there is congenital absence of kidney or when patient has donated one kidney or patient has had nephrectomy on one side due to kidney disease or trauma.

SALIENT CLINICAL FEATURES

- If unilateral palpable kidney is on left side, one should differentiate the kidney from splenomegaly
- In splenomegaly, one cannot insinuate fingers between the mass and left costal margin whereas in kidney mass one is able to insinuate one's fingers between the mass and costal margin
- In splenic mass, there will be dull note anteriorly whereas in front of kidney mass, there will be band of resonance due to colon lying in front
- One may feel notches in a splenomegaly
- Kidney is ballotable bimanually.

INVESTIGATIONS

- Renal function tests to assess whether unilateral kidney is adequate to look after renal functions
- Ultrasonography of abdomen to find the cause, e.g. polycystic kidney, hypernephroma, large cyst can be diagnosed
- Intravenous pyelogram (IVP)—this will help to diagnosis hydronephrosis.

MANAGEMENT

- Medical management, if renal functions are impaired
- Surgical—if it is due to renal carcinoma, surgical removal is done.

VIVA VOCE

Q1. Describe the changes in other kidney when one kidney has been donated.

In large majority of cases, long-term kidney functions are normal. However, these patients have greater chances of developing proteinuria, glomerular disease and renal failure. Therefore it is mandatory to monitor these patients periodically. Majority of donors who carry on doctor's advice and have periodic check-up, maintain normal GFR, normal albumin excretion and lead a normal life. In only few patients renal failure occurs.

Q2. Describe short-term and immediate risks of donation.

- Short-term risk is small and mortality is 0.03%, which is almost same as in other operations done under general anesthesia
- Risk of infection is minor
- Risk of bleeding during or after operation is again minimal
- If kidney is removed laparoscopically, short-term risks are minimal and patient is discharged within few days but after a routine surgical removal, patient may take 4–5 weeks to return to work.

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CASE 5: ACUTE RENAL FAILURE

INTRODUCTION

There is sudden but usually reversible loss of renal function. If patient has previous reports of kidney functions, one should compare. There is rapid decline in GFR (hours to days) and retention of nitrogenous waste products. There is rapid rise in blood urea, serum creatinine and potassium. Some patients may develop oliguria.

CAUSES OF ACUTE RENAL FAILURE

Prerenal

Basically there is decreased effective extracellular volume, which is due to following factors:

- Fluid loss—vomiting, diarrhea, hemorrhage, burns
- Low cardiac output as a result of cardiogenic shock, heart failure due to myocardial infarction, arrhythmias, myocarditis, valvular disorders
- Peripheral vasodilatation—anaphylactic shock, hypotension, septicemia
- Fluid redistribution—nephrotic syndrome, liver disease, intestinal obstruction
- Renal vasoconstriction—hepatorenal syndrome, hypercalcemia, use of alpha-adrenergics
- Efferent arterial vasodilation—ACE inhibitors
- Postoperative and postpartum hemorrhage.

Intrarenal Failure

- Acute tubular necrosis (ATN)
- Glomerulopathy
- Vasculitis
- Interstitial nephritis
- Toxic effect of drugs—NSAIDs, aminoglycosides
- Contrast dye
- Rhabdomyolysis.

Postrenal Failure

- Obstructive uropathy
- Intrarenal—renal stones, papillary necrosis, crystals
- Extrarenal—enlarged prostate, bladder neck obstruction, urethral obstruction, retroperitoneal fibrosis, bladder or pelvic tumors.

One can calculate renal failure index as follows:

$$\textbf{Renal failure index} = \frac{\text{Urine Na} \times \text{serum creatinine}}{\text{Urine creatinine}}$$

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CASE 6: ACUTE TUBULAR NECROSIS

INTRODUCTION

- It is either due to ischemia or toxins (chemical or bacterial) or may be combination. Drugs which are nephrotoxic are aminoglycoside antibiotics such as gentamycin, cisplatin, amphotericin B
- Tubular obstruction occurs due to dead tubular cells obstructing the tubular lumen. In addition to tubular cell damage, there is alteration in renal microcirculation
- Recovery of ATN occurs because tubular cells can regenerate and reform the basement membrane. Renal function usually returns to normal, if the patient is managed properly during regeneration phase. During recovery, there is a diuretic phase when urine output increases rapidly and remains high for several days before returning to normal.

SALIENT CLINICAL FEATURES

- *Prerenal failure:* These patients usually have hypotension, volume contraction and depressed cardiac function. The diagnosis is confirmed when after volume repletion, there is improvement in renal perfusion and improvement in cardiac function.
- In postrenal failure patients, look for enlarged prostate, distended bladder, pelvic tumors, hydronephrosis.
- Acute renal failure (ARF) due to intrarenal disease is confirmed by kidney biopsy. Presence of heavy proteinuria and RBC casts suggests glomerulonephritis. Interstitial nephritis would present with pyrexia, skin rash, and pus in urine and eosinophils in the urinary sediments.

  Ischemic ARF has three phases:
  1. *Initiation phase:* It is the initial period of renal hypoperfusion during which period ischemic injury is evolving.
  2. *Maintenance phase:* It lasts 1–2 weeks during which period renal injury is established with oliguria and retention of nitrogenous products.
  3. *Recovery phase*: During this period there is repair and regeneration leading to return of GFR to normal. At the same time there is diuresis. Later in recovery phase urine output improves and returns to normal. Also BUN and creatinine start decreasing to return to normal.

Course

Acute tubular necrosis starts with oliguria within 24 hours of the insult and may even result in anuria. Usually oliguria continues for 1–2 weeks. But if oliguria goes beyond 2 weeks, consider another diagnosis. The daily rise in BUN and creatinine is around 10–20 mg/dL of urea and 0.5–1 mg/dL of creatinine.

COMPLICATIONS

- Hypertension
- Congestive cardiac failure (CCF)
- Sodium and water overload
- Potassium is raised due to decreased excretion
- Metabolic acidosis due to retention of acids
- Hypocalcemia
- Hypomagnesemia
- Hypophosphatemia
- Hyperuricemia
- Gastrointestinal (GI) bleeding
- Low Hb (anemia)
- Pericarditis
- Paralytic ileus.

MANAGEMENT

- Find out prerenal and postrenal causes and correct them
- See if there is evidence of ischemic or nephrotoxic injury and correct them
- If patient is volume overloaded in ATN, give IV furosemide. But in those who are

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not volume overloaded, administer normal saline 500–1000 mL over 1/2–1 hour
- Insert urinary catheter and keep a strict urine output and intake chart
- Record daily weight
- Administer IV fluids—500 mL + previous day's losses
- If hyperkalemia and acidosis are present, treat them
- Dialysis is considered when conservative treatment fails and when there is volume overload or pericarditis or GI bleeding, severe hyperkalemia and severe acidosis
- When there is pigment induced kidney injury during hemolysis or rhabdomyolysis, it should be treated with alkalinization of urine
- One may consider pushing furosemide infusion (20 mg per hour), if urine output improves after first bolus dose of furosemide
- In pigment induced renal injury, administer 2–3 ampules of $NaHCO_3$ in 1 liter of 5% dextrose IV to alkalinize the urine
- Prednisolone 60 mg orally to start with is indicated to hasten recovery in drug induced acute interstitial nephritis, prednisolone is then tapered slowly according to the response of the patient.

Indications for urgent dialysis:
- Serum K greater than 6.0 mEq/L
- Severe acidosis—pH greater than 7.2
- Daily elevation of blood urea greater than 30 mg/dL
- Serum creatinine greater than 7 mg/dL
- CO_2 combining power less than 13 mEq/L
- Pulmonary edema not responding to diuresis
- Pericarditis, cardiac tamponade
- Rapid renal failure.

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Clinical Cases & Pearls in MEDICINE

This book Clinical Cases and Pearls in Medicine has been written with an aim to facilitate students preparing for medicine practical at final MBBS, MD, DNB, FCPS and MRCP examinations. It is a known fact that to clear medicine practical is tougher than to clear medicine theory. While preparing for medicine practical, students have to revise clinical methods as well as textbook of medicine which is quite laborious. It is quite likely that students may miss important practical points about the clinical cases and viva voce, when they read textbook of medicine.

Keeping these points in view the author has written this book in which 203 clinical cases and 101 pearls in medicine are described. Each clinical case covers all important aspects of the case and relevant symptoms which the student has to elicit in history and pertinent physical findings which the student has to detect during clinical examination, relevant investigations for confirmation of diagnosis, differential diagnosis, wherever necessary, prognosis and management. Large numbers of cases are long cases and some are short and spot cases. Clinical cases are supplemented with 309 illustrations including clinical photographs. Majority of cases are followed by viva voce with questions and answers. In short, clinical cases are capsuled from a mammoth of clinical facts.

There are 101 pearls which are nothing but key messages of clinical medicine. They provide wealth of information for the student to face table viva voce. The cases are written in points and in simple language which student will find it easy to understand and grasp.

From his vast experience (nearly 6 decades) of teaching clinical medicine not only in medical colleges and private teaching hospitals in India but also at 10 foreign universities including Hammersmith Hospital and St Mary's Hospital, London, where he taught MRCP students as a Locum Registrar. Also, from his experience as an Examiner for final MBBS, MD at 18 Indian Universities and as a DNB Examiner several times, he feels that this book will more than suffice for purposes of practical at final MBBS and postgraduate level.

The students will not have to read any other book before appearing for practical. He believes that the book should prove to be a key companion to students appearing for medicine practical.

The book should also prove useful as a ready-reckoner for family physicians and even consultants in medicine and allied branches.

GS Sainani had scholarly career, given double promotion, topper, best outgoing student, passed matric with distinctions in 1947 (Bombay University) and passed I.Sc (Nagpur University) with distinction and received an award from Pandit Jawaharlal Nehru in 1949.

His medical career (MBBS, MD) was brilliant and he was winner of awards and merit scholarships from State and Center. He joined as full-time lecturer, promoted through PSC superceding his two teachers and appointed as Professor and Head, Department of Medicine at 32 years of age.

He was invited as Visiting Professor of Cardiology at Chicago Medical School and Mt Sinai Hospital, where along with training in cardiology, he did path-breaking research, presented and published 10 papers and authored a Primer of Cardiac Diagnosis (USA publication in English and German).

He is now Senior Consultant and Director Research Department of Medicine, Jaslok Hospital; Emeritus Professor (for life) at Grant Medical College, Mumbai; Emeritus Professor, National Academy of Medical Sciences, New Delhi; Emeritus International Advisor; Royal College of Physicians (Lond); Coordinator for Global Research on Geriatrics. He is Past President of 16 national medical associations and he has been Visiting Professor to 10 foreign and several Indian universities.

He has been the Director, All India Heart Foundation (16 years), the Dean, Faculty of Medicine (Pune University) and Dean, Indian College of Physicians; the Chairman, World Congress of Clinical Nutrition; the Chairman, International Congress on Geriatrics, the Chairman, International Congress on Cardiovascular Advances; Member, Institute body of AIIMS, Member of ICMR and MCI expert group and member academic committee AFMC. His Ph.D on Garlic and Onion in Atherosclerosis is widely quoted including Scientific American. He is the first D.Sc (Medicine) from Pune University on the basis of his 30 years research approved by 3 referees.

He is recipient of 30 awards including Padma Shri, 5 gifted teacher awards and 8 life-time achievement awards including 2 by the President of India and has delivered 26 orations including Dr BC Roy. Dr Sainani yearly oration is instituted by 3 national associations and 1 International World Congress on Clinical, Preventive Cardiology and Imaging (WCCPCI), 21 Fellowships have been conferred on him including Hon FRACP, Hon FISC and Hon D.Lit. Decorated with Hon. Brigadier Rank by President of India.

He has been invited as guest speaker/chairman by national and international conferences over 500 occasions.

He has 555 publications, Author, and an Editor-in-Chief of 8 medical books including API Textbook of Medicine (5th and 6th edition), Editor of 6 national journals of which two journals—JAM (19 years) and JAPI (7 years), made him All India Fame Editor. Even WHO selected JAPI to represent India and invited Dr Sainani to participate in International Congress of Editors of Medical Journals in which 30 countries participated.

Shelving Recommendation
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