(سنريهم آياتنا في الآفاق وفي أنفسهم حتى
يتبين لهم أنه الحق أولم يكف بربك أنه على كل
شيء شهيد)

صدق الله العظيم

Nephrology

Acute kidney injury (AKI)

AKI is an acute but potentially reversible decline in glomerular filtration rate, with or without oliguria and associated with retention of nitrogenous waste products

Pre-renal

Circulatory failure ('shock') due to:

Hypovolaemia: **haemorrhage, burns, diarrhoea, sepsis**

Cardiogenic: **congenital heart disease (hypoplastic left heart), myocarditis, sepsis**

Intrinsic

Acute tubular necrosis: circulatory failure, toxins (myoglobinuria, gentamicin), acute nephritis

Vascular pathology: haemolytic uraemic syndrome, renal venous thrombosis or asphyxia

Tubulo-interstitial nephritis: furosemide, NSAIDs.

Post-renal

Congenital anomalies: posterior urethral valves, bladder ureterocoele prolapse.

Obstruction: tumour, stones, or blocked urethral catheter

Neuropathic bladder

Clinical evaluation

Diagnosis usually rests on documentation of elevated plasma creatinine levels (×2–3 of normal)

oliguria, defined as a urine output of <0.5 mL/kg per hour (<1 ml/kg per hour in a neonate).

Non-oliguric renal failure can occur in drug-induce nephrotoxicity.

Examination includes a careful assessment of circulatory status and fluid balance to identify circulatory failure or hypovolaemia consistent with prerenal failure or fluid overload. Important

signs include:

General: dehydration (hypovolaemia) or fluid overload (GN).

Anaemia, jaundice or petechiae in haemolytic uraemic syndrome

CVS: **shock: tachycardia, hypotension, prolonged CRT, fluid overload: hypertension and gallop rhythm**

Respiratory system: **crackles in pulmonary oedema**

Abdomen: **palpable bladder or kidneys in obstruction**.

investigations

Urine tests

Dipstick test for blood and protein and microscopy (casts).

Urine osmolality and Na+ concentration provides useful indicators of whether renal failure is prerenal or intrinsic.

Prerenal failure: urine osmolality >500 mOsm/kg, urine Na+ <10 mmol/L due to avid Na + and water retention

Acute tubular necrosis: urine osmolality <300 mOsm/kg, urine Na+ >40 mmol/L: dilute, Na+ -rich urine.

The fractional excretion of Na+(FENa) is a useful index of tubular function:

FENa = [(urine Na × plasma Cr)/ (plasma Na × urine Cr)] × 100%

FENa is <1% in prerenal failure (acute nephritis, obstruction)

>1% in acute tubular necrosis.

Blood tests

Changes in plasma creatinine (Cr) reflect changes in GFR, a doubling of plasma Cr corresponding to a 50% reduction in GFR

Hyperkalaemia and hypocalcaemia are common

FBC and film: **leukopaenia and thrombocytopaenia suggest SLE or TTP.**

Microangiopathic anaemia suggests TTP or HUS.

Imaging and renal biopsy

Renal ultrasound **identifi es obstructive uropathy, intrinsic renal disease, stones, renal venous thrombosis**

Renal biopsy **is indicated in prolonged, unexplained acute renal failure.**

Treatment

Fluid management: in prerenal failure hypovolaemia should be corrected with a rapid fluid infusion (fluid challenge)

. In intrinsic renal failure, fluid intake is restricted to insensible loses and urine output.

In a fluid overloaded, oliguric patient administration of a loop diuretic (furosemide) is indicated to prevent or treat pulmonary oedema.

A urinary catheter is placed to allow accurate measurement of urine output (and relief of any lower tract obstruction

Electrolytes:

plasma K+ is monitored and hyperkalaemia treated. Hyponatraemia secondary to water excess requires fluid restriction

hypocalcaemia is treated with calcium supplementation.

Renal replacement therapy (dialysis):

indications for dialysis include failure of medical treatment to alleviate severe fluid overload

hyperkalaemia

symptomatic uraemia, or metabolic acidosis

to remove dialysable intoxicants (low molecular weight and not highly protein bound: gentamicin, ethylene glycol).

Table 6.1. Staging of chronic kidney disease		
Stage of CKD	**Description**	**GFR mL/min/1.73 m^2**
0	Risk factors: diabetes, BP+	>90
1	Renal damage (proteinuria) normal GFR	>90
2	Mild ↓GFR	60–89
3	Moderate ↓GFR	30–59
4	Severe GFR	15–29
5	ESRF	<15

Aetiology and pathogenesis

***Infants <2 years*: structural abnormalities including renal dysplasia and obstructive uropathy account for 50% of cases**

***Children 2–5 years:* in addition to renal dysplasia and obstructive uropathy, neonatal vascular accidents and haemolytic uraemic syndrome**

Older children and adolescents: glomerular disease (FSGS, **crescentic GN, lupus nephritis), reflux nephropathy and genetic disorders (Alport syndrome).**

Less common causes include rare genetic diseases such as cystic kidney disease, congenital nephrotic syndrome, cystinosis, hyperoxaluria, drug-induced nephrotoxicity, malignancy, tubular and interstitial disorders

***Failure to thrive**:* anorexia and inadequate calorie intake

Short stature and delayed puberty: multifactorial aetiology including poor nutrition

Anaemia: caused by decreased erythropoietin production, iron and folate deficiency, reduced red cell survival (uraemia)

***Osteodystrophy**:* diminished hydroxylation by 1-⟨-hydroxylase reduces calcitriol synthesis and phosphate retention causes secondary hyperparathyroidism and bone resorption

***Cardiovascular**:* hypertension mediated by the angiotensin–renin pathway. Pro-atherogenic state with hyperlipidaemia and elevated calcium/phosphate product

Metabolic acidosis: contributes to bone disease, growth failure, and hyperkalaemia

Clinical evaluation

failure to thrive or growth failure

lethargy and malaise

anorexia and vomiting

polyuria,bone pain, or headaches

Clues to aetiology may exist in the past history (perinatal complications, oligohydramnios, recurrent UTI) or family history (renal disease, deafness). Examination may be normal but can reveal causal factors or sequelae of renal impairment such as growth failure, anaemia, stigmata of osteodystrophy, hypertension, palpable bladder or kidneys.

Urine tests

Dipstick testing for proteinuria, haematuria, osmolality, microscopy,and culture.

Blood tests

Renal function: creatinine, U&E, capillary blood gases. Total corrected and ionized calcium, phosphate, alkaline phosphatase, PTH, and vitamin D

FBC, ferritin.

Imaging

Renal ultrasound: identifi es small echogenic kidneys, cystic kidneys, obstructive uropathy

Treatment

Preservation of renal function

The aim is to delay progression to ESRF. Strategies include prevention of recurrent upper UTI, relief of obstruction, control of hypertension and proteinuria.

Treatment of complications

Nutrition: aim to promote growth by optimizing calorie and protein intake.

Gastrostomy or nasogastric feeding may be necessary in infants.

Growth: recombinant human growth hormone (rhGH) may be effective in increasing height velocity and fi nal adult height.

Anaemia: iron and folate defi ciency should be addressed before initiation of erythropoietin therapy.

Renal osteodystrophy: the key is adequate control of plasma phosphate by limiting dietary intake and the use of phosphate binders. Supplementation with 1-alfacalcidol may also be required for secondary hyperparathyroidism.

Hypertension: an ACEi is used if there is proteinuria. Additional agents may be necessary

Thank You