Disorders of Potassium

Hypokalemia:

Hypokalaemia is a common electrolyte disturbance and is defined as existing when serum potassium falls below 3.5 mmol/L.

Causes of hypokalemia include:

1. Reduced intake: Dietary deficiency ,Potassium-free intravenous fluids
2. Redistribution into cells:Alkalosis,Insulin,Catecholamines,β-adrenergic agonists, Hypokalaemic periodic paralysis.
3. Increased urinary excretion: see box
4. Increased GIT loss: see box

14.15 Causes of hypokalaemia

Cause	Other features and comment
Reduced intake Dietary deficiency Potassium-free intravenous fluids	Urine K^+ >20–30 mmol/24 hrs
Redistribution into cells	
Alkalosis Insulin Catecholamines β-adrenergic agonists Hypokalaemic periodic paralysis	Caused by flux of K^+ into cells
Increased urinary excretion Activation of mineralocorticoid receptor:	Urine K^+ >20–30 mmol/24 hrs
Conn's syndrome Cushing's syndrome Glucocorticoid excess Carbenoxelone/liquorice	Associated with hypertension and alkalosis
Genetic disorders:	
Liddle's syndrome	Associated with hypertension and alkalosis
Bartter's syndrome Gitelman's syndrome	Associated with hypertension, alkalosis and hypomagnesaemia
Renal tubular acidosis Type 1 (distal) Type 2 (proximal)	Inherited and acquired forms; associated with high serum chloride. Type 2 associated with glycosuria, aminoaciduria and phosphaturia
Acetazolamide	Associated with acidosis
Diuresis: Loop diuretics Thiazides Recovery from acute tubular necrosis Recovery from renal obstruction	Increased sodium delivery to distal tubule
Increased gastrointestinal loss	Urine K^+ <20–30 mmol/L
Upper gastrointestinal tract: Vomiting Nasogastric aspiration	Loss of gastric acid Associated with metabolic alkalosis
Lower gastrointestinal tract: Diarrhoea Laxative abuse Villous adenoma Bowel obstruction/fistula Ureterosigmoidostomy	Associated with metabolic acidosis

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Clinical features:

Patients with mild hypokalaemia (plasma K+3.0–3.3 mmol/L) are generally asymptomatic, but more profound reductions in plasma potassium often lead to *muscular weakness and associated tiredness*. Ventricular ectopic beats or more serious arrhythmias may occur and the arrhythmogenic effects of digoxin may be potentiated.

Typical electrocardiogram (ECG) changes occur, affecting the T wave in particular (flattening or even inversion of T-wave).

Functional bowel obstruction may occur due to paralytic ileus. Long-standing hypokalaemia may cause renal tubular damage (hypokalaemic nephropathy) and can interfere with the tubular response to vasopressin (acquired nephrogenic diabetes insipidus), resulting in polyuria and polydipsia.

Investigations:

Measurement of plasma electrolytes, bicarbonate, urine potassium and sometimes of plasma calcium and magnesium is usually sufficient to establish the diagnosis.

Measurement of urinary potassium may be helpful; if the kidney is the route of potassium loss, the urine potassium is high (> 30 mmol/24 hrs), whereas if potassium is being lost through the gastrointestinal tract, the kidney retains potassium, resulting in a lower urinary potassium (generally < 20 mmol/24 hrs).

Management:

Treatment of hypokalaemia involves first determining the cause and correcting this where possible. If the problem is mainly one of redistribution of potassium into cells, reversal of the process responsible may be sufficient to restore plasma potassium without providing supplements. In most cases, however, some form of potassium replacement will be required. This can generally be achieved with slow-release potassium chloride tablets, but in more acute circumstances intravenous potassium chloride may be necessary.

The rate of administration depends on the severity of hypokalaemia and the presence of cardiac or neuromuscular complications, but should generally not exceed 10 mmol of potassium per hour. In patients with severe, life-threatening hypokalaemia, the concentration of potassium in the infused fluid may be increased to 40 mmol/L if a peripheral vein is used, but higher concentrations must be infused into a large ‘central’ vein with continuous cardiac monitoring.

In some circumstances, potassium-sparing diuretics, such as amiloride, can assist in the correction of hypokalaemia, hypomagnesaemia and metabolic alkalosis, especially when renal loss of potassium is the underlying cause.

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Hyperkaliemia:

Hyperkalaemia is a common electrolyte disorder, which is defined as existing when serum K+ is > 5 mmol/L.

14.16 Causes of hyperkalaemia

Cause	Other features and comment
Artefactual	
Haemolysis during venepuncture Haemolysis in vitro Thrombocytosis/leucocytosis	Release of intracellular K^+ during sample collection, transit or clotting
Increased intake	
Dietary potassium Potassium-containing intravenous fluids	
Redistribution from cells	
Acidosis Insulin deficiency Severe hyperglycaemia β-adrenergic blockers (β-blockers) Hyperkalaemic periodic paralysis Rhabdomyolysis Severe haemolysis Tumour lysis syndrome	Caused by flux of intracellular K^+ into plasma
Reduced urinary excretion	
Reduced glomerular filtration: Acute kidney injury Chronic kidney disease	Plasma creatinine typically >500 µmol/L (5.67 mg/dL)
Reduced mineralocorticoid receptor activation: Addison's disease Congenital adrenal hyperplasia Isolated aldosterone deficiency	
Angiotensin-converting enzyme (ACE) inhibitors Angiotensin-receptor blockers (ARBs)	ACE inhibitors and ARBs reduce aldosterone levels
Calcineurin inhibitors Spironolactone Eplerenone	All block the mineralocorticoid receptor
Heparin	Heparin inhibits aldosterone production
Inhibitors of renin production: Non-steroidal anti-inflammatory drugs (NSAIDs) β-blockers	
Tubulointerstitial disease: Interstitial nephritis Diabetic nephropathy Obstructive uropathy	
Other:	
Amiloride	Blocks K^+ exchange in distal tubule
Gordon's syndrome	Direct effect on K^+ transport in renal tubule

Clinical features:

Mild to moderate hyperkalaemia (< 6.5 mmol/L) is usually asymptomatic. More severe hyperkalaemia can present with progressive muscular weakness, but sometimes there are no symptoms until cardiac arrest occurs. Peaking of the T wave is an early ECG sign, but widening of the QRS complex presages a dangerous cardiac arrhythmia. However, these characteristic ECG findings are not always present, even in severe hyperkalaemia.

Management:

Treatment of hyperkalaemia depends on its severity and the rate of development, but opinions vary as to what level of serum potassium constitutes severe hyperkalaemia and requires urgent treatment. Patients who have potassium concentrations < 6.5 mmol/L in the absence of neuromuscular symptoms or ECG changes can be treated with a reduction of potassium intake and correction of predisposing factors. However, in acute and/ or severe hyperkalaemia (plasma potassium > 6.5–7.0 mmol/L), more urgent measures must be taken.

14.17 Treatment of severe hyperkalaemia

Objective	Therapy
Stabilise cell membrane potential[1]	IV calcium gluconate (10 mL of 10% solution)
Shift K^+ into cells	Inhaled β_2-adrenoceptor agonist IV glucose (50 mL of 50% solution) and insulin (5 IU Actrapid) IV sodium bicarbonate[2]
Remove K^+ from body	IV furosemide and normal saline[3] Ion-exchange resin (e.g. Resonium) orally or rectally Dialysis

[1]If severe hyperkalaemia (K^+ typically >6.5 mmol/L). [2]If acidosis present. [3]If adequate residual renal function.

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