hypokalaemia

general

- Low serum potassium is reported to be the most common electrolyte abnormality in hospitalised patients.
- There are many causes of hypokalaemia as shown in, but drugs and gastrointestinal disease account for a significant proportion.
- Potassium concentration in the blood is also affected by the metabolic status of the patient. In the presence of a metabolic alkalosis, potassium shifts into cells. Hypokalaemia can also contribute to the maintenance of a metabolic alkalosis by enhancing bicarbonate absorption and increasing chloride excretion in the kidney.
- Hypokalaemia is defined as a serum potassium=3.5 mmol/l.. It may be classified as mild (K 3.0-3.5 mmol/l), moderate (K 2.5-3.0 mmol/l) or severe (K < 2.5 mmol/l) and symptoms are more likely with increasing severity.

potassium homeostasis

- Potassium (K) is the most abundant cation in the body.
- Under normal circumstances, only 2% of total body potassium stores are found in the extracellular space and serum potassium concentration is tightly regulated between 3.5 and 5.0 mmol/l.
- Potassium homeostasis is largely regulated by the kidney accounting for excretion of 90% of daily potassium loss. Therefore patients with renal failure, acute or chronic, who have impaired regulatory mechanisms are prone to hyperkalaemia.
- Patients with normal renal function eliminate only 5-10% of their daily potassium load through the gut. However, in patients with end-stage renal failure (ESRF), gut elimination is increased and accounts for up to 25% of daily potassium elimination.

causes

Increase potassium loss
- Drugs—diuretics, laxative abuse, liquorice, steroids
- GI losses—diarrhoea, vomiting, ileostomy, intestinal fistula, villous adenoma
- Renal—renal tubular disorders, Bartter's syndrome, Liddle's syndrome, Gitelman's syndrome, nephrogenic diabetes insipidous
- Endocrine—hyperaldosteronism, Cushing's syndrome, Conn's syndrome
- Dialysis—-haemodialysis on low potassium dialysate, peritoneal dialysis

Transcellular shift
- Insulin/glucose therapy
- Beta-adrenergic stimulation—e.g. salbutamol
- Alkalosis
- Hypokalaemic periodic paralysis

Decreased potassium intake
- Poor dietary intake (less than 1 g/day)

Magnesium depletion (increases renal potassium loss)
- Poor dietary intake
- Increased magnesium loss

symptoms

- Patients with mild hypokalaemia usually have no symptoms.
- As serum potassium level falls further, the nerves and muscles are predominantly affected causing fatigue, weakness, leg cramps, and constipation. In severe cases, rhabdomyolysis, ascending paralysis and respiratory difficulties may occur.
- The probability of symptoms appears to correlate with the presence of pre-existing heart disease (ischaemia, heart failure, left ventricular hypertrophy), and the rapidity of the onset of hypokalaemia.

ECG changes

- There are usually no ECG changes in patients with mild hypokalaemia, but these may become evident in moderate to severe hypokalaemia including the presence of U waves, T wave flattening, or ST segment changes.

arrhythmias associated with hypokalaemia

General
- Severe hypokalaemia predisposes to arrhythmias and cardiac arrest. In patients treated with digoxin, hypokalaemia of any severity can increase the incidence of arrhythmias. Patients with established digoxin toxicity are particularly at risk.

(i) Ventricular tachycardia/fibrillation.
- Hypokalaemia can predispose to ventricular tachycardia or ventricular fibrillation.
- This risk is particularly high following acute myocardial infarction and maintaining the serum potassium above 3.9 mmol/l may reduce the risk of early VF. The arrhythmia may not respond to electrical or chemical cardioversion until the serum potassium is corrected.

(ii) Long QT syndrome and torsade de pointes.
- The long QT syndrome, which may be inherited or acquired, is caused by malfunction of the ion channels responsible for ventricular repolarisation.
- Potassium and/or magnesium depletion are the main metabolic disorders associated with channel malfunction and hence predispose to arrhythmias.
- The mainstay of treatment is the correction of hypokalaemia and administration of magnesium sulphate.

(iii) Patients taking anti-arrhythmic drugs.
- Hypokalaemia may also interfere with the beneficial effects of anti-arrhythmic drugs rendering the patients susceptible to a recurrence of the underlying arrhythmia. In addition, hypokalaemia can compound the effects of Class III antiarrhythmic agents such as sotalol predisposing to arrhythmias.

Arrhythmogenic effects	ECG changes	Arrythmia
Prolongation of rapid repolarization	U waves	Atrial and ventricular ectopy
Hyperpolarization of RMP	ST segment and T-wave changes	Atrial and ventricular
Increased pacemaker activity in Purkinje and ventricular fibres		tachyarrhythmias

treatment of hypokalaemia

- The treatment approach depends on the severity of hypokalaemia and the presence of symptoms and ECG abnormalities.
- As a guide to the deficit in total body potassium, serum potassium decreases by 0.3 mmol/l on average for every 100 mmol reduction in total body potassium stores, but this is variable depending on body mass. Hence, the deficit can be considerable in moderate-severe hypokalaemia
- Many patients who are potassium deficient are also deficient in magnesium. Magnesium is important for potassium uptake and for the maintenance of intracellular potassium levels, particularly in the myocardium. Combined deficiency may potentiate the risk of cardiac arrhythmias. Repletion of magnesium stores will facilitate more rapid correction of hypokalaemia and is recommended in severe cases of hypokalaemia

Life-threatening arrhythmias
- In an emergency such as an arrhythmia, intravenous potassium is required, but the rate of correction of serum potassium causes uncertainty. The maximum recommended intravenous dose of potassium is 20 mmol/h, but more rapid administration (initial infusion of 2 mmol/min for 10 min, followed by 10 mmol over 5-10 min) is indicated for unstable arrhythmias when cardiac arrest is imminent.
- Rapid bolus injection of potassium should be avoided in all circumstances as this may precipitate cardiac arrest.
- Magnesium should be administrated early after initiating potassium replacement, even before the serum magnesium level is known.

Cardiac arrest
- Cardiac arrest may occur in patients known to be hypokalaemic.
- Alternatively, hypokalaemia may only be discovered after resuscitation is underway. Although the metabolic status after cardiac arrest usually favours an increase in serum potassium, total body potassium remains low.
- Prompt correction of hypokalaemia may not only render defibrillation more successful, but may reduce the incidence of further arrhythmias in the post-arrest period as the metabolic status of the patient improves and potassium shifts back into cells.

emergency management