**Core Competency Theme 10: Nutrition**

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|  | **Basic definition, symptoms and management** |
| **Hypernatraemia** | Hypernatraemia is defined as plasma sodium greater than 146 mmol/L. The clinical significance of hypernatraemia depends on its severity, speed of onset and underlying cause. It is usually due to a deficiency of water, rather than an excess of sodium.  Symptoms are primarily neurological and reflect changes in brain volume (shrinkage). They range in severity and include:  • Headache  • Confusion  • Nausea and vomiting  • Lethargy  • Irritability  • Seizures  • Nystagmus  • Myoclonic jerks  • Loss of consciousness  • Coma  There are three common causes for hypernatraemia:   * Low fluid intake * Diabetes insipidus (DI) * Hyperosmolar hyperglycaemic state (HHS), previously called Hyperosmolar non-ketotic state (“HONK”). Oral fluids should be encouraged if able; alternatively fluids should be administered via NG, PEG or intravenously. An accurate fluid balance chart maintained. |
| **Hyponatremia** | Hyponatraemia can be defined as plasma sodium below 133 mmol/L. The clinical significance of hyponatraemia depends on its severity, its speed of onset and its underlying cause. It is usually due to an excess of water, not a deficiency of sodium.  Symptoms are primarily neurological and reflect changes in brain volume. They  range in severity and include:   * Headache * Confusion * Nausea and vomiting * Lethargy * Irritability * Seizures * Loss of consciousness * Coma   Stopping hyponatraemic medications can help to confirm the cause of hyponatraemia as well as resolve the problem, doctors may also ask for a fluid restriction. An accurate fluid balance should be being maintained on all patients in a hyponatraemic state. |
| **Hypokalaemia** | Hypokalaemia has 3 stages and is classed as mild, moderate and severe. Mild Hypokalaemia is classed when the potassium level drops under 3.5mmol/l, moderate at under 3mmol/l and severe when under 2.5mmol/l.  **Signs and symptoms of Hypokalaemia**  **Cardiovascular**  Bradycardia or tachycardia, hypotension, arrhythmias, cardiac arrest and palpitations.  ECG changes (U waves, T wave flattening, ST segment changes)  **Respiratory**  Respiratory distress and respiratory failure, hypoventilation  **Musculosketal**  Cramps, tetany, reduced deep tendon reflexes, reduced muscle strength  **General**  Peripheral oedema, lethargy, constipation, nausea, vomiting, abdominal cramping and paraesthesia  **Causes of hypokalaemia can include:**   * Inadequate diet including anorexia, malnutrition, bulimia * High dietary sodium intake * Gastrointestinal loss including diarrhoea, vomiting, ileostomy, * intestinal fistulae * Renal loss including dialysis * Urinary loss in congestive heart failure * Hypomagnesaemia * Endocrine disorders, hyperaldosteronism, Cushing’s syndrome * Metabolic acidosis * Medication * Transcellular shift (movement of potassium from serum into cells)   **Treatment**   * Gradual replacement of potassium, via the oral route, is preferred if clinically appropriate * Oral potassium should be taken with plenty of fluid, with or after meals. * A drop in serum potassium level of 1 mmol/L represents a loss of about 100-200 mmol of potassium from body stores. * For serum levels between 2-3 mmol/l, a maximum oral daily dose of 100-200 mmol K+ and for serum levels between 3-4mmol/l, a maximum oral daily dose of 50-100 mmol K+ should be considered. * Use IV route in patients with severe nausea, vomiting, abdominal distress or when the oral or enteral route is not available or will not achieve the required increase of serum potassium within a clinically acceptable time. * Use pre-mixed IV infusions. Sodium Chloride 0.9% is the preferred infusion fluid as Glucose 5% may cause trans-cellular shift of potassium into cells. * Before a patient is transferred any prescriptions for solutions containing potassium infusions must be reviewed to ensure that the treatment can be continued on the receiving ward. * Potassium must be replaced cautiously in patients with renal impairment (risk of hyperkalaemia secondary to impaired potassium excretion). Contact the renal team if patient is on dialysis or has severe renal impairment. |
| **Hyperkalaemia** | Hyperkalaemia is classified as a raised serum potassium level:   * Mild: K+ = 5.5-5.9mmol/L * Moderate: K+ = 6-6.4mmol/L * Severe: K+ \_ 6.5mmol/L   An ECG should be performed on all patients with a raised potassium level, if there are changes on the ECG it is important to commence treatment. Other symptoms include arrhythmias, Muscle weakness and constipation.  The causes of Hyperkalaemia include:   * Pseudohyperkalaemia * Acute kidney injury * Chronic kidney disease * Drugs (potassium supplements, potassium-sparing, ACE inhibitors, NSAIDs,b-blockers, digoxin poisoning) * Acidosis, including diabetic ketoacidosis * Mineralocorticoid deficiency (e.g. Addison’s) * Endogenous (tumour-lysis syndrome, rhabdomyolysis, trauma, burns)   A raised potassium level is normally rang through from pathology and should be handed over to the medical staff as soon as possible. Treatment of Hyperkalaemia can include:  Exclude pseudohyperkalaemia.  Stop all potassium supplements (IV and oral).  Review patient’s medication for possible contributors to hyperkalaemia and or acute renal failure.  Commence low potassium diet or ask for a review of NG/PEG feed by dietitian  Ensure adequate hydration and urine output  If potassium > 6.5mmol/l or ECG changes monitor patient’s cardiac rhythm until it is  stable and potassium level is in range |
| **Hypercalcaemia** | Hypercalcaemia is classified as an adjusted (corrected) calcium > 2.60 mmol/L, it can also be a medical emergency if levels are significantly raised and there is renal impairment.  The most common reasons for hypercalcaemia are hyperparathyroidism, malignancy, chronic renal failure and over treatment with vitamin D. However less common causes include :  Immobilisation  Familial hypocalciuric hypercalcaemia  Granulomatous diseases e.g. sarcoidosis or tuberculosis  Thiazide diuretics  Milk alkali syndrome  Hyperthyroidism  Patients who have developed hypercalcaemia acutely are also much more likely to have symptoms,  these include;   * Polyuria and polydipsia * Nausea and vomiting * Constipation * Weakness and tiredness * Depression and psychiatric disturbances * Drowsiness and confusion * Bone Pain * Renal Stones   Treatment is dependant of the cause of the hypercalcaemia, which can be decided upon clinical picture and from specific blood tests such as:  -PTH  -Urea, creatinine and electrolytes, bicarbonate  -Calcium, albumin, total protein,alkaline phosphatase  -Liver function tests, thyroid function tests  -FBC, ESR, immunoglobulins  A urine sample will also need be sent for:  -Calcium, creatinine, sodium, Bence Jones Protein (BJP)  Doctors may also request the following images to be performed:  Chest X-ray,  Abdominal X-ray,  Renal USS,  Abdo CT depending on differential diagnosis |
| **Hypocalcaemia** | Acute hypocalcaemia can be life threatening, necessitating urgent treatment. In severe cases, intravenous calcium forms the mainstay of initial therapy but it is essential to ascertain the underlying cause and commence specific therapy as early as possible.  Symptoms of hypocalcaemia include:   * Peri-oral and digital paraesthesiae * Positive Trousseau’s and Chvostek’s signs * Tetany and carpopedal spasm * Laryngospasm * ECG changes (prolonged QT interval) and arrhythmia * Seizure   The most commonest causes of hypocalcaemia are:  Following selective parathyroidectomy (hypocalcaemia is usually transient and mild)  • Severe vitamin D deficiency  • Mg2+ deficiency (consider PPI-associated hypomagnesaemia)  • Cytotoxic drug-induced hypocalcaemia  • Pancreatitis, rhabdomyolysis, and large volume blood transfusions  Treatment for hypocalcaemia is through oral replacement therapy such as Sandocal™ 1000, 2 tablets BD (Alternatives include Adcal 3 tablets BD, Cacit 4 tablets BD, or Calcichew Forte 2 tablets BD)    However if the calcium level is under 1.9 mmol/l or the patient is symptomatic it is a medical emergency and IV calcium gluconate should be prescribed and administered, cardiac monitoring should occur throughout administration. |
| **Hypophosphataemia** | The normal phosphate level for an adult is 0.8 – 1.45mmol/L, Hypophosphataemia occurs when serum phosphate levels drop below this, there are 3 stages mild, moderate and severe  Level. Mild hypophosphataemia is when the serum phosphate drops under 0.79mmol/l, moderate at under 0.64mmol/l and severe when under 0.32mmol/l.  Signs of hypophosphataemia do not appear until the serum phosphate is under 0.32mmol/L, which is the severe stage it is only through blood tests that a drop in levels is normally noted.  Symptoms include:   * Generalised muscle weakness, myopathy * Confusion, irritability, hallucinations, somnolence * Paraesthesia * Coma * Hypotension * Respiratory failure * Rhabdomyolysis * Decreased cardiac contractility * Abnormal LFTs * Cardiomyopathy, arrhythmias * Seizures   Causes of hypophosphataemia include:  Refeeding syndrome, recovery from diabetic ketoacidosis, alkalosis, sepsis, drugs, Vitamin D deficiency, diarrhoea, malabsorption, vomiting,  Renal tubular defects, hyperparathyroidism, disorders of vitamin D metabolism, diuretics, renal treatment and chronic alcoholism.  Treatment is normally given orally for patients who are able to eat and drink in the form of Phosphate Sandoz® effervescent tablets, however for patients who are nil by mouth and unable to tolerate NG feeding you can administer  9 mmol of potassium acid phosphate 13.6% in 250 ml sodium chloride 0.9% over 12 hours.  For patients who have severe causes or are symptomatic of Hypophosphataemia the intravenous dose of potassium acid phosphate can be increased to:  18-27 mmol potassium acid phosphate in 500-750 ml sodium chloride 0.9% should be infused over 12-24 hours. |
| **Hypomagnesaemia** | Hypomagnesaemia is a low serum magnesium level however this can also cause secondary hypocalcaemia, and also hypokalaemia and hyponatraemia. Therefore correction of magnesium may aid the correction of other electrolytes. Normal magnesium levels are 0.7 – 1.0mmol/L, drops in this level are classed as mild 0.5-0.7mmol/l or moderate to severe under 0.5mmol/L.  Symptomatic hypomagnesaemia is usually associated with additional electrolyte abnormalities and normally the symptoms only manifest only in the moderate to severe stages.  **Signs, symptoms and consequences of hypomagnesaemia**   * Muscle weakness, ataxia, tremor, seizures, carpopedal spasm * Ventricular arrhythmias, ECG abnormalities such as prolonged QT interval and tachycardia * Depression, psychosis * Vertigo * Hyperinsulinism   Causes of **hypomagnesaemia can include:**   |  |  | | --- | --- | | * Gastrointestinal loss; diarrhoea | * Malabsorption | | * Malnutrition | * Acute pancreatitis | | * Renal tubular reabsorption defects | * Chronic alcoholism | | * Hyperaldosteronism | * Lactation | | * Long-term IV nutrition or fluid therapy | * Re-feeding Syndrome | | * Diabetic ketoacidosis   Treatment  Oral corrective treatment is not always well absorbed by the gastro-intestinal tract however if the patient is not symptomatic is it be first choice of treatment using Magnesium –L-aspartate Magnaspartate® (10mmol/sachet) 1-2 sachets daily.  However if the patient is symptomatic they should be treated intravenously using the following regime: | |   **Day 1**  Magnesium Sulphate 50% 40mmol in 500ml glucose 5% over 12hours (No more than 40mmol daily)  **Days 2-5**  Magnesium Sulphate 50% 20mmol in 500mL glucose 5% over 6hours (No more than 20mmol daily) |