

Electrolyte imbalance essays example

[Business](#), [Management](#)



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Electrolytes are charged particles that have the tendency to generate electrical potentials. Therefore, they are kept under strict check so that they remain within certain limits and don't alter the internal hemodynamically stable milieu. But, during a diseased process or because of self-induction, these electrolytes may either become surplus or becomes alarmingly decreased. Such states can have deleterious often lethal effects on human life.

HYPOKALEMIA:

Signs and Symptoms:

According to Papadakis and McPhee (2015), patients with hypokalemia may present with muscular weakness, fatigue, abnormal muscular cramps,

paralytic ileus leading to constipation, decreased reflexes, hypercapnia, tetany and skeletal muscle lysis.

Etiopathogenesis:

The possible causes of hypokalemia are reduced potassium intake, potassium flux into the cell, urinary loss and extra renal potassium losses. Genetic causes of hypokalemia are hypokalemic periodic paralysis, Bartter syndrome, Gitelman syndrome, Little syndrome, Apparent mineralocorticoid excess and Glucocorticoid-remediable hyperaldosteronism. According to Lewis (2013), there are certain drugs that can cause hypokalemia, which are; diuretics like thiazide, loop and osmotic, amphotericin B, Carbenecillin, Theophylline and Penicillin in larger doses.

Hypokalemia can be caused and inducted by physicians as well. excessive use of furosemide in an emergency setting or administration of glucose based potassium replacing fluids can cause iatrogenic hypokalemia. It can also be caused if acidotic condition is attempted to be corrected rapidly.

Diagnosis:

According to Lewis (2013), potassium is diagnosed in the basis of serum K⁺ measurement, EKG and 24 hours urinary K⁺ excretion. Hypokalemia is diagnosed when serum K⁺ is less than 3.5 mEq/L. on EKG, there is drooping of ST segment, depressed T wave and elevated U wave. In hypokalemia, 24 hour urinary clearance is only less than 15 mEq/L.

Management:

Oral potassium supplement is the safest method and can easily be tolerated. It is essential to remember that dietary potassium is found with phosphate

and not with chlorine therefore, hypokalemia in conjunction with chloride depletion can't be corrected with oral supplementation. For mild to moderate hypokalemia, 20 mEq/day is sufficient 40-100 mEq/day is necessitated over a period of days or weeks to replete the loss. Intravenous infusion of potassium is only warranted in individuals with severe hypokalemia. 40 mEq/L at a rate of 10 mEq/hour should be administered but continuous EKG monitoring should be done simultaneously. Serum potassium levels should be checked after every 3-6 hours.

IMPACT:

CVS: Hypokalemia associated hypertension and hypokalemia induced arrhythmias.

ENDOCRINE: Impaired insulin release and end organ sensitivity to insulin.

MUSCULOSKELETAL: Easy fatigability, myalgias, cramping and muscle weakness.

ACID-BASE: Metabolic alkalosis.

POLYURIA: Increased diuresis leading to polyuria.

CNS: Can lead to or exacerbate hepatic encephalopathy.

HYPERKALEMIA:

Signs and Symptoms:

According to American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care (2005), signs and symptoms of hyperkalemia are weakness, uphill paralysis and respiratory failure.

Etiopathogenesis:

According to American Heart Association (2005), the causes of hyperkalemia are classified into endogenous causes, and exogenous causes. Endogenous causes are chronic renal failure, diabetic ketoacidosis, Gordon's syndrome, post-chemotherapy, rhabdomyolysis, renal tubular acidosis, hemolysis, Addison's disease and hyperkalemic periodic paralysis. Exogenous causes include medications such as potassium sparing diuretics, angiotensin converting enzyme inhibitors (ACEI), non-steroidal anti-inflammatory drugs (NSAIDs), potassium rich supplements, penicillin and its derivatives, succinylcholine, heparin and beta blockers. Other exogenous causes include blood transfusion, potassium rich diet and pseudo hyperkalemia secondary to blood sampling.

The drugs that can lead to hyperkalemia are prescribed invariably because they are very effective in controlling various medical ailments. In emergency resuscitation, use of drugs like digoxin and spironolactone has been reported to cause iatrogenic hyperkalemia. Exogenous potassium overload is also one of the causes.

Diagnosis:

According to Ratini (2013), diagnosing hyperkalemia is challenging and thorough history and meticulous examination are pivotal. Serum and urinary potassium are measured. Plasma potassium of greater than 5 mEq/L suggests strongly the diagnosis of hyperkalemia. EKG is done and it may show positive diagnostic features only in later stages of the process. The typical EKG findings are peaked or tall T waves, broadened QRS complex and biphasic QRS-T complex.

Management:

The cause should be withdrawn and etiologic pattern should be ascertained. Definitive treatment measures include administration of sodium polystyrene complex for the removal of potassium from the gut, diuretics to increase potassium excretion from the body, intravenous glucose with insulin to sequester the potassium intracellularly and dialysis in renally compromised patients.

IMPACTS:

CNS: Muscle weakness, paralysis leading to respiratory failure. Tingling and numbness.

CVS: Bradycardia or heart block and tachyarryhtmias.

GIT: Gastrointestinal distress with abdominal pain and distension.

KIDNEY: Acidemia due to sequestration of acids into the blood due to high cellular potassium

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