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Step by Step® Practical Aspects of Emergency Anesthesia

Arun Kumar Paul

2nd Edition

Step by Step®

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Chapter 3

Fluid, Electrolyte, and Acid-base Disorders

DEHYDRATION

Combined sodium and water deficiency.

Causes

- Severe vomiting, diarrhea, intestinal obstruction, pyloric stenosis, cholera, etc.
- Excessive sweating.
- Chronic heart failure, salt losing nephritis, Addison's disease, diabetes.
- Severe hemorrhage.
- Severe plasma loss (burns).

Clinical and Biochemical Manifestations

- Sunken eyes, wrinkled skin, dry tongue.
- Tachycardia, blood pressure low.
- Veins poorly filled.
- Oliguria.
- Plasma volume reduced.
- Raised level of proteins, packed cell volume, and hemoglobin.
- Blood viscosity increases.
- Plasma sodium level diminishes.

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- Rising blood urea indicates renal failure.
- Circulatory failure.

Treatment

- Identify the cause of volume loss and treat it
- Administer saline solutions
- Blood or colloids solution may also be indicated.

HYPERKALEMIA

It is mostly due to renal insufficiency to excrete potassium.

Causes

- Acute renal failure with anuria.
- Terminal uremia of chronic renal failure.
- Addison's disease, hypoaldosteronism.
- Rapid infusion of potassium salts in cases of hypokalemia.
- After massive blood transfusion, hemolysis.
- Drugs those impair potassium excretion: Triamterene, spironolactone, nonsteroidal anti-inflammatory drugs.
- Drugs those inhibit renin angiotensin aldosterone system, β-blockers, ACE inhibitors.

Clinical Manifestations

- Confusion, apathy, paresthesia.
- Severe muscular weakness.
- Cardiac dysfunction, Peaked T wave, loss of P waves, widening of QRS complex in ECG.
- Ventricular fibrillation.
- Serum potassium above 5.5 mEq/L.

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Note

A pre-existing hyperkalemia may be exacerbated in some conditions:

- Following suxamethonium
- Hypoxia
- Adrenaline either exogenous or endogenous
- Hypercapnia.

Treatment

- Avoid potassium-containing drugs or solutions.
- Correction of hypoxia.
- Oxygenation and proper ventilation.
- Calcium gluconate, IV bicarbonate, glucose and insulin.
- Use of ion-exchange resins.
- Peritoneal dialysis, hemodialysis.
- Dietary potassium restriction and sodium polystyrene sulfonate or loop diuretic to lower potassium subacutely.
- Antidigitalis Tc antibodies in patients with digitalis.

Anesthetic Considerations

- Attempt to lower the plasma potassium before elective surgery.
- Adequate monitoring of the adverse effects of hyperkalemia. ECG and plasma potassium estimation should be done.
- Avoid hypoventilation.
- Avoid suxamethonium.
- Avoid IV fluids containing potassium.

HYPOKALEMIA

Plasma potassium concentration is less than 3.5 mEq/L. Loss of potassium usually takes place either from gastrointestinal tract or from urine.

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Causes

- Diuretic therapy, diuresis
- Excessive losses from GI tract, vomiting, diarrhea
- Peritoneal dialysis
- Diabetic ketosis
- Cushing's syndrome, hyperthyroidism
- Cirrhosis of liver
- Congestive cardiac failure
- Excessive cortisol
- Altered distribution of potassium between intra and extracellular fluid
  - Alkalosis
  - Glucose and insulin
  - Hypercalcemia
  - Hypomagnesemia
  - Familial periodic paralysis.
- Magnesium depletion.

Clinical Manifestations

- Lethargy, apathy, anorexia, confusion, amnesia, nausea.
- Skeletal muscle weakness, asthenia.
- Polyuria.
- Metabolic alkalosis.
- Hypotension.
- Myocardial weakness, conduction defect.
- Serum potassium < 3.5 mEq/L.
- *ECG changes*: Flattened T waves with ST segment depression.

Treatment

- Oral or IV potassium supplementation
- Correction of excessive hyperventilation
- Identify and treat the underlying cause.

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Anesthetic Considerations

- Avoid glucose loads.
- Avoid hyperventilation.
- Nondepolarizing muscle relaxants may have prolonged effect.
- Avoid drugs that cause low potassium.
- ECG and plasma potassium estimation should also be monitored.

HYPERNATREMIA

High serum sodium levels (more than 145 mEq/L) may occur in:

- Drug administration, mineralocorticoid excess.
- Excessive IV infusion of sodium-containing fluids, sodium bicarbonate.
- Dehydration (diarrhea, sensible losses, osmotic diuresis).

Clinical manifestations depend on the ECF volume status, circulatory overload or volume contraction.

- Severe thirst, oliguria.
- Altered mental status, delirium, muscle irritability, convulsion, coma.
- In presence of hypovolemia, tachycardia, hypotension, decreased skin turgor.
- Serum sodium level more than 145 mEq/L.
- Serum osmolality more than 300 mEq/L.
- Peripheral edema and hypertension in cases of expanded intravascular fluid volume.

Treatment

- *When ECF volume is excess*: Excess total sodium needs removal either by diuresis or dialysis in cases of renal failure. Water replacement should be done by administering 5% dextrose solution.

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- *When ECF volume is depleted*: In pressence of circulatory impairment, salt and water deficit needs correction with isotonic saline. Following hemodynamic stability, the remaining fluid deficit should be corrected with 5% dextrose solution or hypotonic saline.

HYPONATREMIA

A reduced serum sodium level (<135 mEq/L).

Causes

- *Associated with hypovolemia*: Vomiting, diarrhea, third, space sequestration. adrenal insufficiency, osmotic diuresis, thiazides, hypoaldosteronism, salt-losing nephropathy.
- *Associated with hypervolemia*: Renal failure, congestive heart failure, cirrhosis of liver, nephrotic syndrome, pregnancy.
- *Associated with normovolemia*: Syndrome of inappropriate ADH(SIADH), hypothyroidism. Water intoxication due to excessive administration of water or hypotonic IV solutions particularly in presence of renal insufficiency. Following transurethral prostatectomy, cystoscopic surgery, etc.
- *Hypertonic or isotonic hyponatremia*: Hyperglycemia, IV mannitol infusion.
- *Pseudohyponatremia*: Severe hyperlipidemia, hyperproteinemia.

Manifestations

- Nausea, vomiting, anorexia.
- Lethargy, weakness.
- Confusion, irritability, generalized convulsion.
- Coma.
- Respiratory arrest, death.

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- Serum sodium less than 130 mEq/L. Osmolality less than 280 mEq/L.

Management

- Diagnose the causative factor and treat accordingly.
- Corticosteroids in cases of adernal insufficiency.
- Gradual correction of serum sodium is recommended, not more than 0.5 mEq/L per hour unless serious CNS signs are present.
- If hypovolemic: IV infusion of normal saline.
- If hypervolemic: Water restriction, loop diuretics and cautious replacement of urine output by normal saline.
- Hypertonic saline should not be used unless patient is markedly symptomatic or with a low sodium and low osmolality.

Note

- Sodium content of common infusion fluids in mmol/L:
  Citrated plasma: 150
  Normal saline (0.9%): 150
  Hartmann solution: 131
  Sodium bicarbonate 1.4%: 167
  Sodium lactate M/6: 167
- Sodium deficit (mmol) can be calculated as (140 – plasma sodium) × body weight in kg × 0.6

HYPERCALCEMIA

Calcium is most vital for nerve and skeletal muscle excitability. Nonionized calcium is physiologically active. It is bound to albumin. Normal serum level ranges from 4.5 to 5.7 mEq/L, total 8.5 to 10.5 mg%; ionized 4 to 5 mg%. In hypercalcemia, serum calcium is more than 5.5 mEq/L.

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Causes

- Primary hyperparathyroidism
- Neoplastic diseases with metastasis in bones
- Vitamin D intoxication
- Sarcoidosis
- Paget's disease of bone
- Hyperthyroidism
- Milk alkali syndrome
- Adrenal insufficiency
- Loop diuretics.

Manifestations

- Thirst, dehydration.
- Anorexia, nausea, vomiting, abdominal pain.
- Constipation, polyuria.
- Mild hypertension.
- Altered mental status, psychosis, stupor, coma.
- Renal dysfunction, azotemia.
- Serum calcium raised.
- ECG: Shortened QT interval due to short ST segment a venticular extrasystoles.

Management

- The etiological factor should be determined and treated accordingly.
- Rehydration, volume expansion, diuretics.
- Glucocorticoids, calcitonin, bisphosphonates may be included in certain cases.
- Dialysis.
- Resection of parathyroid adenoma, if present.
- Restriction of intake of calcium.
- Maintain adequate urine output.

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HYPOCALCEMIA

Serum calcium concentration is below 4.5 mEq/L.

Causes

- Vitamin D deficiency, osteomalacia.
- Hypoparathyroidism.
- Chronic renal failure.
- Hypoalbuminemia.
- Hypophosphatemia, hypomagnesemia.
- Loop diuretics.
- Citrate intoxication in cases of massive blood transfusion.

Manifestations

- Muscle cramps, laryngospasm.
- Tetany, convulsion.
- Numbness, circumoral paresthesia.
- Hypotension.
- Diplopia, papilledema.
- ECG changes: Prolonged QT interval, ventricular arrhythmias.
- Serum calcium low.

Management

- Identify the cause and treat accordingly
- IV calcium gluconate to control tetany, convulsion
- Magnesium replacement
- Oral calcium and vitamin D supplementation.

Anesthetic Considerations

- Alkalosis should be avoided.
- Intraoperative hypotension, cardiac depression can occur.
- Severe muscle spasm, laryngospasm, convulsion can occur.

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HYPERMAGNESEMIA

Serum magnesium is more than 2 mEq/L.

Causes

- Hemolysis, renal insufficiency. Mild increase in serum magnesium is also seen in cases with diabetic ketoacidosis, adrenal insufficiency, hyperparathyroidism and lithium toxicity.
- Clinical manifestations: Weakness, hyporeflexia, respiratory muscle paralysis, confusion, altered mentation.
- ECG: Increased PR interval, heart block, and cardiac arrest in severe cases.
- Serious effects of hypermagnesemia are mostly due to calcium antagonism in cardiovascular system.
- Increased uric acid, phosphate, and potassium and decreased calcium may also be seen.

Management

1. Correct renal insufficiency, if possible
2. Intravenous calcium chloride in severe cases
3. Hemodialysis.

HYPOMAGNESEMIA

Serum magnesium is less than 1.5 mEq/L. It is often associated with decreased calcium and potassium.

Predisposing Conditions

1. Drug therapy: Furosemide, aminoglycosides, amphotericin, digitalis, cyclosporine, etc.
2. Diarrhea
3. Alcohol abuse
4. Diabetes mellitus.

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Clinical Manifestations

- Muscle cramps, athetoid movements, twitching, tremor, convulsions, delirium, muscle wasting, hyperreflexia, nystagmus, hypertension, and hypertensive CNS syndrome.
- Electrolyte changes: Hypokalemia, hyponatremia, hypophosphatemia, and hypocalcemia.
- ECG changes: Tachycardia, premature atrial or ventricular beats, increased QT interval, and ventricular tachycardia/fibrillation.

Management

1. Causative factors should be identified and treated.
2. Intravenous magnesium replacement followed by oral therapy.
3. Calcium and potassium supplements may be needed in selected cases.

HYPERPHOSPHATEMIA

Here the serum phosphate level is more than 5 mg/dL or 1.6 mmol/L. Hypocalcemia may be noted in some cases.

Causes

1. Renal insufficiency, impaired renal excretion of phosphates.
2. Excessive phosphate release as in cases with massive cell necrosis as in rhabdomyolysis or tumorlysis.

Clinical Manifestations

Formation of insoluble calcium-phosphate complexes, mostly deposited in soft tissues. Acute hypocalcemia can cause tetany.

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Management

1. Causative factor to be detected and treated, if possible.
2. Aluminum-containing antacids can be used to bind phosphorus in upper gastrointestinal tract.
3. Hemodialysis.

HYPOPHOSPHATEMIA

Here the serum phosphate level is less than 2.5 mg/dL or 0.8 mmol/L.

Causes

1. Common in any type of feeding regimen such as oral, enteral or parenteral feeding. Total parenteral nutrition can cause a severe drop of severe serum phosphate level.
2. Respiratory alkalosis.
3. β-receptor agonists can promote hypophosphatemia.
4. Phosphate binding agents: Aluminum-containing compounds can impede phosphate absorption in upper GI tract and promote phosphate depletion.
5. Diabetic ketoacidosis.

Clinical Manifestations

- Anorexia, myopathy, arthralgia, irritability, confusion, and convulsion.
- Hemolysis in severe cases.
- Hypophosphatemia is associated with depletion of 2,3 diphosphoglycerate and this shifts oxyhemoglobin dissociation curve to the left and thus hemoglobin is less likely to release oxygen to the tissues.
- However, a severe established hypophosphatemia may not show any harmful clinical manifestation.

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Arun Kumar Paul BSc MBBS DA MS (Anesth) is Ex-Professor, Department of Anesthesiology, Medical College and Hospitals, Kolkata, West Bengal, India, with vast experience of teaching for more than 35 years. He is the well-known author of several books on anesthesiology.



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