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A REVIEW ON NEUROLOGIC MANIFESTATIONS IN MAJOR ELECTROLYTIC ABNORMALITIES

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ABSTRACT

Central and peripheral neurologic manifestations are associated with electrolyte disturbances. The electrolyte disturbances in different neurological condition is primarily functional than structural.[1,2] Electrolyte disturbances may affect the brain and tissues and need to be identified as they can cause severe and life-threatening complications when not identified and properly treated. The neurological manifestations reflect the severity of acute neuronal derangement and therefore require emergency treatment.[2-3] The proper functioning of nervous system requires appropriate distribution of electrolytes in its related compartments. Neurologic manifestations can occur when there is a disturbance in the concentration of these gradients. Altered level of phosphate, sodium, and calcium may results in seizures. Imbalances in magnesium, calcium, and phosphate may be a reason for the changes in sensorium.[4,5]

KEYWORDS: Neurologic manifestation, Electrolyte disturbance, seizure.

INTRODUCTION

Central and peripheral neurologic manifestations are associated with electrolyte disturbances. The electrolyte disturbances in different neurological condition is primarily functional than structural.[1,2] Electrolyte disturbances may affect the brain and tissues and need to be identified as they can cause severe and life-threatening complications when not identified and

properly treated. The neurological manifestations reflect the severity of acute neuronal derangement and therefore require emergency treatment.[2-3] Acute or severe electrolyte imbalances can manifest with rapidly progressive neurologic symptoms or seizures. Seizures are mainly observed in patients with sodium disorders (Eg; hyponatremia), hypocalcaemia, and hypomagnesaemia.[3] The proper functioning of nervous system requires appropriate distribution of electrolytes in its related compartments. Neurologic manifestations can occur when there is a disturbance in the concentration of these gradients. Altered level of phosphate, sodium, and calcium may results in seizures. Imbalances in magnesium, calcium, and phosphate may be a reason for the changes in sensorium.[4,5] Several symptoms includes headache, vomiting, nausea, gait disturbances, dizziness, mild dementia, irritability, involuntary muscle contractions are common among mild hyponatremia.[5-7] The earliest clinical symptoms such as nausea and malaise may be observed at 125-130mEq/L of sodium level and decline to 115-120mEq/L may cause convulsions, unconsciousness and respiratory collapse may occur.[7,8, 9]

Table 1: Different degrees of the electrolyte disturbances that most frequently cause seizures.

Electrolyte disturbances	Mild	Moderate	Severe
Hyponatremia	130–134 mEq/L	125–129 mEq/L	<125 mEq/L
Hypernatremia	145–149 mEq/L	150–169 mEq/L	≥170 mEq/L
Hypocalcaemia	1.9–2.2 mEq/L	1.9–2.2 mEq/L	<1.9 mEq/L
Hypercalcemia	2.5–3 mEq/L	3–3.5 mEq/L	3.5–4 mEq/L
Hypomagnesaemia	0.8–1.6 mEq/L	0.8–1.6 mEq/L	<0.8 mEq/L

Disorders of sodium and Osmolarity can be responsible for an encephalopathy characterized by depression of neuronal activity, with confusion, headache, psychomotor slowing and lethargy as the major clinical manifestations, usually associated with signs of irritability. Hypercalcemia and hypomagnesaemia may also produce both aneuronal depression with encephalopathy and neuronal irritability. Hypocalcaemia and hypomagnesaemia lead almost exclusively to CNS irritability clinically manifesting with seizures, whereas disorders of potassium rarely produce symptoms in the CNS, with muscle weakness being their major clinical manifestation.[2,10,11]

Potassium imbalances rarely involve the CNS and are predominantly associated with muscle symptoms. The symptoms include myalgia, muscle weakness and alleviation of the fatigue reported with 3.0 to 3.5mEq/L of potassium and further decline in potassium concentration

2.5 to 3.0mEq/L results in muscle weakness and cramps. If the serum potassium concentration decrease below 2.5 to 2.0mEq/L may lead to Rhabdomyolysis and myoglobinuria. Hypokalemic cerebral symptoms may be mild and rare. Mild muscle weakness is the commonly observed manifestation of hyperkalemia.[1,12,13]

Excitable membranes of muscle and nervous tissues are stabilized by serum calcium. So calcium disorders may result in neurologic manifestations.[1,14] Hypocalcemia may results in neurologic symptoms like seizures and alterations of the mental status. The manifestation of other diseases includes mental disturbances, neurosis, delusion, delirium, mental clouding, dementia and retardation of the mental activity. In the case of chronic hypocalcaemia, chorea and Parkinsonism may be observed. The majority observed indication of hypocalcaemia is tetany.[15] Whereas in a few cases hypocalcaemia leads to the generalized or focal seizures.[16]

Causes of electrolyte disturbances

Table 2: Major causes of electrolyte disturbances.

Electrolyte disturbances	Main causes
Hyponatremia	Depletion of circulating volume Congestive heart failure Cirrhosis Diarrhea Disorders leading to increased antidiuretic hormone (ADH) levels Syndrome of inappropriate ADH secretion Adrenal insufficiency Hypothyroidism Pregnancy Recent surgery Excessive water intake Polydipsia Drugs-Thiazide diuretics, Desmopressin, mannitol, sorbitol, glycine, carbamazepine, oxcarbazepine, eslicarbazepine
Hypernatremia	Excessive water loss Impairment in access to water (infants, elderly) Diarrhea Central of nephrogenic diabetes insipidus Drugs (mannitol) Overload of sodium Hypertonic sodium solutions Water moving into cells Convulsive seizures Severe physical exercise

Hypocalcemia	Hypoparathyroidism Post-surgical (thyroidectomy, parathyroidectomy) Idiopathic Secondary hyperparathyroidism in response to hypocalcemia (renal failure) Drugs Bisphosphonates Calcitonin Severe vitamin D deficiency Insufficient calcium intake (malnutrition) Infants of mothers with vitamin D deficiency
Hypercalcemia	Malignancy Drugs Thiazide diuretics Vitamin D intoxication Lithium Primary hyperparathyroidism
Hypomagnesemia	Loss of magnesium Diarrhea Abuse of laxatives Drugs (loop and thiazide diuretics, cyclosporines, aminoglycoside antibiotics)

Diagnosis

To identify the electrolyte disturbances leading seizures, a complete serum chemistry evaluation, including measurements of sodium, calcium, and magnesium should be performed, in particular in subjects with a first time seizures.[3,17,18] Since between 15 and 30% of acute symptomatic seizures among elderly patients occur in the setting of toxic-metabolite causes,[19] this diagnostic workup is particularly important in the elderly. If the series EEGs are performed, a gradually progressive disorganization of the EEG recordings over the course of the disease can usually be noted. Reactivity to photic or other types of external stimulation is frequently altered.[20] EEG evolution generally correlates well with the severity of encephalopathy; more specifically, the degree and severity of EEG abnormalities correlate with the rate of change of electrolyte balance rather than with the absolute level of a specific electrolyte or metabolite.[21] In metabolic encephalopathies, EEG patterns are usually unspecific, including various degrees of diffuse slowing, epileptiform discharges, intermittent rhythmic slow activity, and occurrence of triphasic waves, which are usually reversible after treatment of the underlying causes.[20,22]

Potassium (K)

The magnitude of the potassium gradient across cell membranes determines excitability of nerve and muscle cells, including the myocardium. Rapid or significant changes in the serum potassium concentration can have life-threatening consequences. Evaluation of serum potassium must consider the effects of changes in serum pH. When serum pH falls, serum potassium rises because potassium shifts from the cellular to the vascular space. When serum pH rises, serum potassium falls because potassium shifts from the vascular space into the cells. Effects of pH changes on serum potassium should be anticipated during therapy for hyperkalemia or hypokalemia and during any therapy that may cause changes in serum pH (eg, treatment of diabetic ketoacidosis).

Hyperkalemia

Hyperkalemia is defined as a serum potassium concentration >5 mEq/L, it is moderate (6 to 7 mEq/L) and severe (>7 mEq/L) hyperkalemia that are life-threatening and require immediate therapy. Hyperkalemia is most commonly seen in patients with end-stage renal disease. Other causes are listed in the Table2. Many medications can contribute to the development of hyperkalemia. Identification of potential causes of hyperkalemia will contribute to rapid identification and treatment. Signs and symptoms of hyperkalemia include weakness, ascending paralysis, and respiratory failure. If hyperkalemia is left untreated, a sine-wave pattern, idioventricular rhythms, and systolic cardiac arrest may develop.[23-25]

Treatment of hyperkalemia

The treatment of hyperkalemia is determined by its severity and the patient's clinical condition. Stop sources of exogenous potassium administration and evaluate drugs that can increase serum potassium. Additional treatment is based on the severity of the hyperkalemia and its clinical consequences.

The following sequences list the treatments for hyperkalemia in order of priority

For mild elevation (5 to 6 mEq/L), remove potassium from the body with

1. **Diuretics:** furosemide 40 to 80 mg IV
2. **Resins:** Kayexalate 15 to 30 g in 50 to 100 mL of 20% sorbitol either orally or by retention enema

For *moderate* elevation (6 to 7 mEq/L), shift potassium intracellularly with

1. Glucose plus insulin: mix 25 g (50 mL of D50) glucose and 10 U regular insulin and give IV over 15 to 30 minutes
2. Sodium bicarbonate: 50 mEq IV over 5 minutes (sodium bicarbonate alone is less effective than glucose plus insulin or nebulized albuterol, particularly for treatment of patients with renal failure; it is best used in conjunction with these medications.[26,27])
3. Nebulized albuterol: 10 to 20 mg nebulized over 15 minutes

For *severe* elevation (>7 mEq/L with toxic ECG changes), you need to shift potassium into the cells and eliminate potassium from the body. Therapies that shift potassium will act rapidly but they are temporary; if the serum potassium rebounds you may need to repeat those therapies. In order of priority, treatment includes the following:

- **Shift potassium into cells**

1. **Calcium chloride (10%):** 500 to 1000 mg (5 to 10 mL) IV over 2 to 5 minutes to reduce the effects of potassium at the myocardial cell membrane (lowers risk of ventricular fibrillation [VF])
2. **Sodium bicarbonate:** 50 mEq IV over 5 minutes (may be less effective for patients with end-stage renal disease)
3. **Glucose plus insulin:** mix 25 g (50 mL of D50) glucose and 10 U regular insulin and give IV over 15 to 30 minutes
4. **Nebulized albuterol:** 10 to 20 mg nebulized over 15 minutes [27-29]

- **Promote potassium excretion**

5. **Diuresis:** furosemide 40 to 80 mg IV
6. **Kayexalate enema:** 15 to 50 g plus sorbitol PO or per rectum
7. Dialysis

Hypokalemia

Hypokalemia is defined as a serum potassium level <3.5 mEq/L. The most common causes of low serum potassium are gastrointestinal loss (diarrhea, laxatives), renal loss (hyperaldosteronism, severe hyperglycemia, potassium depleting diuretics, carbenicillin, sodium penicillin, amphotericin B), intracellular shift (alkalosis or a rise in pH), and malnutrition. The major consequences of severe hypokalemia result from its effects on nerves and muscles (including the heart). The myocardium is extremely sensitive to the effects of hypokalemia, particularly if the patient has coronary artery disease or is taking a digitalis derivative. Symptoms of mild hypokalemia are weakness, fatigue, paralysis, respiratory

difficulty, constipation, paralytic ileus, and leg cramps; more severe hypokalemia will alter cardiac tissue excitability and conduction. Hypokalemia can produce ECG changes such as U waves, T-wave flattening, and arrhythmias (especially if the patient is taking digoxin), particularly ventricular arrhythmias. Pulseless electrical activity or a systole may develop.

Treatment of hypokalemia

Treatment of hypokalemia consists of minimizing further potassium loss and providing potassium replacement. IV administration of potassium is indicated when arrhythmias are present or hypokalemia is severe (potassium level of 2.5 mEq/L). Gradual correction of hypokalemia is preferable to rapid correction unless the patient is clinically unstable. Administration of potassium may be empirical in emergent conditions. When indicated, the maximum amount of IV potassium replacement should be 10 to 20 mEq/h with continuous ECG monitoring during infusion. A more concentrated solution of potassium may be infused if a central line is used, but the tip of the catheter used for the infusion should not extend into the right atrium. If cardiac arrest from hypokalemia is imminent (ie, malignant ventricular arrhythmias are present), rapid replacement of potassium is required. Give an initial infusion of 10 mEq IV over 5 minutes; repeat once if needed.

Sodium (NA^+)

Sodium is the major intravascular ion that influences serum osmolality. An acute increase in serum sodium will produce an acute increase in serum osmolality and decrease in serum sodium will produce an acute fall in serum osmolality. A sudden decrease in serum sodium will produce an acute shift of free water from the vascular into the interstitial space and leads to cerebral edema.[30,31] A sudden increase in serum sodium will produce an acute shift of free water from the interstitial to the vascular space. The rapid correction of hyponatremia leads to the development of pontine myelinolysis and cerebral bleeding,[32-34] so monitor neurologic function in the patient with hypernatremia or hyponatremia, mainly during correction of these conditions. As possible, correct serum sodium slowly and carefully control the total change in serum sodium over 48 hours and avoiding overcorrection.[35,36]

Hyponatriemia

Hyponatremia is defined as a serum sodium level of less than 135mEq/L and is considered severe when the serum level is below 125mEq/L. It is caused by an excess of water relative to sodium. Most cases of hyponatremia are caused by reduced renal excretion of water with continued water intake or by loss of sodium in the urine.

Clinical features

The clinical features of hypotonic hyponatremia are mostly related to CNS dysfunction. The serum sodium concentration decreases severe within hours. When hyponatremia reaches 120mEq/L, it leads to brain cell swelling and herniation with neurologic symptoms as the major complications. If the decline in serum sodium occurs slowly and gradually [>48h], the risk of cerebral edema and neurologic manifestations can be minimized.[37,38]

Because of the large brain-to-skull size ratio, children are at high risk of developing symptomatic hyponatremia. Severe and rapidly evolving hyponatremia may cause seizures, which are usually generalized tonic-clonic, and generally occur if the plasma sodium concentration rapidly decreases to <115 mEq/L. The clinical outcome of neurological complications of hyponatremia is influenced by age, gender and other several factors. Administration of some drugs like desmopressin[39,40] or thiazide diuretics,[40] tricyclic antidepressants, may also lead to hyponatremia and seizures. Clinical conditions including fever and polydipsia can cause hyponatremia. Ingestion of Sodium phosphate or sodium picosulphate/magnesium citrate combination which is used for the evacuation of colon and rectum prior to colonoscopy or colorectal surgery is another cause associated with hyponatremia. Antiepileptic drugs[AED] Carbamazepine[CBZ],Oxcarbazepine[OXC], and Eslicarbazepine[ESL] can cause hyponatremia due to syndrome of inappropriate antidiuretic hormone.[40] AED-induced hyponatremia is usually asymptomatic, but in some cases it may result in headache, confusion, general malaise, somnolence and in exacerbation of seizures.

Treatment

The most common treatment for hyponatremia consists of hypertonic saline (3%), which produces a rapid reduction in brain volume and intracranial pressure. The target serum sodium for therapy should be 120 mEq/L to 125 mEq/L. Aggressive treatment with hypertonic saline solution have risk of shrinkage of the brain leading to osmotic demyelin-ation syndrome manifesting with severe neurologic symptoms such as quadriplegia, pseudobulbar palsy, coma, and even death.[2,10,20] So, the sodium concentration should be corrected at a rate of 0.5 mEq/L/h. Higher correction rates (a rate of 1 mEq/L to 2 mEq/L/h) have been used in young patients who are at a risk for respiratory arrest, severe neurologic sequelae, and death.[21]

If the patient develops neurologic compromise, administer 3% saline IV immediately to raise the serum sodium at a rate of 1 mEq/L per hour until neurologic symptoms are controlled.

After neurologic symptoms are controlled, provide 3% saline IV to correct the serum sodium at a rate of 0.5mEq/L per hour. To determine the amount of sodium (eg, 3% saline) required to correct the deficit, calculate the total body sodium deficit.

The following formula may be used

Na^+ deficit= (desired [Na^+]-current [Na^+]) x 0.6* x body wt (kg) (*use 0.6 for men and 0.5 for women).

Once the Na^+ deficit is estimated, determine the volume of 3% saline (513mEq/L Na^+) necessary to correct the deficit (divide the deficit by 513 mEq/L). Plan to increase the sodium by 1 mEq/L per hour over 4 hours (or until neurologic symptoms improve); then increase the sodium by 0.5 mEq/L per hour. To calculate this amount, use the amount you wish to correct the sodium in an hour (eg, 0.5mEq/L) and multiply by 0.6 (or 0.5 in women) and then multiply by the body weight; that will calculate the amount of sodium to administer that hour. Check serum sodium frequently and monitor neurologic status.

Hypernatremia

Hypernatremia is defined as a serum sodium concentration in plasma >145 mEq/L. It may be caused by a primary gain in Na or excess loss of water. Hypernatremia may cause neurologic symptoms such as altered mental status, weakness, irritability, focal neurologic deficits, and even coma or seizures. The severity of symptoms is determined by the speed and magnitude of the change in serum sodium concentration.

Clinical features

Patients having hypernatremia can have rupture of cerebral vein, intra-cerebral and subarachnoid hemorrhages, due to brain shrinkage which can leads to seizures.[10] Although rapid sodium loading and correction can cause seizures, convulsions but only less than 40% of patients are treated by rapid infusion of hypotonic solutions.[10]

Treatment

Patients having prolonged hypernatremia, cerebral edema may occur when the osmolality is abruptly normalized; this may lead to convulsive seizures, coma, and death. The targeted correction of chronic hypernatremia should not exceed 0.5–0.7 mEq/L/h and 10mEq/L/day to avoid complications. In patients with hypovolemia, ECF volume is restored with normal saline or 5% dextrose in half-normal saline solution to prevent fall in the serum sodium

concentration. Administration of D5W should be avoided as it will reduce the serum sodium rapidly.

The quantity of water needed to correct hypernatremia can be calculated by using the following equation

Water deficit (in liters) = [plasma Na^+ concentration-140]/140 x total body water

Total body water is approximately 50% of lean body weight in men and 40% of lean body weight in women. Once the free water deficit is calculated, administer fluid to lower serum sodium at a rate of 0.5 to 1 mEq/h with a decrease of no more than approximately 12mEq/L in the first 24 hours and the remainder over the next 48 to 72 hours.

Magnesium (Mg^{++})

It is the fourth most common mineral and the second most abundant intracellular cation after potassium in the human body. It plays an important role in stabilizing excitable membranes and is necessary for the movement of potassium, sodium, and calcium in and out of cells. The combination of low potassium and low magnesium is a risk factor for severe arrhythmias. So magnesium balance is always associated with the sodium, calcium, and potassium balance.

Hypomagnesemia

Hypomagnesaemia is defined as a plasma concentration of magnesium <1.6 mEq/L; magnesium values lower than 0.8mEq/L represent severe hypomagnesaemia. It is mainly due to the decreased absorption or increased loss from either the kidneys or intestines. The other cause includes alteration in thyroid hormone function and administration of certain medications like diuretics, alcohol and pentamidine etc. Clinical symptoms include muscular tremor, fasciculation, nystagmus, tetany, altered mental status, ataxia, vertigo, seizures, dysphagia and cardiac arrhythmia.

Clinical features

Symptoms of hypomagnesaemia usually occur at serum ionized $Mg2^+$ levels lower than 1.2 mg/dL, although they do not always correlate with Mg2+ concentrations. Clinical features of hypomagnesaemia include neuromuscular irritability, CNS hyper excitability, and cardiac arrhythmias. At a level of <1 mEq/L, Seizures (usually generalized tonic-clonic) can occur in neonates and adults in association with severe hypomagnesaemia.[2]

Treatment

Table 3: Treatment of hypomagnesaemia.

Type	Serum level	Treatment
Mild and/or asymptomatic hypomagnesaemia	0.8 mEq/L - 1.6 mEq/L	Oral administration of magnesium(e.g.,magnesium gluconate), daily dose of 500mg
Symptomatic or severe	<1.2 mg/dL, <1 mEq/L	Especially if seizures are present, requires an injection of 1 to 2 g of magnesium sulfate over a 5-min period, followed by an infusion of 1 to 2 g per hour for the next few hours, which may be repeated if seizures persist.

In patients with renal insufficiency, these dosages should be reduced.[2] 4g to 6g of loading dose of magnesium sulfate can be given intravenously by diluting in 100ml fluid over 15 minutes followed by continuous intravenous infusion at 1 to 2 g per hour, and can be discontinued in 24 hours after delivery or last seizure.

Hypermagnesaemia

Hypermagnesaemia is defined as a serum magnesium concentration >2.2mEq/L (normal: 1.3 to 2.2mEq/L). The most common cause of hypermagnesaemia is renal failure. Neurologic symptoms of hypermagnesaemia are muscular weakness, paralysis, ataxia, drowsiness, and confusion. Moderate hypermagnesaemia can produce vasodilation; severe hypermagnesaemia can produce hypotension. Extremely high serum magnesium levels may produce a depressed level of consciousness, Bradycardia, cardiac arrhythmias, hypoventilation, and cardiorespiratory arrest.

Treatment

Elimination of sources of ongoing magnesium intake can reduce hypermagnesaemia. Administration of calcium can removes the magnesium from the serum. Cardiorespiratory support may be needed until magnesium levels are reduced. Lethal arrhythmias can be corrected by administration of 10% solution of calcium chloride (5 to 10 mL [500 to 1000 mg] IV) and this dose may be repeated if needed. In case of severe hypermagnesaemia, dialysis can be used. If renal function is normal and cardiovascular function adequate, IV saline diuresis (administration of IV normal saline and furosemide [1 mg/kg]) can be used to increase renal excretion of magnesium until dialysis can be performed.

Calcium (Ca^{++})

Calcium is the most abundant mineral in the body and is essential for bone strength and neuromuscular function. 50% of all calcium in the ECF is bound to albumin and other is in the biologically active, ionized form. Normally the calcium concentration is regulated by parathyroid hormone and vitamin D. Total serum calcium is directly related to the serum albumin concentration.

Hypocalcemia

Hypocalcemia is defined as a plasma calcium level of <8.5 mg/ dL or an ionized calcium concentration <4.0 mg/dL.

Clinical features

Symptoms include paresthesias of the extremities and face, followed by muscle cramps, carpopedal spasm, stridor, tetany, and seizures. Hypocalcemic patients show hyperreflexia and positive Chvostek and Trousseau signs. Cardiac effects include decreased myocardial contractility and heart failure. Hypocalcemia can exacerbate digitalis toxicity.

Treatment

Treatment with intravenous calcium is the most appropriate therapy. 100 to 300mg of elemental calcium should be infused intravenously over a period of 10 min to 20 min. Calcium- infusion drips should be started at 0.5 mg/kg/h and continued for several hours, with close monitoring. Hypocalcemic seizures should be treated with calcium replacement. Treat acute, symptomatic Hypocalcemia with 10% calcium gluconate, 93 to 186 mg of elemental calcium (10 to 20 mL) IV over 10 minutes. Follow this with an IV infusion of 540 to 720 mg of elemental calcium (58 to 77 mL of 10% calcium gluconate) in 500 to 1000 mL D5W at 0.5 to 2 mg/kg per hour (10 to 15 mg/kg). Alternatively, administer 10% calcium chloride, giving 5 mL (136.5 mg of elemental calcium) over 10 minutes, followed by 36.6 mL (1 g) over the next 6 to 12 hours IV. Measure serum calcium every 4 to 6 hours. Aim to maintain the total serum calcium concentration at 7 to 9 mg/dL.

Hypercalcemia

Hypercalcemia is defined as serum calcium levels of ≥2.5mmol/L.

Clinical features

Table 4: Clinical symptoms of hypercalcaemia Symptoms usually develop when the total serum calcium concentration is 12 to 15 mg/dL.

System	Symptoms
Neurologic system	At lower levels- depression, weakness, fatigue, and confusion At higher levels- hallucinations, disorientation, hypo tonicity, seizures, and coma.
Cardiovascular symptoms	Myocardial contractility may initially increase until the calcium level reaches 15 mg/dL. Above this level myocardial depression occurs. Automaticity is decreased Ventricular systole is shortened Arrhythmias occur because the refractory period is shortened. QT interval shortens, PR and QRS intervals are prolonged
Gastrointestinal symptoms	dysphagia, constipation, peptic ulcers, and pancreatitis

Treatment

Vigorous rehydration with normal saline should be initiated at a rate of 200 to 500 mL/h, monitoring fluid overload. Then 20–40 mg furosemide may be administered intravenously, after rehydration has been achieved. If high calcium levels persist, the use of intravenous bisphosphonates (pamidronate or zoledronate), and in second line of glucocorticoids, calcitonin, mithramycin, gallium nitrate, may be considered. In patients with adequate cardiovascular and renal function administer infusion of 0.9% saline at 300 to 500 mL/h (saline diuresis) until any fluid deficit is replaced and diuresis occurs (urine output 200 to 300 mL/h). Once adequate rehydration has occurred, the saline infusion rate is reduced to 100 to 200 mL/h.

Hemodialysis is the treatment of choice to rapidly decrease serum calcium in patients with heart failure or renal insufficiency.19 Chelating agents (eg, 50 mmol PO4 over 8 to 12 hours or EDTA 10 to 50 mg/kg over 4 hours) may be used for extreme conditions. Use of furosemide (1 mg/kg IV) for treatment of Hypercalcemia is controversial.

CONCLUSION

Central and peripheral neurologic manifestations are associated with electrolyte disturbances which are frequently observed in clinical practice. The electrolytes disorganization in different neurological condition is primarily functional than structural. Prompt management of electrolyte imbalances in early stages reverses such neurologic manifestations Neurologic manifestations may occur when there is a disturbance in the distribution and concentration of these gradients. Seizures represent an important clinical manifestation of electrolyte disorders and are more frequently observed in patients with hyponatremia, hypocalcemia, and hypomagnesemia. Early identification and correction of these disturbances are necessary to control seizures and prevent permanent brain damage. A high degree of clinical suspicion and aggressive treatment of underlying electrolyte abnormalities can prevent these abnormalities.

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CONFLICT OF INTEREST

There is no conflict of interest.

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