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Electrolyte Disturbances

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Abstract

Special neurons in the supraoptic and paraventricular nuclei of the hypothalamus contract when the extracellular fluid osmolality increases and ADH (arginine vasopressin) is secreted from the posterior pituitary. ADH enhances the reabsorption of water from the collecting tubules of the kidney. Otherwise, ADH secretion is suppressed and diuresis of water occurs. Carotid barorepressors and atrial strain receptors also stimulate ADH release nonosmotically when there is a 5-10% decrease in blood volume. Pain, emotional stress and hypoxia are other nonosmotic stimulants.

Extracellular fluid osmolality equals the sum of the concentrations of all substances dissolved in extracellular fluid and is responsible for 86% of sodium extracellular osmolality. Effective plasma osmolality can be calculated as follows:

Effective plasma osmolality = [2 x Sodium (mmol / L)] + [Glucose (mg / dl) / 18]

If total body solute content increases according to total body water, hyperosmolarity occurs. Hyperosmolarity with or without hypernatremia. The difference between the measured and calculated osmolalities is called osmolal gap. Increased osmolal gap indicates that osmotically active molecules (ethanol, mannitol, methanol, ethylene gliol etc.) increase in plasma. Osmolal gap also increases in chronic kidney failure, ketoacidosis and after irrigation with glycine.

Keywords: *Sodium; Potassium; Calcium; Magnesium*

Sodium

In hyperlipidemia and hyperproteinemia, the water phase of the plasma decreases, so the total plasma sodium decreases despite the normal sodium concentration of the plasma in the water phase [1].

Hyponatremia

Symptoms may not occur unless the plasma sodium value drops rapidly to 120-125 mmol / l. The symptoms that develop are generally related to central nervous system (CNS) dysfunction: nausea-vomiting, headache, lethargy, confusion, seizure, coma. Also, muscle cramps, rhabdomyolysis and noncardiogenic pulmonary edema may develop. Hyponatremia developing within forty eight hours poses a great risk for cerebral edema. Malnutrition cases, hypokalemic patients, burn cases, and elderly women who use premenopausal or diuretics are at higher risk [2].

Plasma sodium concentration indicates the ratio of total body sodium to total body water. This rate reduction:

1. Loss of total body sodium than total body water
2. Although total body sodium is normal, total body water increases
3. Despite the increase in total body sodium, it may occur due to the increase in total body water.

The addition of solutes (glucose, mannitol, glycerol, radiocontrast dyes) to which the cell membranes are impermeable is associated with a decrease in plasma sodium and an increase in plasma osmolality. Glucose cannot pass through the cell membrane in the absence of insulin. In hyperglycemia, due to extra and intracellular osmotic imbalance, water initially goes out of the cell and extracellular sodium is diluted. When the osmotic equilibrium is restored, although plasma osmolality is increased due to glucose, the plasma sodium concentration

is low. Every 100 mg / dl increase in plasma glucose creates a 1.6 mEq / L decrease in plasma sodium concentration. The addition of solutes that cross the cell membrane into the plasma (such as urea, ethanol, and methanol) is associated with an increase in plasma osmolarity and normal plasma sodium concentration [2].

In most cases, the cause of hyponatremia is taking free water without sufficient water excretion. As the serum sodium level decreases and ADH suppressed, quite normal kidney can release more than 10 liters of diluted urine per day. In cases of volume depletion, insufficient tissue perfusion (low effective blood volume), inappropriate ADH release, ADH is not suppressed and therefore the urine is not diluted maximally.

Causes of Hyponatremia

a. Volume depletion

b. cirrhosis

c. Congestive Heart Failure

d. Secretion

e. Adrenal insufficiency

f. Cerebral salt leak

g. Psychogenic polydipsia

h. Low solute intake

i. Ecstasy (MDMA)

j. Irrigation fluids (glycine, sorbitol)

k. Hyperglycemia

l. End-stage kidney disease

m. Fake (measurement of diluted samples by indirect polarography; hyperlipidemia, paraproteinemia)

Volume depletion stimulates sodium retention to ensure intrvascular volume. As volume depletion continues, ADH secretion creates excessive free water retention that creates a moderate effect (<10%) in intravascular volume. Hyponatremia in hypovolemia is associated with increased urinary osmolality (maximally diluted) and decreased urine sodium concentration (<25mEq / L). However, despite volume depletion, increased urinary sodium diuretic therapy can be seen in adrenal insufficiency and cerebral salt leakage. Electrolyte and volume replacement with hypotonic solution causes acute and severe hyponatremia [3].

Heart failure causes low effective blood volume and low perfusion by lowering cardiac output and increasing cirrhosis arterial vasodilation. Hyponatremia in these cases is associated with an increase in total body sodium (edema). Urinary sodium concentration is low due to the lack of renal perfusion. Nephrotic syndrome, a edematous disease, does not have hyponatremia if there is no significant kidney dysfunction.

Inappropriate ADH release (SIADH) should be suspected in cases of low serum osmolality, maximal undiluted urine (Osmolality> 400 mOsm) and increased urinary sodium level (> 40 mEq / L). The cause may be CNS diseases (tumor, infection, hemorrhage, trauma), malignancy (small cell lung ca) or medications (antiepileptics, selective serotonin reuptake inhibitors, chemotherapeutics, antipsychotics). In subarachnoid hemorrhage, SIADH is more common than cerebral salt leakage.

Polydipsia includes hyponatremia and maximal diluted urine. Intake of more than 10 liters per day exceeds the maximal water discharge capacity of the kidneys. Ectasy intoxication causes ADH release and a feeling of thirst and an acute increase in water intake, causing hyponatremia and is often the cause of death of these cases.Hyperglycemia and mannitol attract intracellular fluid into the extracellular space, causing hyponatremia, but the measured serum osmolality is high. Kidney failure is also a cause of hyponatremia with another high serum osmolality.

Hyponatremia with normal serum osmolality can be seen after irrigation with glycine, sorbitol.

Na Gap = TVS X (130- Plasma Na)

= (0.6 for kg X male or 0.5 for female) X (130- Plasma Na)

In hyponatremia, serum sodium corrections above 20 mmol / L within 24 hours cause osmotic demyelination. It is rare in 10-12 mmol / L corrections per day and in corrections that do not reach the sodium sodium level of 140 mmol / L. Neurological damage caused by quick fixes typically delays up to 2-6 days. Symptoms are often irreversible or partially reversible: dysarthria, dysphagia, spastic money or quadrparesis, lethargy, seizure, coma. Computed tomography and MRI may not be up to 4 weeks.

Treatment of Hyponatremia (<120 Mmol / L)

a. The correction rate should be under 8-12 mmol / L / day

b. Correction limit, serum sodium level should be 125-

130 mmol / L

c. Normal saline should be used when correcting (154 mmol / L), avoid dense solutions

Calculate the correction factor for hyperglycemia in hypertonic hyponatremia. Correct the underlying cause (achieve vaginalemia in hypovolemia, water restriction and diuretics in hypervolemic state, water restriction in vaginalemia, V2 receptor antagonist in CHF, cirrhosis and SIADH, hormone replacement in deficiency) Treatment of hypovolemic hyponatremia can be easily done with isotonic saline. A slight plasma sodium concentration increases when the fluids with a moderate high sodium concentration initially stabilize with plasma. As the volume condition improves and the ADH release warning disappears, a strong free water loss occurs with diluted urine. This second phase is accompanied by an increase in urine flow and a rapid increase in serum sodium concentration.

In cases such as CHF and cirrhosis where effective blood volume decreases, fluid restriction is generally performed and vasopressin receptor antagonists are considered. Administration of fluids containing sodium to these patients increases sodium burden and edema.

Treatment Of Hyponatremia in SIADH

Chronic-moderate (> 120 mEq / L): free water restriction and, if necessary, salt tablet

Symptomatic hyponatremia (Na <110-115 mEq / L): aggressive treatment with 3% saline

In the treatment with 3% saline, serum sodium is raised 1mEq / L / hour for the first few hours. It should not be increased more than 10 mEq / L in the first 24 hours and 18 mEq / L in the first 48 hours.Acute symptomatic hyponatremia may develop in marathon runners, psychotic cases, ectasy users, postoperative cases with intracerebral pathology and SIADH. In these cases, cerebral edema can cause coma and death. Especially in perimenopausal women, the risk of irreversible brain damage is high, and delay in treatment can cause cerebral herniation. In these cases, minimal symptoms should be treated aggressively. It is rapidly given to increase 100 ml of 3% NaCl serum sodium value by 2-3 mEq / L and can be repeated after 10 minutes.

Hypovolemic hyponatremia: Isotonic replacement

Normovolemic hyponatremia:

Adrenal or thyroid dysfunction: hormone therapy

2- Urinary Na> 20 mmol / L: water restriction

Hypervolemic hyponatremia:

a. Heart failure, cirrhosis, nephrotic syndrome (Urinary Na <20 mEq / L): restrict water, loop diuretics

b. Renal insufficiency (Urinary Na> 20 mEq / L): restrict water

Hypernatremia (> 158-160 Mmol / L)

Hypernatremia usually develops as a result of more than sodium water loss (hypotonic fluid loss) or sodium retention. If the loss of water is greater than the loss of sodium (osmotic diuresis, diarrhea, sweating), hypernatremia develops with low total body sodium content. The most common cause of hypernatremia with normal total body sodium content is diabetes insipidus. The most common cause of hypernatremia with increased total body sodium content is hypertonic saline and sodium bicarbonate application [4].

Since free fluid displacement is under strict control of the hypothalamus, hypernatremia occurs in affected mental state, limited free fluid intake, or hypothalamic diseases. If the feeling of thirst is normal and water intake is not restricted, normal sodium concentration is maintained (as in Diabetes insipudus). With sudden shrinkage of brain tissue, cerebral and subarachnoid hemorrhages may occur.

Causes of Hypernatremia

1. Loss of liquids containing less concentration of sodium and potassium than plasma a. Diarrhea (osmotic, nonsecretory) b. Sweating c. Pee

2. Replacement of low sodium and potassium fluid losses with normal saline

3. Diabetes Insipidus

4. Abnormal thirst response to ADH (brain damage, granulomatous diseases)

5. Impaired water intake (impaired mental state, critical illness)

6. Salt poisoning

Hypernatremia usually does not occur in secretory diarrhea such as cholera, since the sodium and potassium content of the diarrhea is similar to plasma. Hypernatremia develops because of low sodium and potassium content in osmotic, viral and bacterial diarrhea. Sodium and potassium content in sweat and gastric secretions is lower than plasma. In diuresis caused by glycosuria and mannitol, sodium and potassium content is lower than plasma. Replacing the loss of liquids with low sodium and potassium content by

isotonic cause's hypernatremia.

Rapid correction of hypernatremia that develops within hours improves the prognosis without the risk of cerebral edema. Serum sodium should be corrected slowly in cases where the formation rate is unknown. If hypernatremia exists for more than 24 hours, the correction rate should not exceed 10 mEq / day, encephalopathy and cerebral edema may develop.

Hypernatremia developing in diabetes insipidus develops acutely with an impaired water intake (<24 hours). In this case, it should be quickly corrected with 5% DW [5].

Total Body Water Volume Defect = (0.6 x Body Weight) [1 - (140 / True Na (mEq / L)]

Total body water: weight x 60% in men, weight x 50% in women

Potassium (plasma K = 3.5-5 mmol / L)

98% of total body potassium is in the intracellular area. The intracellular extracellular potassium ratio provided by active transport is important in determining the cellular membrane potential. Extracellular potassium concentration reflects the balance between potassium intake and excretion. Daily intake of potassium in adults is up to 80 (40-140) mEq. 70 mEq of the potassium intake is lost through the urine and the rest from the gastrointestinal tract. Renal potassium excretion can be as low as 5 mEq and as much as 100 mEq. Almost all of the potassium filter in the glomerule is reabsorbed from the proximal tubule and helne handle. Potassium excreted in urine is the result of distal tubular secretion. Potassium secretion in the distal tubule accompanies sodium reabsorption initiated by aldosterone.

Extracellular potassium concentration is regulated by Na-K-ATPase activity (intercellular distribution of potassium) in the cell membrane and plasma potassium (determinant of urinary and potassium excretion). Insulin and catecholamines directly decrease Na-K-ATPase activity and decrease plasma potassium. Exercise temporarily increases the level of plasma potassium by releasing potassium from muscle cells. In acidosis, extracellular hydrogen ions enter the cell and potassium goes out of the cell to maintain electrical balance. The opposite occurs in alkalosis. A change of 0.1 units in arterial pH causes a change in plasma potassium concentration of 0.6 mEq / L.

Alpha 2 adrenergic activity increases Na-K-ATPase activity, increasing intracellular potassium intake. Beta 2 adrenergic agonists decrease the potassium value by increasing the uptake of potassium by liver and muscle cells. In acute plasma osmolality increases (hypernatremia, hyperglycemia, mannitol), it is associated with the outflow of potassium due to the increase of intracellular potassium concentration due to the osmotic gradient and the increase of intracellular potassium due to intracellular dehydration.

Plasma potassium decreases due to cellular uptake in hypothermia. Administration of potassium to the hypothermic patient causes temporary hyperkalemia after heating.High distal tubular flow rates (such as osmotic diuresis) increase potassium secretion by increasing the capillary-tubular renal gradient required for potassium secretion from distal tubules. Slow tubular flow decreases this gradient by increasing potassium in the tubular fluid [6]

Hypokalaemia (plasma K <3.5 mEq / L)

It can occur in three ways:

a. Displacement of potassium between compartments

b. Increased potassium loss

c. Inadequate potassium intake

The relationship between plasma potassium concentration and total potassium deficit is weak. Potassium loss is approximately 100-200 mEq when plasma potassium drops from 4 to 3 mEq / L. When plasma potassium drops below 3 mEq / L, potassium deficit is up to 200-400 mEq. Potassium enters the cell during attacks of alkalosis, insulin therapy, ve2 agonists, hypothermia, and hypokalemic periodic paralysis. Frozen erythrocytes lose potassium during storage, and when they are infused, they can withdraw potassium and cause hypokalemia. In megaloblastic anemia given folate and vitamin B12 treatment, blood cells can take potassium and cause hypokalemia.

Potassium loss is usually renal or gastrointestinal. Renal loss of potassium is the result of increased diuresis and mineralocorticoid activity. Hypomagnesemia, renal tubular acidosis, ketoacidosis and some medications (carbenicillin, amphotericin B) increase excretion of urine and potassium. Vomiting, nasogastric aspiration, diarrhea, fistulas, villous adenomas increase potassium loss from the gastrointestinal tract.

In uremic cases, despite normal or even high plasma potassium values, there may be total body potassium deficit (mainly intracellular). In these cases, the outflow of potassium due to acidosis causes no hypokalemia. In these cases, hemodialysis may reveal a total deficit and cause hypokalemia.

Causes of Hypokalemia

Excessive Renal Loss

a. mineralocorticoid

b. diuresis

c. Bartter's syndrome

d. Chronic metabolic alkalosis

e. Antibiotics (carbenicillin, gentamicin, amphotericin B)

f. Renal tubular acidosis

Gastrointestinal losses (vomiting, diarrhea)

Displacement from outside the cell

a. Acute alkalosis

b. Hypokalemic periodic paralysis

c. Barium lavage

d. Insulin therapy

e. e.Vitamin B12 treatment

f. thyrotoxicosis

Inadequate intake potassium

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The tissues most affected by severe hypokalemia are muscles and renal tubular cells. In severe hypokalemia (<2.5 mEq / L) myopathy, rhabdomyolysis, myoglobinuria may progress to acute renal failure. Ascetic paralysis and breathing difficulties may develop in plasma potassium values below two mEq / L. Cases with severe cardiac arrhythmias and muscle weakness are treated with intravenous potassium. The aim is not to correct the potassium deficit completely but to prevent sudden dangers. Since potassium irritates peripheral veins, peripheral venous replacement rate should not exceed 8 mEq / L. Solutions containing glucose are avoided as they will increase insulin secretion. Rapid intravenous replacement (10-20 mEq / hour) requires central venous and close ECG monitoring. Intravenous replacement rate should not exceed 240 mEq per day. If there is metabolic alkalosis, potassium chloride, if there is acidosis, potassium bicarbonate or acetate is preferred.

Emergency Treatment Of Symptomatic Hypokalemia

1. Strict cardiovascular and laboratory monitoring

2. KCl IV 20 mEq / hour; adjusted to the clinic until the plasma level is 3 mEq / L

3. 10 mEq KCl in 5-10 minutes in life-threatening hypokalemia

4. Magnesium replacement

5. Switch to oral potassium whenever possible

KCl amp 7.5% 10 ml: Contains 10 mEq K

KCl amp 21% 10 ml: contains 30 mEq K

K Phosphate amp 20 ml: contains 20 mEq K +12 mEq Phosphate

Hyperkalemia (> 5 mEq / L)

Hyperkalemia may occur as a result of potassium displacement between compartments, decreased urinary excretion or increased intake. As in long term turnstile application, if blood samples are taken while erythrocytes are broken, plasma potassium is found to be incorrectly high. Kidneys can excrete up to 500 mEq per day.

Succinylcholine administration may be excreted in potassium cell in attacks of acidosis, cell lysis after chemotherapy, hemolysis, rhabdomyolysis, massive tissue trauma, hyperosmolality, digital overdose, arginine hydrochloride administration, beta 2 blocker application, and hypercalemic periodic paralysis. Wide burns, severe

muscle trauma, and administration of succinylcholine in spinal cord injury can cause severe hyperkalaemia. It can cause hyperkalaemia by inhibiting Na-K-ATPase in the digital cell membrane. Arginine hydrochloride, which is used in metabolic acidosis, can cause hyperkalemia as a result of cationic arginine ions being replaced with potassium.

A decrease in glomerular filtrate, a decrease in aldosterone activity and a decrease in potassium secretion in the distal nephron reduce the renal excretion of potassium. Glomerular filtration rates of less than five ml / min are generally associated with hyperkalaemia.

Aldosterone synthesis is significantly reduced in primary adrenal insufficiency and isolated 21-hydroxylase adrenal enzyme deficiency. NSAIDs inhibit the release of renin initiated by prostaglandin. ACE inhibitors inhibit the release of angiotensin II-induced aldosterone. High doses of heparin can prevent aldosterone secretion. The potassium spironolactone drek antagonizes the activity of aldosterone in the kidneys.

In pseudohypoaldosteronism, the kidneys are resistant to aldosterone. In lupus, sickle cell anemia, obstructive uropathy and cyclosporine nephropathy in transplanted kidneys, the potassium secretion ability of the distal nephron can be impaired. Increased potassium intake in patients with beta-blockers, renal impairment, and insulin deficiency can cause hyperkalaemia. A unit stored for twenty-one days increases plasma potassium value to 3 mEq / L in whole blood. With the administration of erythrocyte suspension, the risk of transfusion-induced hyperkalaemia is reduced, since plasma is not delivered.

The cause of muscle weakness in hyperkalaemia is prolonged spontaneous depolarization and inactivation of the muscle membrane sodium channels and may result in ascending paralysis. Cardiac findings mainly depend on the delay of depolarization. In ECG, symmetrical pointed T wave, QRS complex extension, PR interval prolongation, P wave loss, R wave amplitude loss, ST segment depression, ventricular fibrillation and asystole are generally seen with short QT. Hypcalcemia, hyponatremia and acidosis highlight the cardiac effects of hyperkalemia.

Hyperkalemia exceeding 6 mEq / L should be treated. Calcium (5-10 ml Ca gluconate or 3-5 ml 10% Ca chloride) partially antagonizes the cardiac effects of hyperkalaemia. Calcium should be used with caution in patients taking digoxin, as it will potentially potentiate diogoxin toxicity. In metabolic acidosis, IV sodium bicarbonate provides rapid intake of potassium into the cell. Beta agonists increase the uptake of potassium into the cell. Glucose + insulin therapy (10 U insulin for 30-90 g glucose) introduces potassium into the cell but 1 hour is required for peak effect.

If there is renal function, furasemide provides excretion of potassium. If there is no renal function, oral or rectal non-absorbable cation exchanger resin Na polystyrene sulfonate (oxelate) can be used for potassium elimination. Each gram of resin binds 1 mEq potassium and releases 1.5 mEq sodium [6]. Dialysis is indicated in severe and persistent hyperkalemia. By hemodialysis, 50 mEq / hour potassium can be excreted.

Emergency hyperkalemia treatment (hyperkalemia + ECG findings, acute hyperkalemia> 6mEq / L)

a. Continuous ECG monitoring

b. If there are abnormal ECG findings, 10 -20 ml 10% Ca gluconate IV 2-5 minutes. Its effect starts in 1-3 minutes and lasts 30-60 minutes. It can be repeated in 5-10 minutes. Attention in digital areas.

c. 80 ml 30% dextrose + 10 U regular insulin IV. Dextrose is not required in cases with hyperglycemia. The effect starts in 15-20 minutes, peaks in 30-60 minutes, lasts 4-6 hours. The expected serum potassium drop is 0.5-1.5 mEq / L. Careful blood sugar monitoring.

d. 10-20 mg nebulized albuterol. Its effect starts in 30 minutes, peak effect occurs in 90-120 minutes. The expected plasma potassium drop is 0.6-1 mEq / L.

e. 20-60 ml 8.4% sodium bicarbonate IV

f. Hemodialysis Furosemide 40-80 mg IV

g. Sodium polystyrene sulfonate. Oral dose: 20 g every 4-6 hours with 100 ml of 20% sorbitol. The effect start time is 4-6 hours. Enema dosage: 50 ml with 70% sorbitol + 100-150 ml water, 50 g. Enema is held for 30-120 minutes.

Calcium (normal plasma Ca: 8.5-10.5 mg / dl; 2.1-2.6 mmol / L)

98% of total body calcium is in the bone. In adults, calcium intake is 600-800 mg per day. Calcium is primarily absorbed from the proximal small intestine and 80% of the calcium taken is excreted by intestinal tract secretion. Renal calcium excretion is 100 (50-300) mg per day. 98% of the calcium, which is a glomerular filter, is reabsorbed in parallel with sodium reabsorption in the proximal tubules and helne handle. Calcium reabsorption in the distal tubules depends on the parathyroid hormone. Increased level of parathyroid hormone increases distal tubular calcium reabsorption.

50% of plasma calcium is free ionized, 40% is protein bound (albumin) and 10% is bound to anions such as citrate and amino acids. Physiologically ionized calcium is important and its normal value is 4.75-5.3 mg / dl (2.38-2.66 mEq / L; 1.19-1.33 mmol / L). Changes in plasma albumin level do not affect ionized calcium concentration but affect total plasma calcium concentration. Every 1 g / dl albumin level decreases or increases, decreases or increases the total plasma calcium concentration by 0.8-1 mg / dl. Plasma pH changes affect the level of protein binding, affecting the level of ionized calcium. Every 0.1 unit decrease in plasma pH increases ionized calcium by approximately 0.16 mg / dl.

Calcium passes into the extracellular fluid through absorption from the intestinal system and resorption from bone. It leaves the extracellular space with accumulation in the bone, urinary excretion, intestinal secretion and sweat. Extracellular ionized calcium parathyroid hormone is regulated by vitamin D and calcitonin. Plasma ionized calcium decrease stimulates PTH secretion. PTH causes mobilization of calcium from bone, increased calcium absorption from distal renal tubules and increased intestinal calcium absorption.

Hypocalcemia

The severity of symptoms is related to the rate and degree of occurrence of hypocalcemia and is affected by acid-base status, hypomagnesemia and sympathetic overactivity. Symptoms are primarily due to ionized calcium, and usually no symptoms are observed until serum ionized calcium drops below 0.7 mmol / L (2.8 mg / dl). Neuromuscular, neuropsychiatric and cardiovascular symptoms are evident in acute hypocalcemia. Muscle weakness, pain and cramps, paresthesia, dysphagia biliary and intestinal colic may be seen. Typical signs of carpopedal spasm, Trousseau and Chvostek, hyperreflexia. Laryngospasm, bronchospasm, focal and generalized seizures and papillary edema can be seen. Cardiac contractility decreases, bradycardia and ventricular dysrhythmias can be seen. Typical finding up to ECG is the prolongation of the QT interval [4].

Causes of Hypocalcemia

a. Hypoparathyroidi

b. Vitamin D deficiency (Nutritional. Malabsorption)

c. hyperphosphatemia

d. Calcium sediment (pancreatitis, rhabdomyolysis, fat embolism)

e. Calcium chelation (multiple rapid blood transfusions or albumin infusions)

Acute and symptomatic hypocalcemia is an emergency. Calcium administration should be done slowly because it contains serious cardiac risks such as asystole. It should be remembered that calcium will potentially potentiate digital toxicity. In kidney failure and symptomatic hypocalcemia, calcium can be added to diazylate fluid. Hypomagnesemia should be corrected because it impairs parathormone release and causes refractory hypocalcemia to treatment.

Emergency treatment in hypocalcemia (symptomatic acute hypocalcemia, ionized Ca <0.7mmol / L (2.8mg/dl)

i. 200 mg elemental calcium IV is given slowly in 10-20 minutes (10 ml 10% calcium gluconate 94 mg, 10 ml 10% calcium chloride 272 mg contains elemental calcium) (calcium solutions are irritant for veins and should never contain bicarbonate and phosphate)

ii. Calcium diluted in saline or dextrose is infused at a rate of 0.5-1.5mg / kg / h for 4-6 hours. Expected increase in serum calcium 0.5-0.75 mmol / L (2-3mg / dl)

iii. Oral supplementation is continued

iv. Hypomagnesemia is checked

v. Vitamin D administration is evaluated

vi. The underlying cause is treated

vii. Moderate asymptomatic chronic hypocalcemia is treated with oral calcium (1000-2600 mg / day 2-3 doses)

Hypercalcemia

It occurs when excess calcium derived from bone and / or intestine cannot be removed from the kidneys. In 80-90% of cases, the cause of hypercalcemia is hyperparathyroidism and malignancy. Moderate hypercalcemia (up to 3 mmol / L or 12mg/dl) is generally asymptomatic. Symptoms are generally not specific; nausea, vomiting, weakness, anxiety, depression, abdominal pain, constipation. Peptic ulcer may develop, severe hypercalcemia can cause acute pancreatitis. Hypercalciuria induces nephrogenic diabetes insipidus, type I distal renal tubular acidosis, nephrolithiasis and nephrocalcinosis with polyuria and poildipsia. Cognitive dysfunction and personality changes are seen over 3mmol / L, confusion, organic psychosis, stupor, coma over 4 mmol / L (16 mg / dl). In ECG, ST segment and QT interval shortening occur [4].

Causes Of Hypercalcemia

i. Hyperparathyroidism

ii. Malignancy

iii. Excessive Vit D intake

iv. Paget's disease of bone

v. Granulomatous disorders (sarcoidosis, tuberculosis)

vi. Chronic immobilization

vii. Milk-alkali syndrome

viii. Adrenal insufficiency

ix. Medication (thiazide diuretics, lithium)

Symptomatic hypercalcemia requires prompt treatment. Initially, hydration and loop diuretics are used with isotonic saline to increase calcium excretion (200-300 ml / h urine output should be provided). Diuretic therapy aggravates hypercalcemia without hydration. Although diuresis eliminates cardiac and neurological symptoms, serum calcium does not decrease to normal. Bisphosphates calcitonin should be used to further lower serum calcium.

Emergency treatment of hypercalcemia (symptomatic hypercalcemia> 3 mmol / L (> 12mg/dl)

i. Isotonic saline 1-2 liters in 1 hour, then 4-6 liters per day

ii. 20-40 g of furosemide (with diuresis, potassium, magnesium deficiency may occur every 2 hours after dehydration is corrected (assess volume frequently) (expected calcium decrease 0.25-0.75 mmol / L at the end of 24 hours) (2-6 mg / dl)

iii. Zoledronate (bisphosphonate) 4 mg infusion for 5-15 minutes (effect begins in 2 days, lasts 33 days, dose is not repeated before 7 days)

iv. calcitonin has an additive effect every 12 hours with 4 IU / kg SC or IM (bisphosphonate. Effect starts 4-6 hours. Maximum expected decrease in serum calcium 1-2 mg / dl

v. Serum Ca> 4.5-5 mmol / L (18-20 mg / dl), hemodialysis can be done in heart failure and kidney failure

vi. Vitamin D toxicity, multiple myeloma, granulomatosis, and corticosteroids are used in lymphoma. Hydrocortisone 200-300 mg IV 3-5 days or prednisone 20-30mg / day

Magnesium (1.7-2.1 mEq / L; 0.7-1 mmol / L; 1.7-2.4 mg / dl)

It is an intracellular cation that is a cofactor in many enzyme pathways. 1-2% of total body magnesium is in the extracellular area, 67% is in the bone and 31% is intracellular. Adults receive an average of 20-30 mEq / day (240-370 mg / day) of magnesium, and 30-40% of it is absorbed from the small intestine. The main elimination place of magnesium is the kidneys (6-12 mEq / day). 25% of the magnesium that passes into the glomerular filtrate is absorbed back from the proximal tubules and 50-60% from the emerging arm of the helne handle [5].

Factors that increase magnesium reabsorption from the kidneys:

a. Hypomagnesemia

b. Parathyroid hormone

c. Hypocalcemia

d. Extracellular fluid shortage

e. Metabolic alkalosis

Factors that increase the excretion of magnesium from the kidneys:

a. Hypermagnesemia

b. Acute volume expansion

c. hyperaldosteronism

d. hypercalcemia

e. Ketoacidosis

f. Diuretics

g. Phosphate excess

h. Drinking alcohol

Hypomagnesemia

Hypomagnesaemia is suspected in chronic diarrhea, hypocalcemia, refractory hypokalemia and ventricular arrhythmia. Magnesium deficit is often accompanied by a lack of intracellular electrolytes such as potassium and phosphorus. Carbohydrate intolerance and hyperinsulinism can be seen in hypomagnesaemia, since magnesium is vital for carbohydrate metabolism and energy formation and affects glucose catabolism and insulin sensitivity. Hypomagnesemia can cause hypertriglyceridemia and hypercholestrolemia [5].

Magnesium deficiency may be due to insufficient intake, insufficient absorption and increased excretion. Fractional magnesium excretion can be calculated in separating renal and intestinal excretion:

$$FE\ Mg = \{[UMg \times PCr] / [(0.7 \times PMg) \times UCr]\} \times 100$$

It shows 10-30 mg / day excretion and over 2% FEMg renal magnesium leakage. Hypomagnesemia is frequently

accompanied by hypocalcaemia (Parathyroid hormone disorder) and hypokalemia (renal K excretion). Symptoms of hypomagnesaemia may be accompanied by symptoms of hypokalemia and hypocalcemia. Together with hypokalemia, it potentiates cardiac electrical irritability and digoxin toxicity. The incidence of atrial fibrillation increases in hypomagnesaemia. PR and QT intervals may be prolonged and show that it is usually accompanied by hypocalcemia.

Emergency treatment of hypomagnesaemia (severe symptomatic hypmagnesemia; seizure, arrhythmia)

a. 4-8 mmol (1-2 g) IV in 5-10 minutes

b. 25 mmol IV infusion in 12-24 hours

c. Treatment is continued for 3-5 days in hypocalcemic cases

d. Plasma Mg value is aimed to be above 0.4 mmol / L

e. Accompanying electrolyte and acid-base status are corrected

Hypermagnesemia (> 0.95 mmol / L; 2.2 mg / dl)

The reason may be excessive intake (Mg-containing antacid, laxative), renal disorder (GFR <30 ml / min) or both. Hypermagnesemia may occur in mother and baby during gestational Mg treatment. More rare causes are adrenal insufficiency, hypothyroidism, rhabdomyolysis and lithium treatment. Typically, neurological, neuromuscular and cardiac findings are observed. Hyporeflexia, sedation and skeletal muscle weakness are characteristic findings. Hypermagnesemia impairs the release of acetylcholine and reduces the sensitivity of the motor endplate to acetylcholine. High levels (> 10 mmol / L) cause vasodilation, bradycardia, myocardial depression and hypotension. PR prolongation and QRS expansion are frequently seen. Pronounced hypermagnesemia can cause respiratory arrest [5].

In treatment, Mg intake is stopped. Effects of hypermagnesemia IV is antagonized with 1 g calcium gluconate. Diuresis is achieved with 5% dextrose ½ saline infusion and loop diuretics. It is not recommended as it will potentiate the effects of normal saline and diuresis hypermagnesemia to reduce the possibility of iatrogenic hypocalcemia. Dialysis may be required for significant renal impairment.

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