IJAYUSH
International Journal of AYUSH
AYURVEDA, YOGA, UNANI, SIDDHA AND HOMEOPATHY
http://internationaljournal.org.in/journal/index.php/ijayush/

International Journal
Panacea
Research library
ISSN: 2349 7025

Review Article **Volume 11 Issue 01** **Jan – Feb 2022**

ELECTROLYTE IMBALANCE OF SODIUM AND POTASSIUM

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ABSTRACT

Electrolytes are the compounds which readily dissociate in solution and exist as ions. That is positively and negatively charged particles. The concentration of electrolytes is expressed as milli-equivalents (mEq/l) rather than milligrams. A gram equivalent weight of a compound is defined as its weight in grams that can combine or displace one gram of hydrogen. One-gram equivalent weight is equivalent to 1000 milli-equivalents. Electrolytes are well distributed in the body fluids in order to maintain the osmotic equilibrium and water balance. The total concentration of cations and anions in each body compartment (ECF or ICF) is equal to maintain electrical neutrality. There is a marked difference in the concentration of electrolytes (cations and anions) between the extracellular and intracellular fluids. Na^+ is the principal extracellular cation while K^+ is the intracellular cation. This difference in the concentration is essential for the cell survival which is maintained by Na^+-K^+ pump. As regards anions, Cl^- and $HCO3^-$ predominantly occur in extracellular fluids, while $HPO4^-$, proteins and organic acids are found in the intracellular fluids [1]. Due to electrolyte imbalance of major cations like sodium and potassium it may result in severe ECG changes and common symptoms like tiredness, irritability, mental confusion, convulsions and coma. So before concluding any diagnosis by observing these symptoms must consider there is a chance of occurrence of electrolyte imbalance of cations.

KEYWORDS: Electrolyte, Ion, Body fluid, Cation, Anion, Extracellular fluid, Intracellular fluid

INTRODUCTION

Electrolytes are molecules that dissociate in water to their cation and anion equivalents. Electrolytes play a vital role in maintaining homeostasis in the body. Generally, the consumption of a well-balanced diet supplies the body requirement of electrolytes. In hot climates, the loss of electrolyte is usually higher. Sometimes it may be necessary to supplement drinking water with electrolytes. Dehydration is a condition characterized by water depletion in the body. It may be due to insufficient intake or excessive water loss or both. Dehydration is generally classified into two types, due to loss of water alone and due to deprivation of water and electrolytes. Dehydration may occur as a result of diarrhoea, vomiting, excessive sweating, fluid loss in burns, adrenocortical dysfunction, kidney diseases, deficiency of ADH. Water is the solvent of life and constitutes about 60% of the total body weight, distributed in intracellular and extracellular fluids.

The daily water intake (by drinking, from foodstuffs and metabolic water) and output (loss via urine, skin, lungs and faeces) maintain the body balance of water. Electrolytes are distributed in the intracellular and extracellular fluids to maintain the osmotic equilibrium and water balance. Water and electrolyte balance are usually regulated together and this is under the control of hormones-aldosterone, antidiuretic hormone and renin.[1] Electrolyte imbalance, is an abnormality in the concentration of electrolytes in the body. Electrolyte imbalances can develop by consuming too little or too much electrolyte as well as excreting too little or too much electrolyte. Hyponatraemia, hypernatremia, hypokalaemia, hyperkalaemia is the major electrolyte imbalance of cations like sodium and potassium.

DISCUSSION

Sodium is the major cation of extracellular fluid. Concentration gradient between the ECF and ICF is maintained by the sodium-potassium pump activity. Daily sodium requirement is 50-85 mmol. Sodium is required for the maintenance of osmotic pressure and fluid balance, acid-base balance, transmission of nerve impulses, regulating muscle contractions and cell permeability, absorption and transport of some nutrients, initiating and maintaining heartbeat. Plasma sodium concentration is about 135-145 mmol/l. sodium reserve in ECF is 1400 mmol and ICF is 1000 mmol. Sodium intake is about 140-260 mmol/24 hours, sodium output in urine is 120-240

mmol/24 hours, sodium output in stool is 10 mmol/24 hours, sodium output in sweat is 10-80 mmol/24 hours.

Sodium homeostasis is maintained through sodium intake and sodium excretion. An individual consumes about 150 mmol of NaCl daily. Dietary intake of sodium causes ECF volume expansion, thereby enhanced renal sodium excretion and maintain steady-state Na^+ balance. Sodium excretion is maintained by two main mechanisms like neural control and hormonal control and also regulation of sodium excretion during euvolemia, regulation of sodium excretion with volume expansion and regulation of sodium excretion with volume contraction. In neural control mechanism increased sympathetic activity decreases NaCl excretion by decreasing glomerular filtration rate, increasing renin secretion and by increasing tubular NaCl re-absorption. In hormonal control mechanism increased renin-angiotensin-aldosterone secretion decreases NaCl excretion by increasing proximal tubule absorption, increasing ADH secretion by angiotensinogen II, Increasing tubular re-absorption by aldosterone. Increased ANP secretion increases NaCl excretion by increasing glomerular filtration rate, decreasing renin secretion, decreasing aldosterone secretion, decreasing NaCl and water reabsorption by the collecting duct, decreasing ADH secretion and action of ADH on the collecting duct.

Changes in water volume in the body are expressed by a deviation of serum sodium concentration from the standard level. Increase in water volume in ECF leads to a drop in sodium concentration is called hyponatraemia and loss of water in ECF associated with an increase in sodium concentration is called hypernatraemia. Hyponatraemia is a condition of extracellular osmolality that causes lowered level of plasma sodium, and is characterised by overhydration of the cells and reduction in plasma volume. In mild to moderate hyponatraemia the value of sodium is in between 125- 135 mmol/l. it is < 125 mmol/l in severe hyponatraemia. when it is < 130mmol/l hyponatraemia is clinically significant, and become life threatening when its value is <120 mmol/l. Hyponatraemia may occur due to abnormal ADH release as in SIADH, Psychiatric illness, ADH like substances, vomiting, diarrhoea, haemorrhage, osmotic diuresis, excessive use of diuretics, heart failure, liver failure and renal failure. Tiredness, lethargy, muscular weakness, neuromuscular excitability, reduction in cardiac output, tachycardia, fall of blood pressure, dry tongue, reduced intraocular pressure, softening of the eyeballs, oliguria and uraemia, mental

confusion, stupor, convulsions, prolonged coma and death are the common clinical features of hyponatraemia.

The treatment of hyponatraemia is different in mild and severe cases. In mild cases of hyponatraemia frequent drink of water with added sodium chloride, isotonic saline (solution of 0.9%) by iv injection, 2-4 litres of isotonic saline solution are given by intravenous infusion over 6-12 hours is indicated. In severe cases of hyponatraemia 2-3 litres of isotonic saline solution by intravenous infusion during the first 2-3 hrs, followed by further 2-5 l within 24-48 hrs. In case of associated water intoxication, water intake is restricted to 500 -1000 ml in 24 hrs. hyponatraemia associated with acute adrenal insufficiency 5% glucose solution and hydrocortisone, 100-200 mg, along with intravenous infusion of isotonic saline and appropriate treatment for the underlying conditions is indicated.

Acute hyponatraemia is a condition of decrease in sodium under 125 mmol/l within 48 hours. Treatment in acute hyponatraemia should be aggressive and fast. Confusion, faintness, muscle twitches, headache, vomiting, coma are the common clinical symptoms of acute hyponatraemia. A drop in sodium occurs within more than 48 hours is called chronic hyponatraemia. Aggressive treatment in this condition may have fatal consequences like CNS demyelination. Final result may be vigil coma or exitus. Chronic hyponatraemia is treated slowly with a maximum increase in natraemia of 8-10 mmol/l/24 hrs. if properly treated, patients can survive without consequences even at sodium levels of around 90 mmol/l. Pseudohyponatremia is a condition occurs with high serum protein (> 100g/l) or lipoprotein levels. These large molecules take up considerable space in the plasma. Thereby the water and sodium content in the plasma decreases. Hyponatraemia during dehydration occurs mainly in two conditions like extrarenal losses and renal losses. Diarrhoea, sweating, vomiting, fistulas (intestinal/biliary/pancreatic) are the extrarenal losses resulting in hyponatraemia. Use of diuretics, tubulointerstitial nephritis, polycystic kidney disease, polyuric phase of acute renal failure, transplanted kidney, Addison's disease are the renal losses resulting in hyponatraemia.[2]

Hypernatremia is a clinical disorder that results from excess of sodium in the extracellular fluid, giving rise to cellular dehydration.[3] The Sodium level is 145-160 mmol/l in mild to moderate hypernatremia and when it became severe its value is > 160 mmol/l. ADH deficiency as in diabetes insipidus, iatrogenic causes like administration of hypertonic sodium

solutions, insensitivity to ADH as in lithium, tetracyclines and acute tubular necrosis, osmotic diuresis as in total parenteral nutrition, hyperosmolar diabetic coma and deficient water intake are the main causes of hypernatremia. Restlessness, irritability, lethargy, cerebrovascular damage, coma, tremor, rapid paralysis, dry skin, dry mucus membrane, elevated body temperature, tachycardia, hypertension, erratic heart rate and blood pressure, oliguria, dark and concentrated urine, anorexia, nausea, vomiting are the common clinical features of hypernatremia. In patients of hypernatremia ECG shows variations like tachycardia, QT prolongation, ST segment deviations, ventricular tachycardia in V4 and V5. The treatment of hypernatremia includes the use of diuretics, such as frusemide 40-80 mg orally or 20-40 mg by IV injection and use of hydrochlorothiazide 100mg orally or spironolactone 100-400 mg orally. If sodium depletion occurs in the case of hypernatremia diuretics withheld and the patient is given 5% saline solution, 100-200 ml by slow IV infusion. If sodium depletion occurs along with oliguria in patients with hypernatremia intravenous infusion with 25-100 g of plasma protein fractions should be given. In persons with cardiac or hepatic disease associated with oedema water intake is restricted in them.[3]

Hypernatremia due to dehydration may be caused by insufficient intake or increased losses of pure water. Seniors with a decreased sensation of thirst are predisposed to pure water deficiency due to insufficient water intake. Even a slight water deficiency may lead to mental state alterations. If the situation is not assessed and treated correctly with an I.V supply of pure water (or 5% glucose) or if the patient is sedated by antipsychotic drugs, dehydration & disorientation worsens and coma with fatal consequences will occur. Mainly water and sodium absorption are completed in the ileum. Any loss of ileum and the creation of a jejunostomy or jejuno-colon anastomosis results in significant water and sodium losses and the development of dehydration of the organism. An increased sodium requirement occurs in people with increased losses and limited absorption as in resection of small bowel, the ileum in particular, high-output small bowel fistulas, pancreatic fistulas, intestinal malabsorption's with IBD, coeliac disease, Whipple disease, vasculitis, bowel ischaemia, polyuric phases of renal failure with increased sodium excretion. In originally normovolemic patients like healthy patients with developing diarrhoeas of varied etiology resulting in dehydration with the following symptoms like lassitude, faintness, reduced blood pressure, increased albumin, increased haemoglobin. This condition is treated by infusion. In originally hypervolemic conditions like edema, cardiac

failure, hepatic cirrhosis, sepsis, and acute pancreatitis is treated by mobilization of fluids. Sodium appetite or salt appetite is a motivated behavioural state arises as a response to sodium deficiency. It drives an animal to seek and ingest foods and fluids that contain sodium.[3]

Potassium is the most abundant cation in the body. It is the principal intracellular cation equally important in the ECF for specific functions. Daily requirement of potassium ranges from 1-2 mmol/kg per day. Bananas, oranges, tender coconut water, pineapples, potatoes, beans, chicken are the major sources of potassium. Maintenance of intracellular osmotic pressure, optimal activity of enzyme pyruvate kinase, proper synthesis of DNA and proteins by ribosomes, optimal cell growth, transmission of nerve impulse, generation of cell membrane potential and muscle contraction, cardiac muscle activity, regulation of acid-base balance and water balance in the cells are the main functions of potassium.[3]

Potassium homeostasis is maintained by maintenance of internal and external potassium balance. Internal potassium balance is maintained by rapid redistribution of potassium between ECF and ICF. In conditions like insulin therapy, anabolic states, alkalosis, hypo osmolarity, mineralocorticoid deficiency, adrenergic agonists, hypokalemic periodic paralysis the shift of potassium occurs from ECF to ICF. Epinephrine, insulin, aldosterone are the main hormones maintaining internal potassium balance. Epinephrine causes stimulation of alpha receptors and releases potassium from cells resulting in local hyperkalaemia in muscles. Insulin shifts potassium into cells after ingestion of potassium in a meal by stimulating sodium-potassium ATPase. Aldosterone promotes potassium uptake into the cells by activating sodium-potassium ATPase. When aldosterone level is increased (primary aldosteronism) resulting in hypokalaemia and hyperkalaemia occurs when aldosterone level falls (Addison disease). Average diet contains 100 mEq/day of potassium. 90% of the dietary potassium is absorbed. 10 % is lost through faeces and sweats. Kidneys excrete 90-95% of potassium ingested in the diet. Thereby maintaining the external potassium balance.[3]

Hypokalaemia is a common electrolyte abnormality associated with depletion of potassium level in serum, and is manifested by muscular weakness, mental confusions and paralysis.[3] Hypokalaemia is become life threatening when its value is < 2.5 mmol/l. faster hypokalaemia develops, the more serious is the impact. Increased renal excretion by use of diuretics, increased aldosterone secretion as in liver failure, heart failure, dietary deficiency, gastrointestinal losses as

in vomiting and diarrhoea, renal diseases like renal tubular acidosis, renal tubular damage, use of exogenous mineralocorticoid are the major causes of hypokalaemia. Muscular weakness, tiredness, flaccid paralysis, loss of muscle tone, paraesthesia, muscle twitching, premature ventricular and atrial conduction, ventricular and atrial tachyarrhythmias, increase in systolic and diastolic blood pressure, abdominal distension, secondary polydipsia, hepatic encephalopathy, paralytic ileus, polyuria, respiratory failure, respiratory hyperventilation, mental confusion are the common clinical features. This condition needs to be differentiated from diabetes mellitus, cardiac failure and myasthenia gravis. ECG of hypokalaemia shows slightly prolonged PR interval, slightly peaked P wave, ST depression, shallow T wave and prominent U wave. Treatment is different in mild-moderate cases and severe cases. In mild to moderate cases 80 mmol of potassium daily orally until the levels of plasma potassium and bicarbonate return to normal. In severe cases (unable to oral medications-diarrhoea and vomiting) 20 mmol of potassium in 500 ml of isotonic saline or 5% glucose solution by intravenous infusion at a rate of less than 20 mmol/hour is given. Patient is given high intake of fruit or fruit juice. Chronic hypokalaemia is a condition in which plasma potassium level is <3.5 mEq/l. it occurs in patients who receive diuretics for hypertension, who vomit, undergo nasogastric suction, have diarrhoea, who have hyperaldosteronism and use of laxatives. If 24-hour urine potassium outputs under 20 mmol, then the condition is called extrarenal hypokalaemia. The causes are anorexia nervosa, fasting people, vomiting, gastric juice losses, fistulas and diarrhoea. The causes of renal hypokalaemia are renal tubular acidosis, polyuric phase of acute renal failure, tubulointerstitial nephritis, gitelman syndrome, barter syndrome, magnesium deficiency.[3]

Hyperkalaemia is an electrolyte disorder associated with excess of potassium and is characterised by conduction defect in the heart and myoneural junctions of the muscle. Serum potassium level is 6.5 to7 mmol/l in severe hyperkalaemia conditions. Decreased excretion of potassium as in renal failure, ACE inhibitors, drugs like amiloride, increased release from cells as in acidosis, tumour lysis, vigorous exercise, digoxin poisoning, increased invitro release from leukaemia, thrombocytosis are major causes of hyperkalaemia. Muscular weakness, flaccid paralysis, loss of tendon jerks, atrioventricular and intraventricular conduction defect, varying degree of heart block, ventricular fibrillation, tingling in the feet, hands and face, tiredness, apathy, mental confusion are the common clinical features. ECG of hyperkalaemia tall, peaked T

waves are the earliest change.[4] It also shows flat P wave, prolonged PR interval, widened QRS complex, ST depression.

Treatment of hyperkalaemia include replacement of water loss and correction of electrolyte imbalance, given diet with restricted protein but with as much as fat and carbohydrate. Patient is given 50 ml of 50 % glucose with 20 U of soluble insulin by intravenous infusion. If the situation not call for an emergency- a litre of 10% glucose solution with 10 U of soluble insulin by intravenous infusion. If associated with acidosis sodium bicarbonate, 100-200 mmol by IV infusion over 30 minutes. If associated with circulatory overload administration of sodium bicarbonate is contraindicated. If severe ECG changes observed, patient is given 10% calcium gluconate, 10-20 ml by slow IV injection over 2-3 minutes. During life threatening cases haemodialysis is indicated. Exercise induced changes in plasma potassium not usually produce symptoms and are reversed after several minutes of rest. During exercise plasma potassium may increase by at least 2-4 mEq/l in individuals who take beta 2 adrenergic receptor antagonists for hypertension. Exercise can lead to life threatening hyperkalaemia in individuals who have endocrine disorders that affect the release of insulin, epinephrine or aldosterone, whose ability to excrete potassium is impaired (renal failure) and who take certain medications (beta 2 adrenergic blockers).[3]

Severely undernourished people have an enormous intracellular potassium. A sufficient substitution of potassium of 250-300 mmol/24 hrs is required along with the substitution of phosphorus and magnesium. Failure to maintain an increased supply of intracellular ions at the beginning may lead to potential death of the patient. Cause of death is usually muscular weakness, respiratory failure, coma and cardiac arrhythmia. Chronic hyperkalaemia is a condition in which plasma potassium level is > 5 mEq/l. it occurs in individuals with reduced urine flow, low plasma aldosterone levels and renal diseases. When 24-hour urine potassium outputs exceed 50 mmol, the condition is called extrarenal hyperkalaemia. It occurs as a result of potassium redistribution between the ICF and ECF. Renal hyperkalaemia occurs when potassium output in the urine decreases under 50 mmol/24 hours. It occurs in conditions like Addison's disease and use of medicines like NSAIDs.

A falsely high plasma potassium level is called pseudo hyperkalaemia. It is caused by traumatic lysis of RBC during blood drawing. Red blood cells contain potassium. Lysis of red

blood cells releases potassium into plasma, artificially elevating plasma potassium level and resulting in pseudo hyperkalaemia. The conservative therapy of acute hyperkalaemia includes administration of calcium gluconicum 10-30 ml I.V, administration of bicarbonate 8.4% 80 ml I.V, administration of 500 ml 10% glucose+ 10 U insulin ion exchangers (resonium 100g in the form of an enema). Conservative therapy of slight chronic hyperkalaemia includes decreasing daily potassium supply to under 1.5 g (under 40 mmol), enhanced excretion through the use of loop diuretics (furosemide), sufficient hydration, ion exchangers (resonium 15g 3x a day).

The important systems contributing in the water and electrolyte balance in the body are *pranavahasrotas, mutravahasrotas*, *swedavahasrotas, annavahasrotas* and *raktavahasrotas*. *charaka* also holds the same view that sweat, faeces and urine are similar in their fluid composition because all of them contain the same *udaka*.so *udakavahasrotamsi* should include urinary system, respiratory system, GIT and skin (sweat glands). *Udaka* means water. Water is present not only in the liquid substances like blood, lymph, urine, faeces, and sweat but also is seen in all the *dhatus.* So, the *Udaka* in the *dhatu* is indicative of intracellular fluid and the *Udaka* in other liquids is indicative of the extracellular fluid. The fluid present all over the body which is protected by the skin is indicative of *rasadhatu*. Skin is a good indicator of increase or decrease in the quantity of *rasa*. *Lasika* probably stands for the interstitial fluid of skin and subcutaneous tissues. Role of lungs in the maintenance of water and electrolyte balance is obvious. They form an important route for output of water from the body. *Udakavahasrotamsi* might have been used to indicate respiratory system. Dryness in oral cavity, throat, and lower respiratory tract (*kloma)*are the features of *udakavaha Srotodushti*.[5]

CONCLUSIONS

Tiredness, lethargy, restlessness, irritability, mental confusion, convulsion, coma, oliguria, muscular weakness, vomiting, faintness, anorexia, disorientation, polyuria, paralysis are the most common clinical features due to electrolyte imbalance of cations. So before entering into any conclusions based on these clinical features must consider about the symptoms due to electrolyte imbalance of cations.

ACKNOWLEDGEMENTS

I express my sincere gratitude to all the teachers of the department *Kriyasareera*, Govt. Ayurveda College Kannur for their support and guidance. I am thankful to my institution for providing all the facilities required for the study.

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