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A Rare Case Report of Hypernatremia



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ABSTRACT

Elevated serum sodium levels greater than 145 meq/L is known as Hypernatremia. It is a rare disorder and is associated with high mortality. Since sodium and iron accompanying anions are the major effective extracellular fluid (ECF) osmoles, hypernatremia is a state of hyper osmolality. Increased ECF osmolality leads to contraction of Intra Cellular Fluid volume (ICF). It implies a deficit of total body water relative to total body sodium, caused by water intake being less than the water losses. We hereby report and discuss a case of hypernatremia in 94 years old man.



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INTRODUCTION

Hypernatremia is defined as serum sodium levels >145 meq/L¹. Hypernatremia is a rare disorder when it occurs, it is associated with high mortality ($>50\%$)². Since sodium and iron accompanying anions are the major effective extracellular fluid (ECF) osmoles, hypernatremia is a state of hyper osmolality. Increased ECF osmolality leads to contraction of Intra Cellular Fluid volume (ICF). Hypernatremia may be because of primary sodium gain or water is deficient. Two components of an appropriate response to hypernatremia are increased water intake by thirst and excretion of minimum volume by maximally concentrated urine². Defective of this mechanism leads to electrolyte imbalance. We hereby report and discuss a case of hypernatremia in 94 years old man.

CASE REPORT

A 94 years old man presented with complaints of not taking anything orally since 10 days, loose motions (6-8 episodes per day) for 10 days, altered behavior since 5 days, generalized convulsions for one day. History revealed that patient had lost thirst response. On examination, the patient was disoriented, dehydrated, loss of skin turgidity, dry mucous membrane, collapsed neck vein, blood pressure <90 mm of Hg systolic, tachycardia was also present. Plantar was bilaterally extensor. Investigation revealed – leucocytosis ($13200/\text{mm}^3$), normal platelet count (2.2 lakhs/ mm^3), Hb- 10.2 g/dl, ESR- 46 mm/ 1^{st} hr, blood sugar- 102 mg/dl, blood urea- 97 mg/dl, serum creatinine- 3.2 mg/dl, **serum sodium 168 meq/L**, serum potassium 4.1 meq/L, serum calcium 10.2 mg/dl. Urine examination: albumin/sugar- nil, pus cells- $10-15$ HPF. His bleeding time, clotting time & prothrombin time (INR) was normal. ECG- sinus tachycardia, T wave inversion V_3-V_6 , CT- head diffuse senile cortical atrophy and left old parietotemporal infarct. He was given fresh water by nasogastric tube and 5% dextrose by IV infusion and other symptomatic therapy.

DISCUSSION

Water homeostasis results from the balance between water intake and the combined loss from renal excretion, respiratory, skin and gastro-intestinal sources. In healthy person water intake and loss are regulated. Kidneys play a major role for electrolyte adjustments either by concentration or loss by urine. An intact thirst mechanism usually prevents hypernatremia. In general

derangement of the thirst response or the behavior response (primarily infants, elderly patients, psychiatric patients) or derangement of renal concentration mechanism (nephrogenic diabetes insipidus) or difficult with neurohormonal control of this concentrating mechanism (central diabetes insipidus) or by loss of free water from other sources leads to hypernatremia.

When hypernatremia occurs, cells become dehydrated because sodium primarily an ECF ion and osmotic load of the increased sodium acts to extract water from the cells, this osmotic force also extracts electrolytes across the cell membrane, this alteration results in membrane potential and electrically active membrane. If hypernatremia is not corrected after some hours it leads to structural damage. The effect of cellular dehydration are seen primarily in CNS, where stretching of the shrunken neurons and alteration of membrane potentials from electrolyte flux leads to impaired cell function. If this shrinking is severe enough, stretching and rupture of bridging veins may cause intra-cranial hemorrhage³.

Management

Gradual correction of hypernatremia is mandatory because rapid correction of hypernatremia may results in brain volume and cerebral edema with resulting in seizures and perhaps death⁴. When hypertonicity and hypernatremia are due to pure water loss, therapy is directed to lowering serum sodium concentration by water replacement and correction of the underlying disorder, such as central and nephrogenic diabetes insipidus. Patients with pure solute gain and ECF volume expanded and administration of water to lower serum sodium concentration may precipitate pulmonary edema. Therapy must be aimed at removing excess sodium. Patients with hypotonic fluid loss have both ECF and ICF volume deficits⁵. Once systemic hemodynamic abnormalities begin to improve, correction of hypernatremia can be achieved with intravenous administration of hypotonic saline solutions.

Differential diagnosis

Hypovolemic hypernatremia (i.e. water deficit > sodium deficit)

- External losses - diarrhea, vomiting, fistulas, significant burns.
- Renal losses - osmotic diuretics, diuretics, postobstructive diuresis, intrinsic renal diseases.

- Adipose hypernatremia is secondary to decreased thirst. This can be behavioral or rarely, secondary to damage the hypothalamic thirst centers.

Hypervolemic hypernatremia (i.e. sodium gains > water gains)

- Hypertonic saline, sodium bicarbonate administration, accidental salt ingestion, mineralo corticoids excess.

Evolemic hypertnatremia

- External losses- increased insensible loss (e.g. hyperventilation).
- Renal losses – central diabetes insipidus, nephrogenic diabetes insipidus

Central DI differential diagnosis

- Head trauma, suprasellar or intrasellar tumors, granulomas, histiocytosis, infectious (encephalitis, meningitis, guillain-barre syndrome), vascular (cerebral aneurysm, thrombosis, hemorrhage, Sheehan syndrome), congenital, transient DI of pregnancy.

Nephrogenic DI (deficient renal response to ADH) differential diagnosis

- Advanced renal disease (interstitial disease)
- Electrolyte disturbances - hypokalemia, hypercalcemia
- Systemic diseases - sickle cell disease, sjogren syndrome, amyloidosis, sarcoidosis, renal tubular acidosis, light chain nephropathy.
- Dietary disturbances- excessive water intake, decreased salt intake, decreased protein intake.
- Drugs- lithium, demeclocycline, colchicines, vinblastine, amphotericin B, gentamicin, furosemide, angiographic dyes, osmotic diuretics.
- Miscellaneous- postobstructive diuretics, diuretic phase of acute renal failure, osmotic diuretics, paroxysmal hypertension.

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