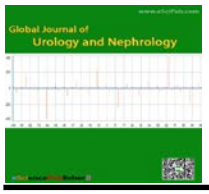
Case Report **GJUN (2019) 2:14**



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Turning complexity into clarity: polyuria and hypernatremia

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Case history

The patient is 65-year old African American male with history of hypertension. He was admitted to hospital with lethargy, disorientation, and confusion. His clinical evaluation and CT of the head revealed subarachnoid hemorrhage. He had h/o stroke 5 years ago. The patient didn't have fever, but did have a diarrhea of 1-day duration. He is receiving parenteral feeding and his urine output is 4L/day. His physical examination revealed blood pressure of 100/70mmHg, with heart rate of 100 BPM. He has dry mucous membranes and the rest of examination was unremarkable. His laboratory investigation showed sodium 159meg/L, K+ 4.6meg/L, chloride 114mEq/L, HCO3 26mEq/L, creatinine 1.9mg/L, Blood urea nitrogen 64mg/L, and glucose 200mg/L His urine sodium 70mEq/L, and urine osmolality 380mOsm/Kg H2O. His volume status is slightly dry, and he weighs 70Kg.

Keywords: hypernatremia, osmotic diuresis, water diuresis, arginine vasopressin, polyuria, polydipsia

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Case discussion

This case illustrated several information including polyuria with parenteral nutrition, low blood pressure and dry mucous membranes denoting low extracellular fluids volume. High serum sodium, and high urine osmolality in the face of hypovolemia and hypernatremia. As a consequence of his illness he suffered from low perfusion to the kidneys with worsening kidney function.

Hypernatremia is a common electrolyte abnormality and defined as a serum sodium >145mEq/L (1,2,3). Hypernatremia is hyperosmolar state caused by a decrease in total body water (TBW), relative to electrolytes content (4). Therefore, hypernatremia is a water problem not sodium problem.

It often occurs in elderly and hospitalized patients with restricted access to water and in those with impaired thirst mechanism (hypodipsia/adepsia) or mental status changes. Developing hypernatremia is virtually impossible if the thirst mechanism is intact and water is available. Hyper-osmolality caused by hypernatremia and water loss can lead to neurological cell shrinkage and brain injury. Loss of volume also can leads to circulatory collapse and organ failure. Hypernatremia is considered a risk factor for acute kidney injury (5). In patients with intra-cranial hypertension and management of hyponatremia treated with hypertonic saline 9% developed acute kidney injury. Hypernatremia is also associated with increased 30-day postoperative morbidity and mortality. Acute hypernatremia (<24-hrs) should be corrected rapidly while chronic hypernatremia should be corrected slowly for fear of cerebral edema during treatment.

Hypernatremia can be due to sodium gain or water loss or combination of both. Since sodium is the dominant extra-cellular cation and solute, hypernatremia is almost always due to water loss (6). The normal plasma osmolality lies between 275-290mOsm/Kg and it is primarily determined by sodium concentration in the extracellular fluid (ECF).

The calculated plasma osmolality = 2(Na) mEq/L + serum glucose (mg/dl)/18 + BUN (mg/dl) (2.8}.

Regulation of the plasma osmolality and plasma sodium concentration is mediated by changes in water intake and water excretion. This mechanism occurs due to

1. Urinary concentration via pituitary secretion and renal effect of antidiuretic hormone arginine vasopressin (AVP) (7,8)
2. Intact thirst mechanism (9).

Water loss can occur from renal loss or extra-renal loss. The extra-renal loss of water comprises of gastrointestinal loss like vomiting, diarrhea, use of drugs like lactulose, cathartics, nasogastric suction, gastrointestinal drains and fistulae. Skin loss of water from burns, perspiration, and cystic fibrosis. Respiratory loss due to fever or infection.

The renal water loss of water is accompanied by polyuria and hypernatremia. Polyuria can be due to water diuresis or solute diuresis. In water diuresis the urine osmolality is <300mOsm/Kg and the 24-hour total urine osmolality will be <800 mosm/kg. Water diuresis is caused by central diabetes insipidus, nephrogenic diabetes insipidus, or gestational diabetes insipidus. In solute diuresis the urine osmolality is >300 mosm/kg and the 24-hour total urine osmolality will be >800mOsm/kg. Solute diuresis can be caused by electrolytes gain like sodium chloride or bicarbonate infusion or the presence of glucose, mannitol, glycerol, BUN, or toxic alcohols in the urine.

Mortality and morbidity increased if serum sodium is >160mmol/L and one of the following factors are present in the hypernatremic patient (10);

- Low systolic blood pressure
- Low blood pH
- Serum sodium >166mmol/L
- Mean sodium reduction rate of 0.134mmol/L/H or less
- dehydration
- pneumonia

Back to our patient, his urine osmolality is 380mosm/kg and the 24-hour total osmolal load is (380 x 4L = 1520mosm/kg). Therefore, the patient has solute diuresis. His urine sodium is 79mEq/L which is high for conditions such as diarrhea. In diarrhea the kidney conserve rather than secretes sodium in the urine. In this patient the solute diuresis caused by osmotic substance from the hyper-alimentation is the cause of water loss and hypernatremia (11-13).

To calculate the water deficit in this example we can use the following formulae.

Formula I

Weight = 70kg

Previous TBW = 70x 0.6 = 42L

Actual sodium = 159mEq/L

Desired sodium = 140

New TBW = 42x 159/140 = 47.7 L

Water deficit = 47.7 – 42 = 5.7 L

Formula II

Water deficit = previous TBW x (actual SNa-1/desired SNa) = 42 x (159-1/140) = 42x158/140 = 47.4

Water deficit = 47.4 – 42 = 5.4 L

Formula III

Stern and Silver (#5) suggested that administration of 3-4ml/Kg of electrolyte-free water can lower serum sodium by 1 mEq/L in a lean individual. Applying this formula on our patient would results in;

Water deficit = 70 x 4 x (159-140) = 280 x 19 = 5.320 L

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