Guidelines

For The Management Of

Hypernatraemia

By

Dr. Sinan Butrus
F.I.C.M.S
Clinical Standards & Guidelines

Dr.Layla Al-Shahrabani
F.R.C.P (UK)
Director of Clinical Affairs

2009 AD 2709 K

Kurdistan Higher Council

For Medical Specialties

Bordi Kurdistan Bo Psporayati Pziski

Setting Clinical and Professional Excellence

Hypernatraemia

- Defined as a rise in serum sodium concentration above 145 mmol/L
- It is a hyperosmolar condition considered to be a water problem & not a sodium homeostasis problem & caused by a decrease in total body water (TBW) relative to electrolyte content.
- It occurs in extreme age patient s who are mentally and /or physically impaired, often with an acute infection, impaired thirst and/or restricted access to water, often exacerbated by pathologic conditions with increased fluid loss
-The mortality rate for chronic hypernatremia (>24 hrs) is approximately 10%, however, the mortality rate for severe acute hypernatremia (<24 hrs) in the ICU setting may reach as high as 75%

Risk factors

- Advanced age & infants with inappropriately prepared milk formula
- Mental or physical impairment
- Uncontrolled diabetes (solute diuresis)
- Underlying polyuria disorders
- Diuretic therapy
- Residency in nursing home, inadequate nursing care
- Hospitalization

  Decreased levels of consciousness
  Tube feeding
  hypertonic infusions
  Osmotic diuresis
  Mechanical ventilation
  Medication (e.g., diuretics, lactulose, sedatives)

Causes

1- Hypotonic fluid deficits (loss of water and electrolytes)

2- Nearly pure-water deficits

3- Hypertonic sodium gain (gain of electrolytes in excess of water).

1- Loss of hypotonic fluid **Hypovolemic hypernatremia**

(*loss of water in excess of electrolytes)*

Patient has decreased extracellular volume & deficit in free water and electrolytes (low total body sodium and potassium) ,hypovolemia may be more life threatening than hypertonicity, fluid resuscitation with normal saline is the first step in therapy

Renal hypotonic fluid loss

- Diuretic drugs (loop and thiazide diuretics)
- Osmotic diuresis (hyperglycemia, mannitol, urea [tube feeding])
- Renal salt wasting

- Post obstructive diuresis
- Diuretic phase of acute tubular necrosis

Nonrenal hypotonic fluid loss

- Gastrointestinal - Vomiting, diarrhea, lactulose, cathartics, nasogastric suction, gastrointestinal fluid drains, and fistulas
- Cutaneous - Sweating (extreme sports, marathon runs), burn injuries

2-Pure-water deficits **Euvolemic hypernatremia**

Patient has normal extracellular volume with normal total body sodium and potassium. This condition most commonly develops when

A. Impaired intake &lack of access to water is combined with increased insensible (respiratory or cutaneous)

B. Renal water losses. Primary renal diseases (obstructive uropathy, renal dysplasia, medullary cystic disease, reflux nephropathy, polycystic disease) or Systemic diseases with renal involvement i.e., sickle cell disease, amyloidosis

C. Drugs (amphotericin, phenytoin, lithium, aminoglycosides, methoxyflurane)

D. Inability of the kidney to concentrate the urine: Central or nephrogenic diabetes insipidus

3- Hypertonic sodium gain **Hypervolemic hypernatremia**

Patient has extracellular volume overload with high total-body sodium, Mineralocorticoid excess (Cushing syndrome, Primary hyperaldosteronism)but mostly occurs due to iatrogenic causes through administration of hypertonic electrolyte solutions (sodium bicarbonate solutions, hypertonic alimentation solutions or in haemodialysis)

History

Ask for: Altered mental status

Factors causing increased fluid excretion (diuretic therapy, diabetes mellitus, diarrhea, vomiting).

Symptoms and causes of possible diabetes insipidus (polydipsia, polyuria, history of cerebral pathology or medication use)

Find out if the hypernatremia developed acutely (< 24 hours) or over time, because this will guide treatment decisions

Physical

Assess volume status: orthostatic blood pressure changes, tachycardia, oliguria, and dry oral mucosa, and abnormal skin turgor,

Assess cognitive function: lethargy, confusion, abnormal speech, irritability, seizures, nystagmus, myoclonic jerks

Differential Diagnoses

Liver cirrhosis, Diabetes,Hypocalcemia,Hyponatremia & all other etiologies for metabolic or drug-induced encephalopathy

Laboratory Studies

Serum electrolytes (Na^+, K^+, Ca^{2+})
Glucose level Urea, Creatinine
Urine electrolytes (Na^+, K^+)
Urine and plasma osmolality
24-hour urine volume
Plasma antidiuretic hormone ADH level
Corticotropin (ACTH) , Aldosterone ,Cortisol

First step *: estimate the volume status* (intravascular volume)

Hypovolemic patient

urine with a UNa^+ <10 mEq/L extrarenal fluid losses (GI, dermal)
urine with a UNa^+ >20 mEq/L indicates renal fluid loss (diuretics, intrinsic renal disease)

Hypervolemic patient

the urine sodium level is more than 20 mEq/L

Euvolemic patient

Hypernatremia is most likely due to pure-water losses, urine sodium is variable.
Nonrenal causes with appropriately high urine osmolality - Isolated hypodipsia, increased insensible losses.
Renal water loss indicated by inappropriately low urine osmolality, in Diabetes insipidus [central, nephrogenic, partial or gestational diabetes insipidus] U_{osm} <300 mOsm/kg H_2O).

Patients with central diabetes insipidus have low Antidiuretic hormone(ADH)level ,while patients with nephrogenic diabetes insipidus have elevated level of ADH,
then, determine the response of the urine osmolality to a dose of ADH (or desmopressin).
Generally, an increase in urine osmolality of greater than 50% reliably indicates central diabetes insipidus, while an increase of less than 10% indicates nephrogenic diabetes insipidus;
responses between 10% and 50% are indeterminate

Water deprivation test

This test should be done under close monitoring & senior doctor supervision. Patients with diabetes insipidus, water deprivation induces serum hyperosmolality and hypernatremia, but urine osmolality does not increase appropriately.

Imaging Studies

A magnetic resonance imaging (MRI) or computed tomography (CT) scan of the brain may be helpful in cases of central diabetes insipidus to exclude CNS lesion.

Goals of management

(1) recognition of the symptoms, when present;
(2) identification of the underlying cause(s);
(3) correction of volume disturbances;
(4) correction of hypertonicity

Acute symptomatic hypernatremia, defined as hypernatremia occurring in a period of less than 24 hours, should be corrected rapidly.

Chronic hypernatremia, however, should be corrected more slowly due to the risks of brain edema during treatment

Treatment guidelines of symptomatic hypernatremia

- Standard supportive attention to the ABCs is appropriate. Hypovolemic patients with signs of hemodynamic compromise (e.g., tachycardia, hypotension) should receive volume resuscitation with isotonic sodium chloride solution
- Correct the serum sodium at an initial rate of 1-2 mEq/L/hr
- Replace 50% of the calculated water deficit over the first 12-24 hours
-Replace the remaining deficit over the next 24 hours
- Perform measurements of serum and urine electrolytes every 2-4 hours
- Perform serial neurologic examinations and decrease the rate of correction with improvement in symptoms
- Chronic hypernatremia with no or mild symptoms should be corrected at a rate not to exceed 0.5 mEq/L/h and/or a total of 10 mEq/day
- If a volume deficit and hypernatremia are present, intravascular volume should be restored with isotonic sodium chloride prior to free water administration.

Estimation of the replacement fluid

TBW(Total body water) = weight (kg) x correction factor
Children & Nonelderly men: 0.6
Elderly men &Nonelderly women: 0.5
Elderly women: 0.45

*Free Water D*eficit =Body Weight (kg) X Percentage of Total Body Water (TBW) X $\left\{ \frac{(\text{Serum Na})}{140} - 1 \right\}$

Common infusates and their Na^+ contents
5% dextrose in water (D_5 W): 0 mmol/L
0.45% sodium chloride in water (0.45NS): 77 mmol/L
Ringer's lactate solution: 130 mmol/L
0.9% sodium chloride in water (0.9NS): 154 mmol/L
3% sodium chloride in water(3%NS) :513 mmol/

Change in serum Na^+ = (infusate Na^+ - serum Na^+) ÷ (TBW + 1)
Decrease in serum sodium/litre of infused glucose 5% =
measured sodium concentration (mmol/L) /total body water (kg) + 1

Example 1

Man of 75 kg, 50 years old has serum sodium concentration: 168 mmol/L

target serum sodium concentration: 145 mmol/L

Decrease in serum sodium/liter of infused glucose 5% $\frac{168}{\{(75 \times 0.6) + 1\}} = \frac{168}{46} = 3.7$ mmol/L

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To achieve a decrease of 10mmol/L: infuse 10/3.7 of 1 liter = 2.7 L glucose 5%

To achieve a decrease of 10 mmol/L in 24 hours: infusion rate = 2.7 L/24 hours = 112.5 mL/hour

To achieve the desired target serum sodium concentration of145 mmol/L i.e. an additional decrease of 13 mmol/L: infuse for a further 1.3 days at the same rate (112.5 mL/hour)

Total infusion: 6.2 liters of glucose 5% over 2.3 days at 112.5 mL/hour

Example 2

70 kg weight, his serum sodium concentration is 165 mmol/L

TBW = (0.5 x 70) = 35 L

the retention of 1 L of D_5 W will reduce his serum sodium by (0 - 165) ÷ (35 + 1) = - 4.6 mmol.

The goal is to reduce his serum sodium by no more than10 mmol/L in a 24-hour period. Thus: (10 ÷ 4.6) = 2.17 L of solution is required.

About 1-1.5 L will be added for obligatory water loss to make a total of up to 3.67 L of D_5 W over 24 hours, or 153 mL/h

Example 3

A serum sodium level of 155 in a 60-kg young man represents a fluid deficit of

60 X 0.6 X ([155 / 140] - 1) or 3.9 L.

With another 900 mL of insensible losses,

the patient requires 4.8 L of fluid in the next 48 hours, resulting in an infusion rate of 100 mL/h.

The above formulae can only guide therapy, but ***serial measurements of serum sodium are prudent***

The selection of intravenous fluid is based on the following:

- If the patient is hypotensive, normal saline (lactated Ringer solution, should be used regardless of a high serum sodium concentration.
- In hypernatremic dehydration, 0.45% or NaCl should be used as a replacement fluid to prevent excessive delivery of free water and a too-rapid decrease in the serum sodium concentration.
- In cases of hypernatremia caused by sodium overload, sodium-free intravenous fluid (5% dextrose in water) may be used, and a loop diuretic may be added.
-The serum sodium concentration should be monitored frequently to avoid too rapid correction of hypernatremia which lowers plasma osmolality, leading to cerebral edema
- Once the patient is urinating, add 40 mEq/L KCl to fluids to aid water absorption into cells.
- Calcium may be added if the patient has an associated low serum calcium level

Medication

Nephrogenic diabetes insipidus: diuretic therapy (i.e., thiazides), NSAIDs

Central diabetes insipidus, desmopressin administered orally or intranasally

Partial central diabetes insipidus (chlorpropamide, clofibrate, and carbamazepine) are used to increase circulating AVP.

Hydrochlorothiazide

Adult 25-100 mg PO qd; not to exceed 200 mg/kg/d

Pediatric

<6 months: 1-3 mg/kg/d PO divided bid, total range 12.5-37.5 mg/d

6 months to 2 years: 1-2 mg/kg/d PO divided qd/bid, total range 12.5-37.5 mg/d

2-12 years: 1-2 mg/kg/d PO divided qd/bid, not to exceed 37.5-100 mg/d

Desmopressin (DDAVP)

Adult

2-4 mcg IV/SC divided bid ,Alternatively, 2-4 mcg/kg/dose intranasally

Pediatric

3 months to 12 years: 5-30 mcg/d intranasally qd or divided bid. Alternatively, 2-4 mcg/dose intranasally

>12 years: Administer as in adults

Vasopressin (Pitressin)

Adult

5-10 U IM/SC bid/qid; titrate to effect

Continuous IV infusion: 0.5 mU/kg/h (i.e., 0.0005 U/kg/h) IV initially; double dosage every 30 min prn, not to exceed 10 mU /kg/h IV (i.e., 0.01 U/kg/h)

Pediatric

2.5–10 U IM/SC bid/qid; titrate to effect

Continuous IV infusion: Administer as in adults

Diet

Hypernatremia in infants is largely due to inappropriately reconstituted infant bottle formula. Avoid preparing homemade infant formulas, and never add salt to any commercial infant formula.

In diabetes insipidus, a sodium-restricted and protein-restricted diet should be prescribed.

Patients with nephrogenic DI must be trained to avoid salt and to drink large amounts of water.

Consultations

Ask for the help of Nephrologist (nephrogenic etiologies for hypernatremia), Endocrinologist (diabetes insipidus or diabetes mellitus) & Neurosurgeon (head trauma)

Transfer

Patients with hypernatremia who are renal failure or fluid overloaded may require hemodialysis. If the serum sodium concentration is more than180 mEq/L, peritoneal dialysis should be performed using a high-glucose, low-sodium dialysate.

Complications

- Severe hypernatremic dehydration induces brain shrinkage, which can tear cerebral blood vessels, leading to cerebral hemorrhage, seizures, paralysis, and encephalopathy.
- In patients with prolonged hypernatremia, rapid rehydration with hypotonic fluids may cause cerebral edema, which can lead to coma, convulsions, and death

- Acute hypernatraemia < 24 hrs stretching of bridging veins can result in subdural hemorrhages.
- Venous congestion can lead to thrombosis of the intracranial venous sinuses.
- Arterial stretching can result in subcortical hemorrhages and cerebral infarctions.
- Seizures are possible.
- Chronic hypernatremia of more than 2 days' duration is associated with an increased mortality especially if corrected too rapidly, brain edema and associated neurologic sequelae can occur
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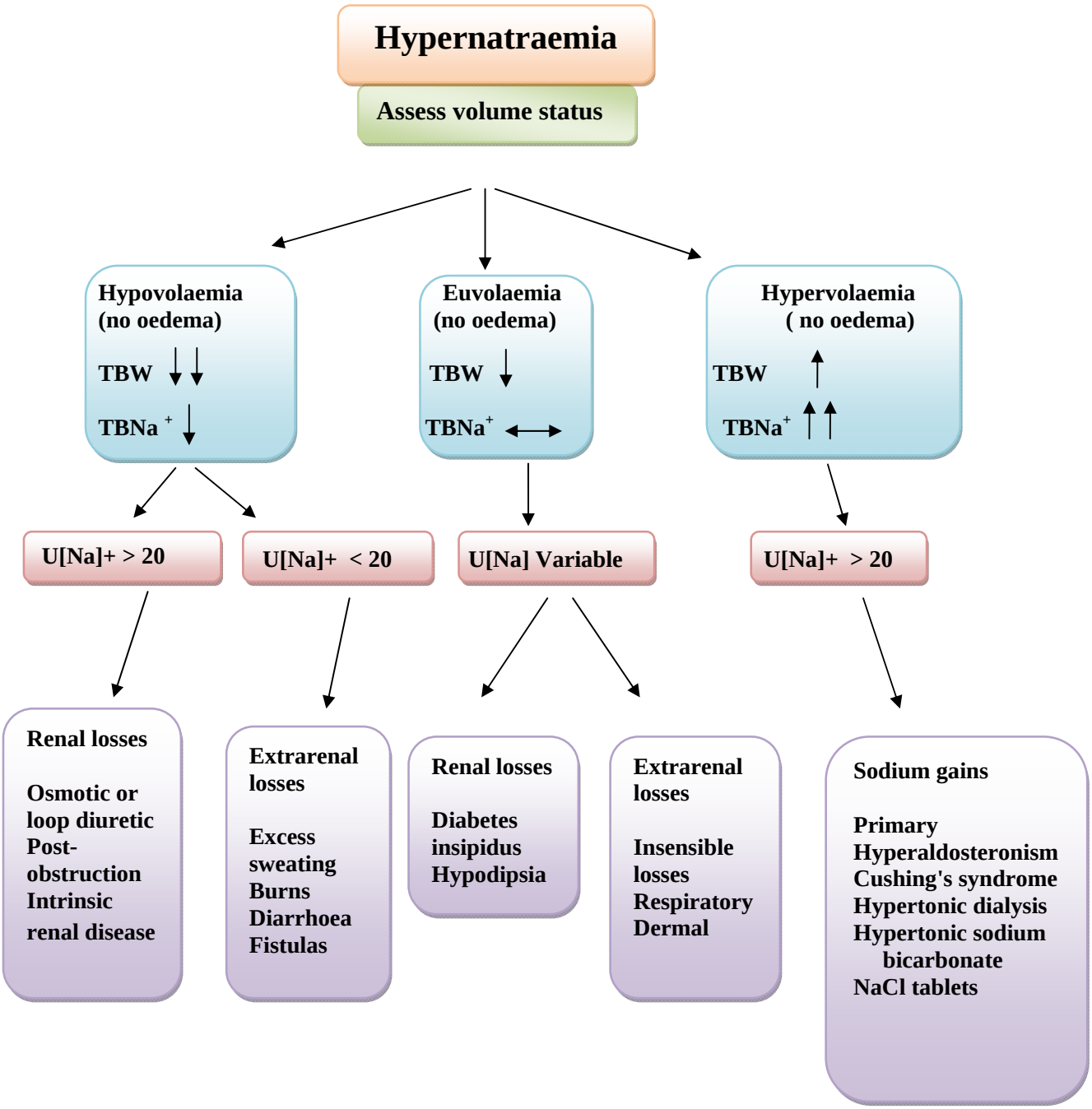
Hypernatraemia
Assess volume status
Hypovolaemia (no oedema)
TBW ↓↓
TBNa+ ↓
Euvolaemia (no oedema)
TBW ↓
TBNa+ ↔
Hypervolaemia (no oedema)
TBW ↑
TBNa+ ↑↑
U[Na]+ > 20
U[Na]+ < 20
U[Na] Variable
U[Na]+ > 20
Renal losses
Osmotic or loop diuretic
Post-obstruction
Intrinsic renal disease
Extrarenal losses
Excess sweating
Burns
Diarrhoea
Fistulas
Renal losses
Diabetes insipidus
Hypodipsia
Extrarenal losses
Insensible losses
Respiratory
Dermal
Sodium gains
Primary Hyperaldosteronism
Cushing's syndrome
Hypertonic dialysis
Hypertonic sodium bicarbonate
NaCl tablets