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HYPERNATREMIA IN CHILDREN AND NEUROLOGICAL DISORDERS, ITS MANAGEMENT

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Annotation

Hypernatremia (HRN), defined as serum sodium >145 mmol/l, represents hyperosmolality. Although it reflects a deficiency of water relative to sodium, total body sodium may be high, normal or low. HRN is mirror image of hyponatremia. However, no large-scale studies have been conducted in the pediatric setting to inform this recommendation. Therefore, this study aimed to report the association between the rate of correction of hypernatremia, neurological outcomes, and all-cause mortality in children.

Keywords: hypernatremia, diarrhoea, neurological deficit, metabolic encephalopathies.

The usual causes of hypernatremia are: dehydration in which water loss exceeds sodium loss and overhydration with hypertonic saline solutions. Hypernatremia is a medical emergency, and if not corrected promptly may lead to permanent brain damage and death.

Clinical features, Hypernatremic dehydration may be a consequence of vomiting or diarrhoea, especially if water intake is restricted. Overzealous correction of hyponatremia causes iatrogenic hypernatremia. Rapid alterations in sodium concentration are much more likely to cause encephalopathy than are equivalent concentrations attained slowly. The symptoms of hypernatremia are referable to the nervous system and include irritability, lethargy progressing to coma, and seizures.

The presence of focal neurological deficits suggests cerebral venous sinus thrombosis.

Diagnosis

Symptomatic hypernatremia develops at sodium concentrations greater than 160 mEq/L (160 mmol/L). EEG shows the nonspecific slowing associated with metabolic encephalopathies. Focal slowing on the EEG or focal abnormalities on examination warrants neuroimaging to look for venous sinus thrombosis. Central diabetes insipidus (DI) causes recurrent episodes of hypernatremia and is typically seen in children with congenital brain malformations (e.g., septo-optic dysplasia sequence) or a history of extensive brain injury. This is a chronic disorder that requires lifelong surveillance.

Management

Rapid water replacement can lead to cerebral edema. The recommended approach is to correct abnormalities of intravascular volume before correcting the water deficit. For children with DI, refer to an endocrinologist who can then assist with sodium monitoring and administration of desmopressin (a synthetic form of antidiuretic hormone). Basic principles:

1. Identify and treat the underlying cause.
2. HR should be corrected slowly (particularly if HR is of unknown duration or chronic) as rapid correction can induce cerebral edema, seizures, permanentneurological damage and death (rate of correction of Na should be <0.5 mmol/l/hour or <12 mmol/l/day). It is usually corrected over 48 hours but over 72 hours if serum Na is > 170 mmol/l.
3. Those with chronic HRN tend to be the least symptomatic and are at higher risk of cerebral edema if rapidly corrected. If serum Na drops fast during correction and the patient is symptomatic, hypertonic saline might be needed (4-6ml/kg of 3% sodium chloride). Acute HRN can be corrected relatively rapidly.
4. Monitor (paired) osmolality and electrolytes in both serum & urine frequently (6 hourly) to make necessary adjustments to IV therapy.
5. Fluids (dioralyte, water or diluted feeds-based on etiology) can be administered orally. However it is advisable to use IV route if HRN is severe or GI intake or absorption is disturbed (e.g. vomiting or diarrhea).

6. Strict I/O chart, twice daily wt and strict control over Na administered in any form.
7. HRN impairs insulin and PTH release and hence patients should be monitored for hyperglycemia and hypocalcaemia during correction period. Hyperglycemia in these patients is usually not treated with insulin as it may precipitate cerebral edema.
8. It is important to keep the possibility of diabetes insipidus in mind if the history or assessment is suggestive, as even hypotonic fluid administrated in error might significantly increase serum sodium levels.

Conclusion, HRN induced osmotic gradient result in water movement out of the cells into ECF (ECF volume relatively well maintained, hence the less evident signs of hypovolemia). This cellular dehydration in brain cells (cerebral dehydration result in local hyperosmolality and reduced brain volume) is responsible for the neurological symptoms seen in HRN.

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