EDITORIAL VIEW

Hyponatremia still remains largely undiagnosed and untreated

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ABSTRACT

Sodium deficiency is one of the most common entities a clinician encounter in daily life. Various different causes and types are there. Appropriate assessment of cause and proper in time management is key as it is most frequent electrolyte disorder and more important is it is also most frequently neglected and over looked one. So timely diagnosis and management decreases morbidity and mortality.

Key words: Hyponatremia; Hyponatremia, Causes; Hyponatremia, Types; Hyponatremia, Management

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Chronic mild hyponatremia is defined as plasma sodium concentration between 125 and 135 mEq/L for more than 72 hours without causing any obvious symptoms. It is most common in ambulatory patients and is without any obvious sign and symptoms in most of patients[1]. Hyponatremia is most common electrolyte imbalance that a clinician faces in his clinical life.[2,3,4,5] A lot of etiological factors are responsible for this.[4] Most common being the syndrome of inappropriate antidiuretic hormone (ADH) secretion.[6] Other factors responsible for this are cerebral salt wasting,[7] chronic kidney disease, and liver and heart failure.[2] Use of drugs like furosemide and mannitol[8] may also contribute. In all these cases the mechanism of regulating fluid and electrolytes is impaired.[9] Detail of types and causes are as below.

A rather simple clinical classification of hyponatremia is based on volume status. Various types are: hypovolemic (decreased total body water with greater decrease in sodium level), euvolemic (increased total body water with normal sodium level), and hypervolemic (increased total body water compared with sodium). As per clinical presentation the terms of 'severe symptomatic hyponatremia' and 'pseudo hyponatremia' are well established entities.

To understand hyponatremia we need to know about plasma osmolality, which is the total concentration of solutes in solvents and it has a vital role in it. The patient may be hypovolemic (decreased total body water with greater decrease in sodium level), euvolemic (increased total body water with normal sodium level), and hypervolemic (increased total body water compared with sodium).[10] There is a very precise regulatory system of plasma osmolality which is maintained by arginine-vasopressin system and thirst. It is also called antidiuretic hormone (ADH) system. ADH affects kidneys and these in response retain water. This results in decrease in serum osmolality. ADH secretion is a direct response to serum osmolality. If osmolality decreases, secretion of ADH also decreases. It results in renal excretion of excess water thus maintaining homeostasis.[11,12]

Clinical features range from mild conventional symptoms to life threatening conditions like seizures, cardiorespiratory distress and cerebral edema. Many different factors impart profound effects on overall quality of patients' life, ranging from lack of awareness among clinicians to lack of targeted treatment.[4] Not only lack of recognizing hyponatremia contributes towards patients ill health but incorrect treatment of

profound hyponatremia also increases morbidity causing osmotic demyelinating syndrome[5] and even mortality.[1] So recognizing the cause and its appropriate treatment is of utmost importance,[3] as limited clinical guidance and lack of appropriate assessment plays a major role.

A brief elaboration of different types of hyponatremia helps both in clinical guidance and assessment. These are:

Hypovolemic Hyponatremia: Head injury and intracranial hemorrhage are important causes that can result in cerebral salt wasting. Sodium is the main salt that is lost in urine. Many different hormones like mineralocorticoid deficiency impart a profound impact on urine osmolality that can raise more than 20 mEq/L. Various central and renal causes are responsible for this. Addison's disease, pituitary dysfunction, hypothalamic failure is the most frequent of them. Many benign and simple diseases like diarrhea and vomiting can result in significant loss of sodium, which may result in dropping urinary osmolality below 20 mEq/L. Other causes like elevated glucose levels and use of mannitol can also result in this type of hyponatremia.

Clinical features in this type of hypovolemia are mainly because of volume depletion. These include vomiting, diarrhea and tachycardia. Laboratory investigation shows elevated 'blood urea nitrogen to creatinine' ratio.[10,12] Treatment generally consists of volume repletion with isotonic (0.9%) saline, occasional use of salt tablets, and treatment of the underlying condition.[12,13] Most common side effect of over hydration can be avoided by monitoring urinary output and keeping it below 110 ml/h.

Euvolemic hyponatremia: In this type plasma volume remains in normal limits but sodium level drops. Most common causes of this are SIADH, hypothyroidism and glucocorticoid deficiency. Diagnosis is made on urinary sodium level which is greater than 20 mEq/L. Along with it low serum uric acid levels and normal blood urea nitrogen to creatinine ratio helps in making diagnosis. Different contributory factors like use of diuretic therapy and low-salt diet can artificially influence the sodium levels. Treatment generally consists of fluid restriction and correcting the underlying cause. Fluid restriction should be limited to 500 ml less than the daily urinary volume.[12]

Hypervolemic hyponatremia: Main causes of this are renal, cardiac and liver dysfunction. It results in retention of fluid and electrolytes. In volume overload states, the effective arterial blood volume is decreased compared with venous volume, resulting in excess ADH secretion. Treatment consists of correcting the underlying cause, sodium and fluid restriction, and diuretic therapy.[12,13]

Severe symptomatic hyponatremia: Rapid fall of plasma sodium levels in quick succession of time results in this type of hyponatremia. It occurs classically when serum sodium levels fall below 120 mEq/L. However it can also occur even in serum sodium at 125 mEq/L. Seizures are main clinical manifestation. This dilutional hyponatremia can be treated by excretion of excess water from body with drugs Furosemide. Other pharmacological agents like vaptans seem to have no role in it.[13] To avoid drastic complication like central pontine demyelination the rate of sodium correction should be 6 to 12 mEq/L in the first 24 hours and 18 mEq/L or less in 48 hours.[11,13] An increase of 4 to 6 mEq/L is usually sufficient to reduce symptoms of acute hyponatremia.[14]

Pseudohyponatremia: There are actual normal sodium levels in this form of hyponatremia. Many different clinical entities like hyperglycemias, hyperproteinemias and use of drugs e.g. mannitol, contributes in it. Laboratory errors also play a part. Patients with form of hyponatremia usually have normal volume status and urinary osmolality is also unchanged.[11,12]

Like all medically ill patients detailed history and physical examination helps in clinical diagnosis of hyponatremia. Even asymptomatic considered patients can have conventional signs and symptoms irritability, nausea and weakness. Conscious level of patients can vary from altered mental status to coma. Frequent laboratory investigations are usually not indicated as diagnosis is usually apparent from the history.[15,11,16,17]

However, to overcome the difficulty various appropriate clinical guidelines are devised by European Society of Endocrinology, European Society of Intensive Care Medicine, European Renal Association and European Dialysis and Transplant Association, which not only help in investigating the cause but also in treating such patients.[4]

Management of hyponatremia is with replacement of sodium, treatment of parent cause and appropriate fluid management (fluid restriction, hypertonic saline).[10] Determining volume status and calculating the total body water deficit are important.[16]

However, it is worth mentioning that in order to

avoid debilitating demyelination not more than 6 to 12 mEq/L per day of sodium replacement is recommended. Various drugs like desmopressin[5], vaptans like tolvaptan[10] (Samsca), conivaptan (Vaprisol) may also be used. Vaptans are vasopressin-receptor antagonists approved for the treatment of hospitalized patients with severe hypervolemic and euvolemic hyponatremia. However, their use in the management of sole hyponatremia is controversial. Various different studies demonstrated that vaptans increase sodium levels in patients with cirrhosis and heart failure.[19]

So early recognition is of utmost importance and for this a clinician must keep in mind this possible derangement in all patients with conventional symptoms like vomiting, diarrhea, tachycardia and other signs and symptoms of volume depletion[10,12] in addition to rule out other possibilities. More so, timely and early appropriate treatment not only saves life but also decreases morbidity.[5]

Conflict of interest: Nil

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