FREQUENCY AND SIGNIFICANCE OF ELECTROLYTE ABNORMALITIES IN PNEUMONIA

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ABSTRACT

To determine the frequency of electrolyte disturbance in pneumonia, we studied 264 hospitalized children with pneumonia for serum sodium and potassium concentration, and plasma osmolality (Posm) on the day of admission. Urine osmalality (Uosm) and urine spot sodium concentration were measured in those who had a serum sodium ≤130 mEq/L. Hyponatremia was found in 27%, hypernatremia in 3.7%. Hypokalemia (serum potassium ≤3.5 mEq/L) in 19 and 2% had hyperkalemia (serum potassium ≥6.5 mEq/L). Of all the hyponatremia, 68% were secondary to syndrome of inappropriate ADH secretion (SIADH) as suggested by a concomitant lowering of Posm ≤280 mosm/kg and increased urinary osmolality and sodium excretion. Hyponatremia was associated with 60% longer hospital stay, two fold increase in complications and the 3.5 times higher mortality compared to that of normonatremia. The above variables were affected further, if hypokalemia coexisted with hyponatremia.

Key words: *Serum electrolytes, Sodium, Potassium, Hyponatremia, Hypokalemia, Plasma osmolality, Pneumonia.*

Electrolyte disturbances especially hyponatremia have been described in a wide variety of acute infections including pneumonia(1). Water retention, fall in serum concentration of chloride and fixed base and diminished osmolality were described in lobar pneumonia in 1920s(2-6). Further studies have shown an increase in plasma volume and extravascular fluid(7), and severe hyponatremia(8) in association with pneumonia. Inappropriate secretion of antidiuretic hormone (SIADH) has been suggested as the likely underlying mechanism for the above changes in fluids and electrolytes(9-10). We have studied frequency and significance of hyponatremia, osmolality changes and potassium abnormalities in children who were hospitalized for pneumonia.

Material and Methods

Included in the study were children who were admitted with a diagnosis of pneumonia to the Pediatric Emergency Unit of Nehru Hospital, PGIMER, between August 1987 to February 1989. Those who had measles, whooping cough and asthma or signs and symptoms suggestive of diarrhea and dehydration, congestive heart failure, meningitis, nephrotic syndrome, nephritis or adrenocortical disorders were excluded. The diagnosis of pneumonia was based on the characteristic clinical symptoms and signs and radiological features,

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viz., presence of alveolar infiltrate and/or consolidation on chest *X*-ray. All the decisions regarding antimicrobials and supportive therapy were taken by the attending physician and were in no way influenced by the study. This was guided by the standard protocol of the unit.

Venous blood was obtained on the day of admission for estimation of serum sodium and potassium (by flame photometery) and plasma osmolality (Posm) (by freezing point depression method). Urine was collected on day 1 and submitted for urinary osmolality (Uosm) and urine spot sodium concentration (Usod) if the serum sodium was ≤130 mEq/L. Blood urea (monoxime method) and creatinine (Jaffe's method) were also measured to rule out any renal dysfunction.

The demographic data (age, sex and nutritional status), length of hospital stay and outcome (graded as recovered fully, recovered after complication or left with sequalae, and died) were recorded in all the cases.

Serum sodium concentration of ≤130 mEq/L was considered as hyponatremia and ≥150 mEq/L as hypernatremia. Hyponatremia was further categorized as normovolemic, hypovolemic or dilutional depending on serum osmolality. Serum potassium concentration of <3.5 mEq/L was labelled as hypokalemia and >6.5 mEq/L as hyperkalemia.

Statistical significance of the comparisons was determined by chi-square, or t-test whichever was appropriate.

Results

Serum sodium concentration: Distribution of 264 study children by age, sex and nutritional status, and serum sodium concentration is shown in *Table I*. Hyponatremia was seen in 71 (27%), and hypernatremia in 19 (3.7%). Severe hyponatremia (S Na^+ ≤125 mEq/L) was seen in 4.5% (12/264). The distribution of hyponatremia or hypernatremia was not related to age, sex, nutritional status and the season of the year (*Table I*).

Of the 71 hyponatremic children, 48 (68%) had dilutional hyponatremia. It was probably secondary to SIADH as all these children had Posm ≤280 mOsm/kg, Usod >20 mOsm/kg, and normal blood urea and creatinine. In 7% (5/71) children hyponatremia was associated with high plasma osmolality above 300 mOsm/kg and normal or low urinary sodium excretion (Usod <20 mEq/L) indicating hypovolemic hyponatremia. In the remaining 25% (18/71) children it was accompanied by normal Posm (281-299 mOsm/kg), urinary sodium excretion and Uosm. Among 183 children with normal serum sodium (131-149 mEq/L), 5.5% had a plasma osmolality >300 mOsm/kg, 43% a Posm ≤280 mOsm/kg while the remainder had normal plasma osmolality.

Serum potassium concentration: Twelve per cent children (32/264) had hypokalemia while 2% (5/264) had hyperkalemia. The occurrence of hypokalemia was not related to child's sex (Boys 13.0%; 26/198, Girls 9%; 6/66) ($p > 0.05$) or age (*Table II*). About 47% (15/32) children with hypokalemia also had hyponatremia; Posm was ≤280 in 8 of them while none had Posm ≥300.

Outcome: As shown in *Table III*, in hyponatremic children on average the duration of hospitalization was prolonged by 60%, occurrence of complication was about two fold and the mortality about 3.5 times higher as compared to normonatremic patients. The mean length of hospital stay was significantly prolonged in children who had hyponatremia with

TABLE III– *Mean ± SE Length of Hospital Stay, Complication Rate and Mortality of Children with Pneumonia with Respect to Serum Sodium (Na^+) and Potassium (K^+) Concentration at the Time of Admission*

Electrolyte status	n	Length of hospital stay (days)	No. with complications (%)	Number died (%)
1. Hyponatremia and normal $K^{\pm}$	56	7.1 ±0.86*	7 (12.5)$^+$	5 (9.8%)$^{\pm}$
2. Hyponatremia and hypokalemia	15	10.0 ±2.1*	4 (26.6)	1 (6.6)
3. Normal Na^+ and hypokalemia	17	6.1 ±1.4	3 (17.6)	2 (11.7)
4. Normal Na^+ and K^+	171	5.5 ±0.39	12 (6.1%)	5 (2.6%)

* $p < 0.05$, t-test, between 1 & 4, 2 & 3, 1+2 & 4, 1+2+3 & 4.

\+ p-ns, χ^2-test, 1 compared to 4 (normal Na^+ and K^+), $p = 0.04$, 1+2 compared to 4, and 1+2+3 compared to 4.

± $p = 0.06$, χ^2-test, 1 compared to 4, and 1+2 compared to 4. $p = 0.035$ χ^2-test, 1+2+3 compared to 4.

TABLE IV–*Details of 13 Patients with Pneumonia who Died*

Age	Hospital stay (days)	Severity of illness	Na^+	K^+	Posm
1½ mo	5	severe	125	6.0	269
2 mo	1	severe*	126	6.3	274
5 mo	6		138	4.0	269
5 mo	3		142	6.5	280
6 mo	2		126	4.9	269
7 mo	10		136	3.2	292
8 mo	1		140	4.0	292
9 mo	1	severe	124	3.9	270
1 yr 5 mo	2	severe	132	2.5	270
1 yr 6 mo	1		132	2.6	262
4 yrs	1		140	4.4	294
11 yrs	8	severe$^+$	122	5.4	264
3 yrs	4	severe	128	4.5	272

Complications **Staph. aureus* septicemia, $^+$ Staphylococcal pneumonia with empyema thoracis.

Discussion

We found that electrolyte disturbances in children with pneumonia in our population were common. The most frequent abnormality was hyponatremia (25%) which was followed by hypokalemia (12%). Other abnormalities like hypernatremia (3.7%) and hyperkalemia (2.0%) were seen in a small number of children. These findings are very much relevant to management of hospitalized pneumonia cases.

The hyponatremia seen in acute infections is not always explained by an absolute water excess or sodium depletion. It seems to represent a new steady state of relative

dilution, as a result of disturbance in the mechanism that regulates volume and contents of body fluid(1). Increased secretion of ADH and increased catabolic process at the cellular level have been postulated as explanations for this phenomenon(1,12). Recent studies by Hannon and Boston have shown that significant intracellular shifts of sodium, chloride and water occur in septic animals(12). These animals had significantly reduced extracellular (ECW) and intracelluar water (ICW) as compared to the controls. Hyponatremia and hyposmolality occurred when these animals received 100% of their fluid as an electrolyte free solution. In 68% of the children in whom hyponatremia was dilutional in nature, above mechanism and SIADH may probably explain the hyponatremia.

It was significant to note that in 7% of children hyponatremia was hypovolemic in nature. In none of the five children with this type of hyponatremia any clinical sign of dehydration was apparent. Yet a state of fluid deficit was present in them as plasma osmolality in all of them was 300 mmol/kg or above, and urinary sodium excretion was below normal. An additional 5.5% of normonatremic children also had a plasma osmolality >300 mmol/kg. Possible causes for the finding could be poor oral intake, high insensible water loss because of fever, and high respiratory water loss due to tachypnea.

The data presented here neither give a precise answer to causal mechanisms of hyponatremia nor the possible state of absolute body sodium contents which may be low, normal or high in presence of hyponatremia. Because of this uncertainty on causal mechanism of hyponatremia and state of body fluids and sodium in a given patient, the therapeutic implications of the hyponatremia remain empiric. It may be reasonable to suggest that giving of excessive amount of free water should be avoided in these patients, as this may lead to very low osmolal concentration of body fluids. However, the value of routine water restriction as suggeted by Shann and Germer(11) remains questionable in face of data in experimental animals that ECW and ICW volume are depleted in septic animals. Increased secretion of ADH in this situation may be a physiological response to ECW volume depletion(12).

Our data suggests that there is the need to individualize fluid therapy in children with pneumonia. Those who have hyponatremia with hyperosmolality need liberal fluids while those with hyposmolality may need fluid restriction. Both these fluid regimens are recommended in the literature(11,14). Advocates of fluids restriction argue that SIADH is associated with increased mortality and poor outcome(11). World Health Organization advocates increased fluid intake in children with pneumonia, because of fear of dehydration and drying up of secretions due to excess water loss(14). This may be applied to children with normal serum sodium levels. Clearly electrolyte free solutions (including N/5 saline in 5% glucose) are not appropriate intravenous fluids for severe pneumonia patients.

We found that hyponatremia and hypokalemia were associated with an adverse outcome. Hyponatremia at admission significantly affected the outcome in terms of prolonged duration of hospitalization, and two fold increase in mortality. The outcome was further affected if hyponatremia was associated with hypokalemia.

Hypokalemia can have deleterious effects on membrane potentials and affect the excitability of cardiac and smooth muscle(15). Thus, severe hypokalemia may

result in life threatening complications such as cardiac arrhythmia, cardiac arrest and respiratory failure. Unexplained sudden deaths have been reported in conditions associated with hypokalemia without any warning signals. In our patients, increased mortality associated with hypokalemia could have been a result of one or more of the above complications. However, no cause and effect relationship can be defined from our data. Nonetheless, it may be prudent to monitor and treat serum potassium disturbances in children with severe pneumonia.

We conclude that regular estimation of serum electrolyte concentration and plasma and urine osmolality is necessary to guide appropriate fluid and electrolyte management of children with severe pneumonia requiring hospitalization. Contrasting recommendations of routine fluid restriction or liberal fluid intake in children with pneumonia can not be generalized. There is a need to investigate the therapeutic benefit if any, of the routine fluid restriction. There is also a need for further studies to evaluate the significance of serum potassium disturbances in children with pneumonia.

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