WATER ELECTROLYTE HOMEOSTASIS IN ACUTE BRONCHIOLITIS

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ABSTRACT

Children with acute bronchiolitis frequently require hospitalization and parenteral fluid therapy. Water retention due to impaired renal water excretion has been described in several pulmonary conditions in children.

We studied 20 infants (3.6 ±2.9 months), hospitalized consecutively for acute bronchiolitis for water and electrolyte changes during the acute stage and compared them to those on recovery. Serum sodium and plasma osmolality, urinary sodium and osmolality were measured in all infants. Ten infants each were assigned alternatively to study body water compartment or renal water handling (water load excretion and free water excretion capacity) on the day of hospitalization and after recovery.

Mean (±SD) value of serum sodium of the infants at admission was 132.7 ± 7.2 mEq/L which increased to 137.1 ± 5.4 mEq/L on recovery (p <0.05). Plasma osmolality chaned from 284 ± 14 mOsm/kg at admission to 294 ± 10 mOsm/kg at recovery (p <0.05). There was a significant decrease in urinary sodium from 54 ± 39 mEq/L to 20 ± 18 mEq/L and urinary osmolality from 415 ± 213 mOsm/kg to 252 ± 204 mOsm/kg at admission and at recovery, respectively. All 10 infants showed significant increase in total body water (mean ± SD; 22.8 ± 7.5 ml/kg) at admission as compared to that at recovery. The total body water (TBW) excess was mainly in extracellular water compartment (16.3 ±3.6 ml/kg). Seven of 10 infants had significant impairment in renal water excretion. Increase in maximum free water clearance of these 7 infants on recovery was 0.69 ± 0.27 ml/min, i.e., 15 times more than that at admission. It is concluded that bronchiolitis of infancy is characterized by water retention which is caused by impaired renal water excretion. In the management of severe bronchiolitis careful attention to fluid therapy is mandatory; liberal fluid therapy may lead to water intoxication.

Key words: *Bronchiolitis, Water electrotyte homeostasis.*

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Acute bronchiolitis is a common disease of lower respiratory tract of infants and young children. The mainstay of therapy of infants hospitalized for acute bronchiolitis have been oxygen and parenteral hydration(l). Oral and nasogastric feeding are generally avoided in severe cases as the high respiratory rate may interfere with feeding while nasogastric tube obstructs the upper airway and increases the likelihood of reflux(l). Moreover, a full stomach may impair diaphragmatic movements. However, there are conflicting recommendations in pediatric textbooks on the amount of parenteral fluid to be adminsitered(2-4). Excess fluid upto 1.5 to 2 times the normal maintenance requirement has been recommended to decrease respiratory mucous viscosity(2). On the other hand judicious use of diuretics or careful fluid restriction has been advised to reverse hypervolemia and thus decrease

lung water(4). The later recommendation stems from the knowledge that the syndrome of inappropriate antidiuretic hormone secretion (SIADH) occurs frequently in several pulmonary conditions including bronchiolitis(1-10). A recent study has shown an increased plasma antidiuretic hormone (ADH) and hyperreninemia with secondary hyperaldosteronism in infants with brorichiolitis(10). These hormonal changes are likely to induce water retention through impaired renal wter excretion without altering serum sodium, as these changes counterbalance each other with respect to renal sodium excretion(4). Thus, a liberal fluid intake may not be advisable in acute bronchiolitis. The optimal fluid regimen in bronchiolitis has not been studied so far. Systemic data on renal water handling and body water changes are needed to guide parenteral fluid therapy in children with acute bronchiolitis.

The objectives of this study were to examine changes in body water compartment and evaluate the renal water handling in infante hospitalized for acute bronchiolitis.

Material and Methods

Included in this study were 20 infants between 1.5-13 months of age consecutively hospitalized with a diagnosis of acute bronchiolitis. The children were brought to the Pediatric Emergency Services of Nehru Hospital, PGIMER, over a period of 3 months (November 90 to January 91). Only moderate and severely distressed children were hospitalized. Bronchiolitis was defined as the first episode of acute wheeze under 2 years of age, preceded by upper respiratory symptoms, accompanied by tachypnea (respiratory rate >60/min), chest indrawing, rhonchi, evidence of hyperinflation of chest on physical examination and supported by typical radiological findings of hyperinflation, patchy atelectasis and peribronchial thickening. No viral study was done. Children with bronchopulmonary dysplasia, heart disease, pneumonia, known bronchial asthma, diarrhea, renal, CNS or any chronic diseases were excluded.

On admission, demographic data and respiratory symptoms and signs were recorded on each infant. Venous blood samples were obtained for serum sodium, potassium, creatinine, blood urea and plasma osmolality estimation, and urine samples were collected for sodium and osmolality from each patient at admission and discharge.

Ten infants each, assigned alternatively, were studied for renal water handling or body water compartments. To study renal water handling, 20 ml/kg 5% dextrose was given by intravenous infusion over a period of one hour. Urine and serum specimens were collected 1 and 3 hours after the beginning of water load. The volume of each collected urine specimen was recorded and the urine was analyzed for osmolality. Urine volume passed over 1 and 3 hours was calculated as percentage of the water load administered, to derive water load excretion.

Free water clearance (ml/min) was determined as urine flow rate-osmolar clearance, where

$$\text{Osmolar clearance} = \frac{\text{Urine osmolality x urine minute vol}}{\text{Plasma osmolality}}$$

For determination of total and extracellular body water compartments 2 ml/kg of a solution containing 0.6 mmol/ml of sucrose and 2-3 uCi of tritiated water was given intravenously. Blood sample was drawn just before and 3 hours after the intravenous

administration of the tracer. Before each estimation, children were weighed carefully with the help of a beam balance (accuracy ± lOg). Extracellular water (ECW) was determined from the distribution volume of sucrose in plasma using the method of Zweens and Frankena(11). The total body water (TBW) was determined from the distribution volume of ^{3}H activity measurement in RBC water by liquid scintillation counting(12). Details of both the methods have been published recently(13). Intracellular water (ICW) was calculated as the difference between TBW and ECW. All results relating to body water contents were expressed as ml per kg of body weight. The difference between body water contents at admission and discharge was termed as body water excesses.

Serum and urinary sodium were measured by flame photometry, blood urea by monoxime method and serum creatinine by Jaffe's method. Plasma and urinary osmolality were measured by freezing point depression method.

All the infants received humidified oxygen and steam inhalation(14). Intravenous fluids were given as N/5 saline in 5% glucose, to all the children as they were having marked respiratory distress and inability to feed at admission. The fluids were started at a rate of 100-120 ml/kg/day and adjusted according to their need to maintain normal hydration. Oral feeds were started as and when an infant was able to feed, and free of chest retraction.

Descriptive statistics (percentage, mean, standard deviation) were applied for various characteristics of study population and to the study variables. Changes in studied variables between admission and recovery were tested by paired 't' test. Statistical significance was indicated by $p < 0.05$.

Results

The mean age of 17, boys and 3 girls studied was 3.6 ± 2.9 months. There was no difference in the age and sex distribution and frequency of various respiratory symptoms and signs in the infants assigned to body water or renal water excretion study. The mean duration of upper respiratory symptoms (cough, runny nose, fever) before admission was 3.6 ± 2.2 days. Of 20 infants, 16 had flaring of alae nasi, 18 had inability to feed, 14 had marked subcostal, intercostal recessions and 8 had temperature >38°C. The mean respiratory rate at the time of admission was 79 ± 8 per minute. Hyperinflation on X-ray chest was seen in all the cases (this was an essential criteria for inclusion in the study).

The mean ± SD values at admission and discharge of serum sodium, plasma osmolality, urinary sodium, urinary osmolality are shown in *Table I*. At admission, mean serum sodium and plasma osmolality were comparatively lower than those at discharge. Seventeen of the 20 infants showed an increase in values of both the parameters at discharge. On the other hand, urinary sodium and osmolality at admission were in above normal range, and higher than those at discharge. Almost all the infants showed a fall in both the parameters towards normal range at the time of discharge *(Table I)*. Serum potassium, urea and creatinine values of all infants, both at admission and discharge were normal.

Results of total body water (TBW) and extracellular water (ECW) compartments estimation are shown in *Fig. 1*. The TBW excess during the acute stage was 23 ± 17.5 ml/kg, 72% of this excess was due to ECW increase. The ECW excess was 16 ml/kg while ICW excess was 6 ml/kg *(Table I)*.

TABLE I– *Mean (±SD) Values of Serum Sodium, Plasma Osmolality, Urinary Sodium, Osmolality, TBW, ICW, Maximum Free Water Clearance and Water Load Excretion at Admission and Discharge.*

S No.	Parameters	n	Admission (Mean±SD)	Discharge (Mean±SD)	Difference	'p' value*
1.	Serum sodium (mEq/L)	20	132.7±7.2	137.2±5.4	3.8±8.5	>0.05
2.	Plasma osmolality (mOsm/kg)	20	284±14	294±10	13±14	<0.001
3.	Urinary sodium (mEq/L)	20	54±39	20±18	34±42	<0.01
4.	Urinary osmolality (mOsm/kg)	20	415±213	252±205	163±262	<0.05
5.	TBW (ml/kg)	10	699±31	676±33	23±7	<0.001
6.	ECW (ml/kg)	10	348±32	332±30	16±4	<0.001
7.	ICW (ml/kg)	10	351±36	345±41	6±8	<0.01
8.	Max. free water clearance (ml/min)	10	0.12±0.26	0.35±0.45	0.25±0.54	<0.05
9.	Water load excretion in 3 h (%)	10	50.3±17.6	78.0±20.5	26.4±18.85	<0.01

* Paired 't' test

Data on maximum free water clearance and excretion of water load at the end of 3 hours is shown in *Fig. 2.* Seven out of ten infants showed a relative impairment of renal free water clearance and nine showed decreased excretion of water load in the acute stage as compared to that after recovery. In these infants, maximum free water clearance was about 15 times less and water load excretion was 44% less at the time of admission than at discharge *(Table I).*

Discussion

Hyponatremia and SIADH has been known to occur in acute bronchiolitis but there had been no detailed study of renal water handling and body water compartment in this condition. However, studies have shown abnormal renal water handling in children with acute asthma(8) and adults with pneumonia(9) which are known to be associated with hyponatremia and SIADH. To the best of our knowledge, this study has for the first time shown a reversible defect of water excretion and increased body water in children with acute bronchiolitis. All the 10 infants, in whom body water estimations were done, had increased total body water at admission, whereas 7 out of 10 infants studied for renal water handling had impaired free water clearance and decreased excretion of a water load. Sequential changes in urinary sodium excretion and osmolality also suggested an impaired free water excretion in acute bronchiolitis. Thus it may be assumed that the increase in body water was a consequence of impaired renal water excretion.

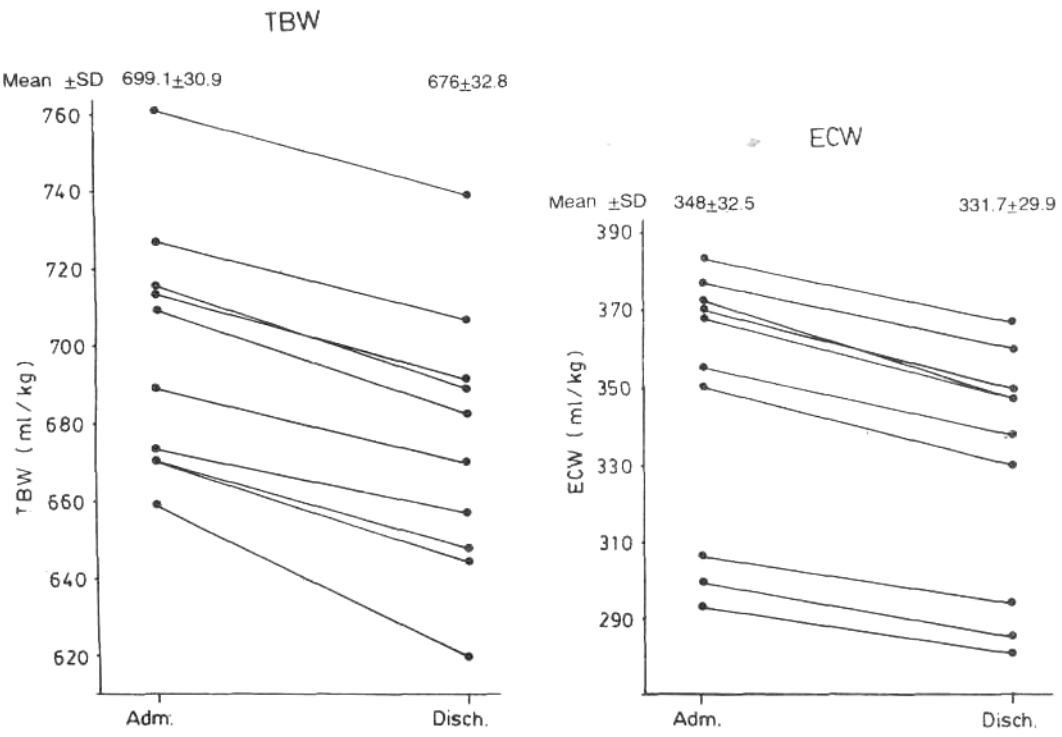

Fig. 1. Changes in TBW and ECW from admission to discharge.

Rivers *et al.*(6) studied 4 children with bronchiolitis and found that 3 children had hyponatremia and an increase in body weight upto 250 g during the acute stage. The ADH levels were high in 2 children in whom it was measured. They postulated that excessive secretion of ADH caused retention of water (reflected in increased body weight) which in turn led to hyponatremia. Gozal *et al*.(10) studied 23 consecutive children with bronchiolitis, 22 of them had evidence of SIADH. Plasma ADH levels were high at admission in all 22 children. These children showed a decrease in body weight by 1.9 ± 1.4% on recovery. The authors postulated that excessive fluid retention secondary to increased ADH secretion was the cause of weight gain in the acute stage. Thus, both the above referred studies had indirectly documented an increase in body water in those patients with acute bronchiolitis who had hyponatremia and/or SIADH. Our study offers for the first time, direct evidence of increased body water (mostly as extracellular water) in acute bronchiolitis. We found hyponatremia in eight of the 10 infants, in whom TBW excess was documented. For practical purposes, it may, therefore, be concluded that a significant increase in body water is likely if hyponatremia and other typical findings of SIADH are present in acute bronchiolitis.

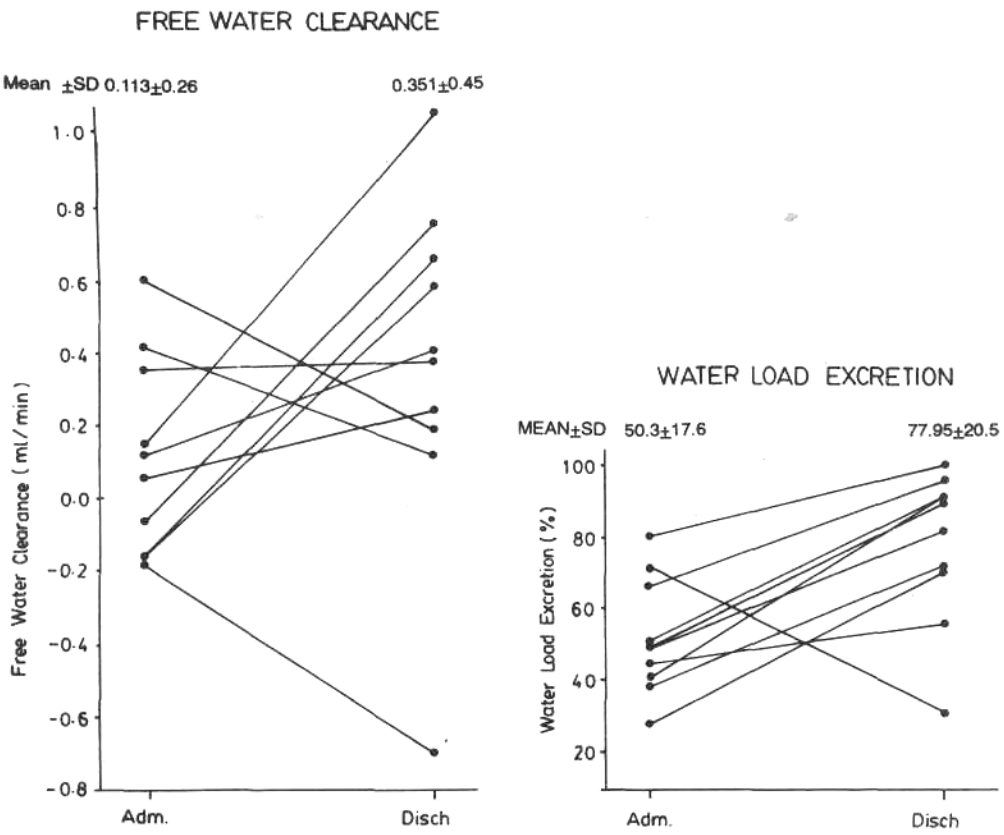

Fig. 2. Changes in free water clearance and water load excretion from admission to aiscnarge.

The impaired renal water excretion, apparently was not due to a renal disease or dehydration, congestive cardiac failure, cirrhosis of liver, drugs like catecholamines and thyroid or adrenal dysfunction because sample selection and exclusion criteria precluded these. Absence of significant renal disease could be further assumed from normal blood urea and creatinine values that our patient had. SIADH was probably the most likely cause of impaired renal water handling in the study patients. Decreased free water clearance and water load excretion following an acute water load are characteristic features of SIADH and have been used as a diagnostic test. Singleton *et al.* (8) had shown decreased free water clearance and water load excretion in 5 children with bronchial asthma during the acute stage; all of them had, high ADH levels while none had hyponatremia. Dreyfuss *et al.(9)* had documented marked impairment of renal water excretion in nine adult patients with pneumonia, all of them had high ADH levels during the acute stage without hyponatremia or low plasma osmolality. They postulated that impaired renal water excretion was due to resetting of the

vasopressin osmostat. Other authors have also documented SIADH on the basis of elevated plasma ADH levels in the absence of apparent change in serum and urinary electrolyte and osmolality(6,9,10). We may not be able to say conclusively as to whether excessive ADH was responsible for impaired renal water excretion. Nonetheless, it may be said that impaired renal water excretion was the most likely cause of increased body water in acute bronchiolitis.

We conclude that acute bronchiolitis is commonly accompanied by water retention, and impaired renal water excretion is its most likely cause. The changes in body water may not necessarily manifest as typical SIADH or dilutional hyponatremia. In the management of hospitalized cases of bronchiolitis, therefore, restricted fluids should be given during the first 1-2 days, liberal, fluid therapy may run the risk of water retention and perhaps increase the risk of water intoxication and pulmonary edema.

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