

Frequency and Significance of Potassium Disturbances in Sick Children

S. Singhi
S. Gulati
S.V.S.S. Prasad

Potassium is known to have profound effects on cardiac, skeletal and smooth muscle(1,2). Hypokalemia maybe associated with serious adverse effects(1,3,4), and increased mortality and morbidity(4-6). Likewise, hyperkalemia may also be potentially fatal. While there are a few studies available regarding the frequency of potassium disturbance in hospitalized adult patients(7-9), similar studies in children are lacking(10-12). The objectives of this study were to determine the frequency and the prognostic significance of hypokalemia and hyperkalemia in sick children.

Subjects and Methods

This prospective study included 727 acutely ill children upto 12 years of age who attended the Pediatric Emergency Services

From the Department of Pediatrics, Postgraduate Institute of Medical Education and Research, Chandigarh 160 012.

Reprint requests: Dr Sunit Singhi, Additional Professor, Pediatric Emergencies and Intensive Care, Department of Pediatrics, PGIMER, Chandigarh 160 012.

Received for publication: April 27,1992;

Accepted: October 3, 1993

during the summer months of May, June, July and August and winter months of November, December, January and February. On each day first five children who required hospitalization were included in the study irrespective of their primary diagnosis or severity of the illness. The study was approved by the Institute Ethics Committee.

Venous blood samples were obtained at the time of initial evaluation and analysed for serum sodium and potassium by flame photometry, blood urea by monoxime method and serum creatinine by Jaffes method. Age, sex, hospital stay, primary diagnosis and the outcome were also recorded. Hypokalemia and hyperkalemia were defined as serum potassium concentrations 3.5 mEq/L or less and 6.0 mEq/L or more, respectively. Serum sodium concentration more than 145 mEq/L was defined as hypernatremia and 130 mEq/L or less as hyponatremia. Statistical significance of the frequency distribution was determined by 'chi-square' test, and of continuous data by one way analysis of variance (ANOVA) or unpaired t-test depending on the number of the groups. Odds ratio and their 95% confidence interval for the risk of mortality with various electrolyte abnormalities was calculated by the method applicable to incidence studies(8).

Results

Of the 727 children studied, 531 (73%) were boys and 197 (27%) girls. Two hundred and twenty eight children were under one year of age, 1% between 1-2 years, 146 between 3-5 years and 157 were 6 years and above. Hypokalemia was found in 101 (13.9%) children and 39 (5.4%) had hyperkalemia while 587 children were normokalemic. The hypokalemic, hyperkalemic

and normokalemic children were similar with regards to either the age or sex distribution. The common underlying diseases associated with hyperkalemia were pneumonia (20%), diarrhea (30%), and meningoen- cephalitis (1%) (*Table I*). The highest incidence of hypokalemia within a disease category was seen in children with congenital heart disease who were on diuretic therapy for congestive cardiac failure, followed by diarrhea. Of the 101 hypokalemic children 52 had associated hyponatremia, while 47 were normonatremic and the remaining two had associated hypernatremia.

There was no statistically significant difference in the mean \pm SEM duration of hospital stay in the hypokalemic (6.7 ± 0.5 days) or hyperkalemic (6.7 ± 0.9 days) children as compared to normokalemic children (5.9 ± 0.1 days) ($p > 0.05$, ANOVA). The mortality rate among hypokalemic (15/101, 15%) as well as hyperkalemic

children (4/39, 10.3%) was higher than that of normokalemic children (26/587, 4.4%). The odds ratio for mortality with hypokalemia was 3.53 (95% confidence interval 2.52-4.95, $p < 0.001$) and with hyperkalemia 2.32 (95% confidence interval 0.85 - 6.3, $p = 0.21$). A significant increase in the mortality was seen with worsening of hypokalemia ($p = 0.0243$, *Table II*).

Presence of concomitant hyponatremia with hypokalemia perhaps further aggravated the risk of mortality. The mortality among hyponatremic-hypokalemic patients was 19.2% (10/52) in contrast to 8.5% (4/47) among normonatremic-hypokalemic patients (odds ratio 2.26, 9.5% confidence interval 0.76-6.73). However, hypokalemia had a clear independent influence on the mortality; the mortality among 52 hypokalemic-hyponatremic patients being significantly higher than that of 7.5% (12/159) among normokalemic-hyponatremic patients (odds

TABLE I—Incidence of Hypokalemia and Hyperkalemia by Diagnostic Categories

Diagnosis	n	Hypo K ⁺ (n=101)	Hyper K ⁺ (n=29)	Normo K ⁺ (n=587)
1. Diarrhea	129	31 (24.0)	7 (5.5)	91 (70.5)
2. Pneumonia	161	20 (12.4)	11 (6.8)	130 (80.7)
3. Meningoencephalitis	75	10 (13.3)	2 (4.0)	62 (82.7)
4. Septicemia	47	4 (8.5)	3 (6.4)	40 (85.1)
5. Heart disease	12	5 (38.5)	2 (15.4)	6 (46.1)
6. Asthma	54	3 (5.4)	2 (4.0)	51 (91.0)
7. Acute renal failure	41	2 (4.9)	1 (2.4)	38 (92.7)
8. Hepatitis	40	6 (15.0)	1 (2.5)	33 (82.5)
9. Nephrotic syndrome	16	3 (18.7)	2 (12.5)	11 (68.8)
10. Miscellaneous	149	17 (11.4)	8 (5.4)	124 (83.2)

Figures in parentheses indicate percentages.

BRIEF REPORTS

TABLE II—Mortality Rates Among 101 Hypokalemic Patients by Serum Potassium Concentration

K ⁺ -concentration (mEq/L)	n	Mortality* (%)
≤2.5	13	5(38.5)
2.6 - 3.0	31	5(16.1)
3.1 - 3.5	57	5 (8.8)

χ^2 - 7.436, *P=0.0243

ratio 2.55, 95% confidence interval 1.17-5.55, p=0.033).

Discussion

It is evident from this study that the disturbance of serum potassium were fairly frequent among sick children and these were associated with an increased mortality. Diarrhea and pneumonia were the commonest underlying diseases associated with hypokalemia as well as hyperkalemia. The observation probably reflects the frequency of these diseases in our population. Published data from our country on a similar study-population are not available for comparison.

It is said that most of the clinically relevant hypokalemic states occur because of net loss of potassium from the body(2). Extrarenal potassium loss due to diarrhea, and the renal loss due to an underlying renal disease or diuretic therapy to patients with heart disease could have been the cause of hypokalemia in 40% of our patients. Hypokalemia in patients with pneumonia, and meningoencephalitic illness needs more understanding. Both of these illnesses are associated with inappropriately high circulating antidiuretic hormone (ADH) levels(12,13). Apparently, ADH promotes distal tubular potassium secretion by mecha-

isms independent of urine flow(14). Finally, stress of the acute illness causes an endogenous catecholamine release which can induce cellular uptake of potassium by indirectly stimulating potassium ATP-ase, and cause hypokalemia(15,16). Whether these mechanisms contributed to potassium wasting in patients with pneumonia and meningitis is unclear, as urinary and intracellular potassium were not measured.

Hypokalemia was associated with increased risk of mortality which correlated with the severity of the disturbance. Hyponatremia was an additional risk factor in these cases. Potassium has an important role in regulating 'biologic electricity'(2). Derangements in potassium homeostasis affects body's bioelectric processes, and can cause potentially life threatening consequences like cardiac arrhythmias, cardiac arrest, muscular paralysis, respiratory failure and paralytic ileus(1,3,4,9,10). Although in our cases, cause of death was attributed to primary disease and above mentioned complications were not systemically recorded, it is possible that some of these might have contributed to the mortality.

We conclude that hypokalemia and hyperkalemia occur frequently in acutely ill children and is associated with a poor outcome. Regular estimation of serum potassium concentration and appropriate management of hypokalemia in sick children hospitalized for emergency care must receive due attention.

REFERENCES

1. Knochel JP. Neuromuscular manifestations of electrolyte disorders. *Am J Med* 1982, 7: 51-535.
2. Brem AS. Disorders of potassium homeostasis, *Pediatr Clin North Am* 1990, 37: 419-427.

3. Cannon P. Recognizing and treating cardiac emergencies due to potassium imbalance. *J Cardiovasc Med* 1983, 4: 467-476.
4. Frank BS. Hypokalemia following fresh water submersion injuries. *Pediatr Emerg Care* 1987, 3: 158-159.
5. Ionescu-Torgo"iste C, Mincu I. Danger of inadequate administration of potassium. *Med J Aust* 1974, 2: 305-306.
6. Wareham DV, Deligani S, Brandt G. Utilization review of intravenous potassium imbalance. *Hosp Pharm* 1978, 13:650-671.
7. Paice BJ, Paterson KR, Onyanga-Omara F, Donnelly T, Gray JMB, Lawson DH. Record linkage study of hypokalemia in hospitalized patients. *Postgrad Med J* 1986, 6: 187-191.
8. Newmark SR, Dluhy RG. Hyperkalemia and hypokalemia, *JAMA* 1975, 231: 631-633.
9. Kruse JM, Carlson RW. Rapid correction of hypokalemia using concentrated intravenous potassium chloride infusions. *Arch Intern Med* 1990, 150: 613-617.
10. Schaber DE, Uden DL, Stone FM, Singh A, Katkov H, Bessinger FB. Intravenous potassium supplementation in pediatric cardiac surgical patients. *Pediatr Cardiol* 1985, 6: 25-28.
11. MaloneDR,McNamaraRM,MaloneRS, Fleisher GR, Spivey WH. Hypokalemia complicating emergency fluid resuscitation in children. *Pediatr Emerg Care* 1990, 6: 13-16.
12. Dreyfuss D, Leviel F, Paillard M, Rahmani J, Coste F. Acute infectious pneumonia is accompanied by a latent vasopressin-dependent impairment of renal water excretion. *Am Rev Resp Dis* 1988, 138: 583-589.
13. Kaplan SL, Feigin RD. The syndrome of inappropriate secretion of antidiuretic hormone in children with bacterial meningitis. *J Pediatr* 1978, 92: 758-761.
14. Field MJ, Stanton BA, Biebisich GH. Influence of ADH on renal potassium handling. A micropuncture and micropertusion study. *Kidney Int* 1984,25: 502-511.
15. Bromen MJ, Brown DC, Murphy MB. Hypokalemia from P₂-receptor stimulation by circulating epinephrine. *N Eng J Med* 1983, 309: 1414-1419.
16. Their SO. Potassium physiology. *Am J Med* 1986: 80 (Suppl. 4A): 3-7.

Defective Opsonophagocyte Function in Newborns Studied by Luminol Dependent Chemiluminescence

S.S. Deo
A.C. Kapadia
S.M. Merchant

The ability of polymorphonuclear cell (PMNC) to mount a normal respiratory

burst is dependent upon both humoral (opsonic) and cellular (oxidative) metabolic factors and defects in either of these

From the Department of Immunology, Aditya Pediatric Research Laboratory, Bai Jerbai Wadia Hospital for Children, Parel, Bombay 400 012.

Reprint requests: Dr S.S. Deo, Immunology Research Laboratory, Bai Jerbai Wadia Hospital for Children, Parel, Bombay 400 012.

Received for publication: October 17, 1993;

Accepted: October 3, 1993