

HYPOKALEMIA IN A PEDIATRIC INTENSIVE CARE UNIT

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Objective: To examine the frequency, severity, risk factors and mortality of hypokalemia, and efficacy of therapy used for its correction. **Design:** Descriptive, retrospective analysis. **Sample:** 290 patient records admitted consecutively to a Pediatric Intensive Care Unit (PICU) over a period of one year. **Results:** Forty three (14.8%) patients had 54 episodes of hypokalemia. Predisposing factors included the nature of primary disease (renal disease 19%, septicemia 19%, acute diarrhea 14%, heart disease with congestive failure, and meningoencephalitis 12% each), malnutrition (weight for age less than 80% in 72%) and therapy with drugs (diuretics, corticosteroids and antiasthma drugs). For correction of hypokalemia all the patients received 4-6 mEq potassium per 100 ml of intravenous fluids (slow correction). Seven patients (9 episodes), with ECG changes of hypokalemia, also received infusion of 0.3 mEq potassium/kg/hour till ECG became normal (rapid correction). Normal potassium level was achieved in all nine episodes where rapid correction was given, and in 40 of 45 episodes which received slow correction. The overall mortality among patients with hypokalemia (25.6%, 11/43) was significantly higher than that among the remaining PICU patients (10.9%, 27/247; odd's ratio 2.34; 95% confidence interval 1.3-4.2) ($p < 0.05$). All the patients receiving rapid correction survived. **Conclusions:** Hypokalemia is a common problem among PICU patients. Early detection through regular monitoring and rapid correction may help in improving the outcome.

Key words: Hypokalemia, Electrolytes, Electrolyte imbalance, Intensive care units, Potassium.

POTASSIUM disturbance especially hypokalemia, is known to occur in a number of patients during hospitalization. Hypokalemia can have profound effects on electrical activity in cardiac, skeletal and smooth muscle. If severe, these may result in life threatening conditions like cardiac arrhythmias, cardiac-arrest, respiratory failure, muscular paralysis and paralytic ileus (2-4). Hypokalemia appears to be one of the most

common electrolyte disturbances in sick children. Studies addressing its incidence and outcome are few (5-10) and chiefly from developed countries. The present study describes our experience with hypokalemia in children admitted to the Pediatric Intensive Care Unit (PICU) with reference to its frequency, severity, associated risk factors, mortality and efficacy of the therapy used for its correction.

Subjects and Methods

This retrospective study included hospital records of 290 sick children up to 12 years of age, who were admitted to the PICU of our Institute from January 1993 to December 1993. The patients were routinely monitored for serum and urinary electrolytes depending upon the clinical need. Serum and urinary potassium were measured by flame photometry. Hypokalemia was defined as 'Serum potassium concentration less than 3.5 mEq/L'. It was diagnosed if a repeat estimation confirmed the concentration. Data on all those patients, who had hypokalemia documented on at least one occasion during their PICU stay, was analyzed further. Details of patients regarding their age, sex, weight, diagnoses, clinical course, and outcome were obtained from the records. Details of biochemical parameters, *i.e.*, electrolytes, acid-base status and renal function parameters, along with ECG and details of treatment were noted. Hypokalemia was graded as mild if serum potassium ranged between 3.0-3.4 mEq/L, moderate if between 2.0-2.9 mEq/L and severe if <2.0 mEq/L. Urinary potassium loss was considered excessive if it was >10 mEq/L. All the patients received slow intravenous correction in form of increased potassium content of intravenous fluids to 40 to 60 mEq/L. Patients with ECG changes characteristic of hypokalemia received rapid correction with concentrated potassium chloride solution (200 mEq/L) under continuous ECG monitoring (11). Rapid infusion was given at a rate of 0.3 mEq/kg/h until normalization of ECG findings. Results were analyzed as percentage distributions, by chi-square test, and calculation of odds ratio as applicable to incidence studies (12).

Results

Fifty four episodes of hypokalemia were documented in 43 (14.8%), out of 290

children admitted to PICU. Only 9 patients had more than one episode of hypokalemia. Four out of 54 episodes of hypokalemia (7.4%) were of severe grade, 37 (68.6%) moderate and 13 (24%) episodes mild. Of 43 patients, 29 (67.4%) were boys and 14 (32.6%) girls. Eighteen children were under 1 year of age, 14 between 1-5 years and 11 were above 5 years of age.

Most cases of hypokalemia belonged to diagnostic categories of renal failure (19%), septicemia (19%), diarrheal dehydration (14%), heart disease with congestive cardiac failure and meningoencephalitis (12% each). The frequency of hypokalemia was highest with renal disease, followed by septicemia, and acute bronchial asthma (Table I). Other causes included diabetic ketoacidosis, hepatic encephalopathy and poisonings.

TABLE I-Frequency of Hypokalemia in PICU

Diagnosis	Number of patients	
	Total (n=290)	Hypokalemia (n=43)
Pneumonia	49	3 (6.1)
Septicemia	35	8 (22.8)
Diarrhea with dehydration	33	6 (18.1)
Acute renal failure	32	8 (25)
Heart disease		
With CCF	27	5 (18.5)
Meningoencephalitis	27	4 (14.8)
Other neurological causes	27	1 (3.7)
Acute severe bronchial asthma	20	4 (20)
Upper airway obstruction	7	1 (14.3)
Others*	33	4 (12.1)

Figures in parentheses indicate percentages.

*Include diabetic ketoacidosis in 2, hepatic coma in 1, and acute iron poisoning in 1 case.

Poor oral intake (with inability to replace adequate potassium orally) in 15 (27%) patients was the most important predisposing factor prior to hospitalization. In the hospital, presumably, the use of medications known to predispose hypokalemia was most important. These included diuretics in 11 (20%); corticosteroids in 6 (11%), and β -agonists in 7 (13%). Excessive potassium loss in urine was noted in 13 (24%) patients who either had renal disease or were on diuretic therapy. Excessive gastrointestinal (GI) losses presumably contributed to 9 (16.6%) episodes; upper GI losses in 4 and lower GI losses in 5. No apparent cause could be found in 32% episodes. Severe hypokalemia was always associated with more than one predisposing factor. Drug related hypokalemia was mostly mild to moderate grade. On analyzing nutritional status of the patients in relation to hypokalemia, 31 out of 43 (72%) had malnutrition (weight less than 80% of expected weight).

The relationship between serum potassium and ECG changes was not consistent.

Only 9 episodes of hypokalemia were associated with ECG changes. All of them were given rapid correction under continuous ECG monitoring. Pre-infusion serum potassium in these patients ranged between 1.8-2.8 mEq/L. Typical ECG changes were flat or absent T-waves (in 8), ventricular premature beats, bigemini and supra-ventricular tachycardia (in 2 each). Following 1-3 corrections of potassium ECG became normal in all, and post-correction serum potassium ranged between 2.5-3.4 mEq/L. Serum potassium returned to normal in all of them over next 4-144h (median 48 h). Correction of hypokalemia was achieved in 40 of 45 episodes receiving slow correction only (Table II).

The mortality in hypokalemic patients was 25.6% (11/43), in contrast to 10.9% (27/247) among remaining PICU patients (odds ratio 2.34; 95% confidence interval 1.3-4.2; $p < 0.05$). It is noteworthy that there was no death among 7 patients who received rapid correction while 31% (11/36) of patients on slow correction died ($p < 0.05$). The overall mortality among patients whose hypokalemia

TABLE II-Hypokalemic Episodes in Relation to Management and Mortality

Degree of hypokalemia (mEq/L)	N	Potassium infusion rate (mEq/L of fluid)			Rapid correction	Correction achieved	Duration of correction (mean \pm SD) h	Mortality
		40	50	6				
Severe <2.0	4	1	2	1	4	4	19.8 \pm 5.8	-
Moderate 2.0-2.4	16	2	14	-	3	14	46.9 \pm 37.6	5
2.5-2.9	21	17	4	-	2	18	48.6 \pm 35.6	4
Mild 3.0-3.4	13	13	-	-	-	12	30.9 \pm 14.81	2
All	54	33	20	1	9	48	-	11

*Infusion of 0.3 mEq/kg/h of a 200 mEq/dl solution of potassium chloride till ECG became normal.

got corrected was 13.5% (5 out of 37), in contrast to 100% (6/6) in those where hypokalemia persisted ($p < 0.05$). Of the later six, only two had cardiac arrhythmia terminally. Both these patients had serum potassium between 2.1 to 2.5 mEq/L. Other 4 patients died because of primary illness (one each because of staphylococcal septicemia with shock and disseminated intravascular coagulation, Gram negative septicemia with acute renal failure, cardiomyopathy and cardiogenic shock, and acute iron poisoning); their serum potassium level ranged between 2.4-3.0 mEq/L. All the five deaths among patients whose serum potassium had returned to normal were due to their primary illness (pneumonia with respiratory failure—2, meningococemia with meningitis, meningitis with ventriculitis, and post-varicella meningo-encephalitis with pericarditis—one each).

Discussion

Our results show that hypokalemia is common among acutely ill children and is associated with a significantly higher mortality. Patients with underlying renal disease, septicemia, bronchial asthma, heart disease with congestive cardiac failure, severe diarrhea, and meningoencephalitis were most likely to show evidence of hypokalemia. The apparent cause for development of hypokalemia in most patients was a loss of potassium from the body through GI or urinary tract (8-10), either because of underlying disease process, or because of associated use of drugs such as diuretics, glucocorticoids or mineralocorticoids (10) and antiasthma medications (13). In one patient with diabetic ketoacidosis hypokalemia could be attributed to correction of acidosis (14) and use of insulin (15). In seriously ill patients hypokalemia could also be caused by massive endogenous release of epinephrine

because of stress of the illness (16). High levels of circulating epinephrine cause a shift of potassium from extra cellular fluid to intracellular fluid which might have contributed to hypokalemia. In patients with acute respiratory distress secondary to pneumonia, congestive cardiac failure, and meningo-encephalitis, associated inappropriate secretion of ADH may have an important role in development of hypokalemia. It is suggested that ADH acts through some mechanism independent of urine flow (17) and causes increased secretion of potassium in the distal tubules (18).

We found that hypokalemia was associated with significantly increased risk of mortality. This is in agreement with our previous study in patients admitted to emergency services (5). In two patients mortality could be directly attributed to fatal cardiac arrhythmias caused by severe hypokalemia. Potassium ion is important in regulating 'biologic electricity' and hypokalemia can cause potentially life threatening complications like cardiac arrhythmias or cardiac arrest and muscular paralysis (8-10). Hypokalemia could have contributed to but may not be causative in death of another nine patients. All these patients had severe disease with multisystem involvement, and had moderate hypokalemia. Correction of hypokalemia was however, not accomplished in six of these patients. No significant association was found between associated malnutrition and mortality among hypokalemic patients.

Management of hypokalemia depends upon its severity and underlying condition. Some authors do not recommend treatment of mild hypokalemia (9), while other favor oral supplements (10,19) or rectal administration of potassium chloride solution (20). Intravenous potassium infusion is generally recommended for

severe hypokalemia (9,10,19,20). Even here there is no unanimity regarding concentration of potassium solution to be used and the rate and duration of therapy. The concentration of solution used in adults has ranged from 20-480 mEq/L(21) and the recommended rate of infusion from 0.2-1 mEq/kg/h(19,21). We, for past several years have used higher potassium content of maintenance IV fluids (4-6 mEq/ dl) to correct mild to moderate hypokalemia. In addition to the above, rapid correction with concentrated potassium chloride (20 mEq/ dl) at rate of 0.25 to 0.3 mEq/kg/h is attempted if ECG changes characteristic of hypokalemia are present(11). Oral supplements are used only as a preventive measure in patients predisposed to hypokalemia such as those on diuretic therapy. Using these guidelines hypokalemia correction could not be achieved in 6 patients although they were receiving 4-5 mEq potassium/kg/ day. None of these patients had any ECG abnormality known to be associated with severe hypokalemia. It may be speculative to comment as to whether achieving normokalemia in these patients through rapid correction would have helped in their survival. However, it is noteworthy that all those patients who received rapid correction survived even though all had characteristic ECG abnormalities and four of them had a serum potassium <2 mEq/L. In view of the above and absence of consistent relationship between serum potassium concentration and ECG changes, we feel that rapid correction of hypokalemia should be used more often in treatment of moderate and severe hypokalemia even in absence of ECG abnormalities. Rapid correction has been shown to be safe and useful in hypokalemic adults(21) and children with ECG abnormality(1).

Our findings suggest that regular monitoring, of serum potassium, along with

supportive ECG monitoring, is useful for proper management of PICD patients. Rapid correction of hypokalemia may be life saving.

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NOTES AND NEWS

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