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## Hypokalemic Paralysis

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Hypokalemia is a relatively common problem and may be due to gastrointestinal losses (infectious diarrhea, vomiting, laxative abuse)(1,2) or renal losses (diuretic abuse, renal tubular acidosis). It is usually defined as serum potassium less than 3.5 mEq/L(3).

Hypokalemic paralysis is a dramatic clinical entity. The sudden onset of flaccid

paralysis could be life threatening, but if diagnosed in time and properly treated, the response and recovery can be equally dramatic and gratifying. We present 5 cases of rapidly progressive paralysis, caused by severe derangement in the potassium homeostasis. The role of potassium in muscle weakness and paralysis and management of hypokalemia is discussed.

## Case Reports

The details of the 5 cases admitted with the diagnosis of hypokalemic paralysis is given in *Table I*. They were 2 males and 3 females, between the ages of 7 months to 5 years.

## Discussion

Potassium, with other electrolytes, is lost in diarrheal stool at all ages, but more so in children(4). Disturbance of potassium equilibrium may produce a wide range of clinical disorders, including myopathy; marked muscle wasting, diminution of muscle tone, power and reflexes(5-9). Severe potassium depletion can result in two major neuromuscular consequences: paralysis and rhabdomyolysis. Paralysis has a predilection for extremities, with legs more involved than arms. Trunk musculature can also be involved and may result in life threatening respiratory paralysis. Severe paralytic complications usually occur

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*Received for publication March 26, 1990;  
Accepted February 22, 1991*

TABLE 1—Profile of Cases

Feature	Case 1	Case 2	Case 3	Case 4	Case 5
Age/Sex	1yr 6 mo/M	7mo/F	8mo/M	5yr/F	4yr/F
Symptoms & signs	Gastroenteritis Dehydration Quadriparesis (2/5) with respiratory paralysis	Ac. GE, PEM, Lowerlimb paresis (3/5) Intercostal muscle weakness, dehydration	Ac. GE Quadriparesis Dehydration	Rickets, Lower limb paresis (2/5) Renal tubular acidosis	Ac. GE Quadriparesis (2/5) No dehydration
S. electro- lytes (mEq/L) K <sup>+</sup> —at admn. — rpt.	Na <sup>+</sup> 122 1.1 2.3	Na <sup>+</sup> 124 2.6 5.8	Na <sup>+</sup> 114 1.3 3.4	Na <sup>+</sup> 150 2.6 3.5	Na <sup>+</sup> 142 2.0 4.7
Treatment	Respiratory support, dehy- dration/Na <sup>+</sup> /K <sup>+</sup> correction with IV fluids	IV fluids K <sup>+</sup> Correction with IV/oral supplements	Dehydration/ Electrolyte correction with IV fluids	K <sup>+</sup> correction with IV/oral supplements	Oral K <sup>+</sup> supplements
Time taken for neuro- muscular recovery (h)	48	48	48	48	48
Follow up	Doing well	Died of unrelated problems (PEM/sepsis)	Doing well	Doing well	Lost for follow up

Figures in parentheses indicate muscle power grading on admission, Ac. GE = Acute gastroenteritis; PEM = Protein energy malnutrition.

with serum potassium values less than 2.6 mEq/L(10).

In most reported cases, the cause of hypokalemia has ranged from familial periodic paralysis and sporadic hypokalemic paralysis(8,9) to diuretic or laxative abuse, renal tubular defects, and chronic alcoholism. Though gastroenteritis is a well known cause of hypokalemia, not much information is available about muscle paralysis, including respiratory paralysis, due to hypokalemia following gastroenteritis. Zaman *et al.*(11) have reported that of 307 children, nearly 66.6% had evidence of mild to severe hypokalemia (severe in 3%), with almost 86% showing evidence of muscular involvement in the form of muscle weakness.

Four of our 5 cases had acute gastroenteritis and dehydration as a cause of hypokalemia (with Grade III PEM also contributing in one).

Potassium deficits must be replaced quickly if severe muscle weakness or paralysis is present, with careful watch on serum potassium and ECG monitoring to document the effects of changing potassium levels. Whenever possible potassium deficits should be replaced by oral rather than intravenous route, 3 mEq/kg/day plus maintenance needs being appropriate to initiate potassium repletion. If intravenous potassium is used, care must be taken to avoid abrupt rise in serum potassium. It can be given in a concentration of 20 to 30 mEq/L in dextrose or saline infusion, concentrations above 40 mEq/L should be used only with ECG monitoring(3,10). The normal rate of infusion should be 0.25 mEq/kg/h except in cases of quadriplegia with threatened respiratory insufficiency, when the initial rate can be as high as 1 mEq/kg/h(12).

In conclusion we feel that in all patients with gastroenteritis and dehydration, serum potassium level should be estimated and neuromuscular effects of hypokalemia be looked for. In all patients with rapidly progressive severe muscle paralysis, hypokalemic paralysis must be strongly considered and urgently tackled, if present.

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## NOTES AND NEWS

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Guest Editor : Dr. Vimlesh Seth

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