Total Body Potassium and Serum Electrolyte Concentrations in Protein Energy Malnutrition

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SUMMARY

The total body potassium (TBK) and serum electrolyte concentrations of malnourished children in Cape Town are similar to those in Jamaica. The TBK is almost always low and hyponatraemia is common, but there was no relationship between the TBK and serum Na and K concentrations. These abnormalities, particularly a very low TBK, appear to influence the prognosis.

The expression of the observed TBK as a percentage of the expected value for a normal child, of the same weight and height, appears to be a useful index of the severity of the total body potassium deficit.

Abnormal total body and serum electrolyte concentrations are common in protein energy malnutrition (PEM), and appear to be related to the prognosis. In Jamaica, 7 of 9 children with a serum sodium concentration less than 121 mEq/litre died, while only 12 deaths occurred in a series of 98 children who had a serum sodium of more than 121 mEq/litre. The presence of hyponatraemia does not mean that a total body sodium deficit exists. There is an excess of sodium in an even greater excess of water. In contrast to the relationship between total body sodium and the serum concentration, a normal serum potassium level is frequently found in the presence of a very low total body potassium (TBK). This article reports the TBK and serum electrolyte concentrations of a series of children suffering from kwashiorkor.

RESULTS

The mean and standard deviation of the TBK, serum electrolyte and albumin concentrations, and weight as a percentage of the expected weight for age and height, are given in Table I.

| TABLE I. MEAN AND STANDARD DEVIATION OF THE TBK, SERUM SODIUM, POTASSIUM, CHLORIDE AND ALBUMIN CONCENTRATIONS AND OF THE WEIGHT AS A PERCENTAGE OF THE EXPECTED WEIGHT FOR AGE AND FOR HEIGHT (56 CHILDREN) |
|-----------------------------------------------|------------------|------------------|
| TBK (mEq/kg) | Mean | SD |
| Precentage predicted TBK | 91.65 | 12.57 |
| Serum sodium (mEq/litre) | 135.8 | 8.6 |
| Serum potassium (mEq/litre) | 3.98 | 0.87 |
| Serum chloride (mEq/litre) | 101.0 | 6.6 |
| Serum albumin (g/100 ml) | 1.77 | 0.54 |
| Weight as % exp. weight for age | 61.6 | 10.6 |
| Weight as % exp. weight for height | 84.3 | 12.2 |

Frequency of Abnormal TBK and Serum Electrolyte Results

The normal range of percentage predicted TBK is 100 ± 3.3%. Only 7 of the 56 children were not below the lower limit of normal. All were less than 99%.

Four children had a serum potassium level less than 3 mEq/litre and only 1 child had more than 6 mEq/litre.
The serum sodium concentration was less than 130 mEq/litre in 14 cases and greater than 150 mEq/litre in 2. The serum chloride concentration was less than 90 mEq/litre in 5 cases and greater than 110 mEq/litre in 4.

Relationship between the TBK in mEq/kg and Percentage Predicted TBK

The 7 children who had a normal percentage predicted TBK, i.e. greater than 96.7%, all had a TBK of greater than 37.5 mEq/kg. When the TBK was between 32.5 and 37.5 mEq/kg, the percentage predicted TBK was 92.5 - 96.7%. The percentage predicted TBK was less than 92.5% in all but 2 of the patients who had a TBK of less than 30 mEq/kg (Fig. 1).

Mortality and TBK, and Serum Electrolyte and Albumin Concentrations

Six of the 56 children died. All deaths occurred within the first 5 days after admission to hospital. One child had bronchopneumonia with extensive consolidation of all lobes, and the other 5, who died suddenly, had no specific abnormalities. Four of the 6 children had a TBK of less than 30 mEq/kg on admission, and 1 of these had a serum sodium of 121 mEq/litre. The 2 children who had TBKs above 30 mEq/kg had serum sodium concentrations of 117 and 158 mEq/litre. The serum albumin concentrations ranged from 0.78 to 2.55 g/100 ml. 4 of the children having levels between 1.51 and 1.91 g/100 ml.

DISCUSSION

The serum electrolyte concentrations and the TBK in mEq/kg are similar to those found in Jamaica.\(^1\,3\) This suggests that the results of Jamaican studies relating the TBK in mEq/kg to change in muscle potassium concentrations can be used quantitatively as well as qualitatively in the interpretation of the results of this study. This is supported by the results of a few muscle biopsy analyses\(^6\), which were similar to those reported by Nichols et al.\(^7\). As the TBK drops, the potassium concentration in muscle declines progressively when referred to all tissue denominators. When the TBK is less than 35 mEq/kg, the concentration of potassium in fat-free whole muscle, total muscle water, dry solids and total nitrogen, was significantly reduced from the concentration found in recovered controls. In concentrations based on water content, fat-free whole muscle and muscle water, the decrease is significant when the TBK is below 40 mEq/kg.

Alleyne et al.\(^8\) used the concept of potassium capacity. This is defined as the sum total of anions and other chemical groups outside of the extracellular space capable of holding or binding potassium ions.\(^9\) A modest reduction in the potassium content of muscle when the TBK is between 30 and 40 mEq/kg, was found. They concluded that if the changes in muscle are an indication of the state of the other potassium-containing organs, the fall in TBK in this range is a reflection of a reduction mainly of potassium capacity. When the TBK falls below 30 mEq/kg there is a marked fall of potassium concentration in muscle. There is now not only a fall in potassium capacity, but this capacity is unsaturated and there is true potassium depletion.

The relationship between the TBK in mEq/kg and the percentage predicted TBK can be regarded as evidence supporting the validity and value of the prediction method, when it is examined in the light of the results of Nichols et al.\(^7\) and of Alleyne et al.\(^8\). In all the children who had a 'normal' percentage predicted TBK, the TBK in mEq/kg was greater than 37.5. There was probably little or no
decrease in muscle potassium concentration in these children. The majority (88%) of children studied had a percentage predicted TBK below the normal range. Their TBK was less than 37.5 mEq/kg, and relating this to the results of the Jamaican studies suggests that a reduction of muscle potassium content had occurred. The percentage predicted TBK was 5-10% less than normal, and it is possible that at this level the pathological effects of potassium depletion start to appear. It is difficult to be precise on this point, mainly because of the problem of detecting potassium depletion clinically. The lowest percentage predicted TBK occurred in those with a TBK of less than 30 mEq/litre. This is the level below which Alleyne et al. considered that true potassium depletion, rather than a reduction in potassium capacity, occurred. It is probably significant that 4 of the 6 patients who died were below this level. The lack of relationship between the TBK and the serum potassium demonstrates the unreliability of serum estimation in detecting potassium depletion.

In the malnourished child there are numerous factors which may influence the serum sodium concentration, and the absence of any relationship between TBK and the serum sodium level is not unexpected. It does not exclude the possibility that the potassium deficit is at least partly responsible for the increase in total body sodium. The correlation between TBK and the serum chloride concentration was significant at the 5% level. It may be a chance finding, but there is a relationship between potassium and chloride depletion. If a chloride deficit is present, it is probably relative rather than absolute, since total body sodium, total body water and extracellular fluid volumes are increased.

The importance of electrolyte disturbance in malnourished children is shown by the abnormal TBK and/or serum sodium concentrations of the children who died. The degree of malnutrition as judged by their serum albumin concentrations was similar to that of those who survived. Kritzinger et al. found that plasma renin activity was increased threefold in children who subsequently died. It is possible that this and the disturbance of electrolyte metabolism are related.

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REFERENCES