SHORT COMMUNICATION

Potassium changes in megaloblastic anaemia

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Summary

1. Serial measurements of serum potassium and total-body potassium were made on eighteen patients with megaloblastic anaemia before the start of therapy and during the period of recovery.

2. In those patients who presented with an initial packed cell volume of less than 25%, a mean decrease in serum potassium of 0.4 mmol/l occurred on average within 2.5 days of the start of therapy. This was followed by a significant increase in serum potassium during the period of recovery. There was a significant increase in total-body potassium in these patients, but this could be explained by increases in erythrocyte mass, erythrocyte potassium concentration and lean body mass.

Key words: megaloblastic anaemia, potassium.

Introduction

James & Abbott (1952) reported a fall in serum potassium after treatment in megaloblastic anaemia. This was confirmed by Lawson, Murray & Parker (1972), who suggested that the unexpectedly high mortality in severe megaloblastic anaemia, after commencement of treatment, might be related to a low concentration of serum potassium and to a depletion of total-body potassium. Since more than 97% of body potassium is intracellular, this can be regarded as synonymous with intracellular potassium (Edmonds & Jasani, 1972).

A sudden cellular demand for potassium, after commencement of specific therapy, was postulated as the cause of the observed initial fall in serum potassium (Lawson et al., 1972; Editorial, 1972). Results were presented to show that serum potassium and erythrocyte potassium were low before therapy, and that they returned to normal after therapy.

In view of these reports, measurements of total-body potassium were included in the routine tests carried out on a series of patients undergoing treatment for megaloblastic anaemia to see whether potassium depletion, which might require treatment, occurred.

Methods

Eighteen patients with megaloblastic anaemia were studied. There were nine males and nine females, aged 17-77 (average 58) years. By the classification employed by Lawson et al. (1972) the degree of severity varied on presentation from severe (PCV(1) < 25%, seven patients) to mild (PCV > 35%, eight patients).

The clinical diagnoses were pernicious anaemia (eight cases), folate deficiency (four cases) and dietary vitamin B12 deficiency (six cases).

Regular blood samples were obtained from each patient over the period of investigation. Each sample was analysed by means of a Coulter counter model S (Chanarin, England & Hoffbrand, 1973) to determine PCV, haemoglobin, the number of erythrocytes per unit volume, and mean cell volume. Serum potassium was determined by flame photometry.

Total-body potassium was measured by means of a

(1) Abbreviations: PCV, packed cell volume.
multi-detector whole-body counter situated inside a low-background steel cubicle. Four detectors were situated above, and four below, the supine patient. Each of the eight gamma-ray detectors incorporated a NaI(Tl) crystal, 10 cm diameter x 7.5 cm thick.

On each occasion the patient was weighed and total-body potassium measured for 2000 s; the background was measured for 4000 s, with a phantom containing distilled water in the counting position.

The 1.46 MeV gamma-rays associated with $^{40}$K were recorded in the energy range 1.35-1.55 MeV. The standard errors on a measurement of total-body potassium attributable to radioactivity counting and calibration ranged from $\pm 120$ mmol on 3600 mmol in a 75 kg man, to $\pm 85$ mmol on 1800 mmol in a 45 kg woman.

An average of six measurements were performed on each patient starting before treatment and continuing throughout the treatment period (33-128 days, mean 62 days). The significance of changes was assessed as described by Bailey (1969).

Results

There were no significant changes in serum potassium or in total-body potassium in the mild or intermediate cases.

The mean values for serum potassium in the severe cases before and during treatment are given in Table 1. On average the minimum value associated with the early decrease in serum potassium (early post-therapy value) occurred at 2.5 days after commencement of treatment. The mean decrease in serum potassium was 0.4 mmol/l (SEM 0.2, $n = 7$, $P > 0.05$) and the largest decrease was 0.7 mmol/l. Subsequently, the mean serum potassium gradually increased significantly by 0.8 mmol/l (SEM 0.2, $n = 7$, $P = 0.02$) (Bailey, 1969).

The mean value for the change in total-body potassium (Table 1) was 177 mmol (SEM 52, $n = 7$, $P < 0.05$), and this represented an increase of 8.7% (SEM 2.0, $n = 7$, $P < 0.02$) of the late post-therapy value.

By using the data for all eighteen patients, a high degree of correlation ($r > 0.90$) was found between changes in total-body potassium and changes in PCV, haemoglobin and erythrocyte count after allowance was made for the experimental errors in total-body potassium (Healy, 1958).

Discussion

Although the concentrations of serum potassium varied in similar ways in both sets of data (Table 1), the fall in early post-therapy values was less significant in our severe cases than in the cases reported by Lawson et al. (1972). Potassium supplements were not given to any of our patients. The mean presenting value for PCV in their group of patients was 18.0% (SEM 1.0, $n = 34$), which was not significantly different from our value of 17.8% (SEM 1.7, $n = 7$) (Table 1).

Because a wide range of values for total-body potassium can be found in groups of similar individuals, we have carried out serial measurements on every patient so that each could serve as his or her own control. The late post-therapy values were

<table>
<thead>
<tr>
<th>Current work ($n = 7$: six females, one male)</th>
<th>Lawson et al. (1972)</th>
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</thead>
<tbody>
<tr>
<td><strong>Total-body K</strong> (mmol)</td>
<td><strong>PCV (%)</strong></td>
</tr>
<tr>
<td>Pre-therapy</td>
<td>1852</td>
</tr>
<tr>
<td></td>
<td>$\pm 62$</td>
</tr>
<tr>
<td>Late post-therapy</td>
<td>2029</td>
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<tr>
<td></td>
<td>$\pm 102$</td>
</tr>
<tr>
<td>Change</td>
<td>177</td>
</tr>
<tr>
<td></td>
<td>$\pm 52$</td>
</tr>
</tbody>
</table>

$^{(1)}$ Early post-therapy values.
assumed to represent the best estimates of normal total-body potassium. On average, these late post-therapy values were not significantly different from estimated normal values, but the pre-therapy values represented on average 0.94 (SEM 0.02, n = 7) of the estimated normal values (Boddy, King, Hume & Weyers, 1972).

The average increase in total-body potassium in the severely anaemic patients was 177 mmol (Table 1), of which an estimated 93 mmol could be accounted for by the increase in the number of erythrocytes. This estimation was based on the data given in Table 1, together with published data, on blood volumes (Chanarin, 1969; Altman & Dittmer, 1961) and potassium in erythrocytes (Lawson et al., 1972). An average increase in lean body mass of 1.3 kg (average weight increase 2.8 kg; Table 1) could account for the remaining 84 mmol (Burkinshaw & Cotes, 1973; Boddy, King, Womersley & Durnin, 1973).

Thus in patients with severe megaloblastic anaemia the total-body potassium before treatment tends to be low and increases during treatment. The observed increase in total-body potassium can be accounted for by increase in lean body mass, erythrocyte mass and erythrocyte potassium concentration.

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References


