Letter to the Editor

Erythrocyte sodium in chronic liver disease with ascites

(Received 14 June 1990; revision received 3 October 1990; accepted 8 October 1990)

Dear Editor,

Sodium retention is usual in advanced liver cirrhosis, and is manifest as ascites and edema. Hyponatremia often develops in the later stages, despite an increase in total body sodium [1]. It is generally presumed that this hyponatremia is dilutional, water being retained in excess of sodium. However, an alternative possibility is that a defect in cellular membranes permits cells to take up more sodium, lowering extracellular sodium concentrations [1]. Intracellular concentrations of sodium and potassium in erythrocytes are widely used as markers of events occurring in body tissues [2]. We therefore compared erythrocyte sodium and potassium in 23 healthy volunteers with that in nine patients with HBsAg negative cirrhosis of the liver all of whom had ascites.

Of the patients with cirrhosis, two had Wilson's disease, two had alcoholic cirrhosis, and the rest had cryptogenic cirrhosis. Spontaneous bacterial peritonitis was excluded by examination of the ascitic fluid. All these patients were on treatment with spironolactone with or without added furosemide.

Fasting blood, drawn into heparinized tubes, was processed within 30 min. Following separation of plasma and leukocytes, erythrocytes were washed thrice with ice-cold isotonic MgCl₂-Tris buffer (270 mOsm/kg, pH 7.4) and resuspended. The hematocrit was then obtained, the cells pelleted again and hemolyzed [3]. Intracellular sodium and potassium were determined by flame photometry and expressed as mmol/l packed cells.

Erythrocyte sodium was significantly decreased in patients with cirrhosis, as shown in Table I. Erythrocyte potassium concentrations tended to be higher in the cirrhotics, but this difference was not statistically significant. The serum sodium was significantly lower in patients with cirrhosis, but serum potassium was not significantly different.

Defects of the cell membrane or sodium pump have been described in patients with uremia [4,5]. If a similar situation were to be postulated in cirrhosis, we would expect erythrocyte sodium content to be elevated, and not decreased as observed here. Kessler et al. [6] reported low erythrocyte sodium concentrations in cirrhotics with ascites. They noted that diuretic treatment was associated with a return to normal of the erythrocyte sodium, and speculated that the low sodium was a result
TABLE I

Erythrocyte and serum sodium and potassium in healthy adults and cirrhosis of the liver with ascites (mean ± SEM)

<table>
<thead>
<tr>
<th></th>
<th>Erythrocyte</th>
<th>Serum</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Na⁺</td>
<td>K⁺</td>
</tr>
<tr>
<td>Controls</td>
<td>9.03 ± 0.43</td>
<td>92.6 ± 3.3</td>
</tr>
<tr>
<td>Cirrhosis</td>
<td>6.25 ± 0.74</td>
<td>100.5 ± 5.5</td>
</tr>
<tr>
<td>Significance *</td>
<td>P &lt; 0.01</td>
<td>n.s.</td>
</tr>
</tbody>
</table>

* P obtained by Wilcoxon two-tailed signed rank test. n.s. = not significant.

of hyperaldosteronism. In the present study, all the cirrhotics were receiving diuretics – in particular the aldosterone antagonist spironolactone – at the time of study. This suggests that aldosterone is not directly responsible for the low erythrocyte sodium, and supports the concept that the associated hyponatremia is due to retention of water in excess of sodium. Furthermore, the tendency to elevation of intracellular potassium suggests that the cellular sodium pump may be overactive, pumping cellular sodium out to maintain extracellular concentrations.

B.S. Ramakrishna, R. Kumaresan and K.A. Balasubramanian

*The Wellcome Research Unit and the Department of \Gastroenterology, Christian Medical College Hospital, Vellore 632 004, India*

References


Correspondence to: Dr. K.A. Balasubramanian, Professor of Biochemistry, Christian Medical College Hospital, Vellore 632 004, India.