
Refeeding after acute gastroenteritis

Sir,

Dugdale et al.1 compared the results obtained in two groups of infants suffering from acute diarrhoea who were submitted to either a rapid or a graduated refeeding regimen, and concluded that weight loss during the first 24 hours of refeeding was less and the length of hospital stay after milk introduction was shorter in the first group. However, the P values of the differences (0.1>P>0.05) are widely considered to be either not significant or on the threshold of significance.

The data concerning the number of days in hospital after refeeding are confusing as 7 (25%) of 28 infants in the first group and 3 (9%) of 32 in the second group had to go back to clear fluids because of recurrent vomiting or diarrhoea, which means that in an appreciable number of infants (17%) milk introduction was tried more than once.

Dugdale et al. gave no indication of the nutritional status of their patients; however in view of the fact that the mean age was about 22 months in both groups, and that 61% of the infants had mild or non-evident signs of dehydration (weight loss <5%), we can infer that diarrhoea was generally not severe. They stated that in developing countries children with acute diarrhoea are left on milk, particularly if breast fed; this is so because the biological and nutritional properties of human breast milk make it uniquely suitable both in health and disease (except in the case of some inborn metabolic diseases). Its high lactose content, which might have been an inconvenience during acute diarrhoea or soon after it, is not harmful in practice. The same is true for infants with cows' milk protein-sensitive enteropathy for whom breast milk is usually the best feeding alternative despite the frequent coexistence of lactose intolerance. However, in the non-breast fed infant with acute gastroenteritis and malnutrition it is thought best to interrupt, or decrease, the use of a lactose formula.

Severe diarrhoea and delayed recovery are more likely to occur in the first months of life particularly in malnourished infants.4 The problem is not when and how to refeed an older and well nourished child or infant with mild gastroenteritis, but how to treat the young or malnourished infant who has severe diarrhoea. In this case there are no strict rules and the physician may have to vary the procedure concerning the reintroduction of a formula in order to avoid delaying recovery.

References


Dr Dugdale and co-workers comment:

We agree with most of the points made by Dr Salazar de Sousa.

We regret that we did not give data on the nutritional status of the children. The usual methods based on weight-for-age or weight-for-height can be misleading in the presence of dehydration. However, none of these children showed clinical evidence of malnutrition.

Some gastroenterologists, and texts on the subject, suggest that early introduction of milk in children with gastroenteritis is contraindicated as being potentially harmful to the child.1, 2 Our study was designed to show that rapid reintroduction was possibly beneficial, not harmful. The statistical method used tested the hypothesis that rapid refeeding gave weight gains that were different from those with standard treatment, and showed that there may be a significant difference favouring rapid refeeding. If we were to test the hypothesis that rapid refeeding was demonstrably worse than standard treatment, then the level of statistical significance favouring rapid refeeding would be high. Therefore using these criteria on this group of children we have demonstrated that rapid refeeding is not worse than standard treatment, and may, perhaps, be better. We conclude that this form of management of acute gastroenteritis is an option in western-style communities. It is significant that as long ago as 1962 Darrow2 made the point that giving milk early may demonstrate an existing malfunction rather than cause it.

Although we did not consider gastroenteritis in malnourished children, we have experience of this problem. In developing countries it is common practice to continue breast feeding children with gastroenteritis but to stop breast-milk substitutes. If breast milk is tolerated under these conditions and improves the outlook for the child, it is possible that breast milk substitutes would also help. We know of no study which has tested this but we believe that such data are sorely

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needed; paediatricians would then have guidelines for treatment rather than the present ad hoc methods described by Dr Salazar de Sousa.

References


Premature thelarche

Sir,
Dumić et al.¹ suggest that premature thelarche may be an adrenal disorder; I do not think they have provided sufficient evidence for this.

Although patients and normal controls were comparable in number and age range, no information was given on the mean or median age of each group. Since the peak age of onset and presentation of premature thelarche was between 1 and 3 years I assume that most of their patients were clustered around the lower part of the relevant age range. Comparison of plasma dehydroepiandrosterone (DHEA) concentrations in the age range 1–3 years for each group would be more meaningful, since the profound effect of adrenarche on plasma DHEA concentrations would have been eliminated. Both groups showed a large SD in plasma DHEA concentrations, presumably the result of adrenarche having started in some of the subjects. The coefficient of variation of the DHEA assay was also rather high; therefore the values reported in this study were only similar to those reported in the literature, when compared with a 4–8-year-old band, but not the 1–4-year-age band.²

The authors rightly state that plasma oestradiol and gonadotrophin concentrations are generally normal in children with premature thelarche. That DHEA and its sulphate may serve as a substrate for oestrogen synthesis in this disorder is an attractive hypothesis. However, it is not possible to conclude that plasma DHEA concentrations are increased unless patients and controls are studied during the peak age range of this disorder—that is 1–3 years.

References


Dr Dumić and co-workers comment:

Dr Hughes has made a useful suggestion and we present in the Table our data on the mean ages and the mean DHEA concentrations of our children with premature thelarche and in normal controls divided into two age groups in the ranges of 1–4 years and >4–7 years.

In the younger group there is a very significant difference in the respective DHEA concentrations with no difference in the mean ages.

This makes our data on the increased DHEA in girls with premature thelarche even more meaningful, as suggested by Dr Hughes.

The lack of a significant difference in the DHEA concentration in children of the older age group can be explained as being due to adrenarche that has already started in some of the children of this age.

Table  Dehydroepiandrosterone concentrations (ng/100 ml) in normal girls and girls with premature thelarche

<table>
<thead>
<tr>
<th>Age group (years)</th>
<th>Mean age (years)</th>
<th>n</th>
<th>Mean</th>
<th>SD</th>
<th>SE</th>
<th>Normal children</th>
<th>Mean age (years)</th>
<th>n</th>
<th>Mean</th>
<th>SD</th>
<th>SE</th>
<th>Premature thelarche</th>
<th>t test</th>
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</tr>
</thead>
<tbody>
<tr>
<td>1–4</td>
<td>1.75</td>
<td>15</td>
<td>123.4</td>
<td>73.8</td>
<td>19.10</td>
<td>1.95</td>
<td>12</td>
<td>207.9</td>
<td>81.83</td>
<td>23.62</td>
<td>-2.676</td>
<td>-0.02</td>
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<tr>
<td>&gt;4–7</td>
<td>5.45</td>
<td>9</td>
<td>177.6</td>
<td>127.6</td>
<td>42.54</td>
<td>5.25</td>
<td>7</td>
<td>195.8</td>
<td>68.3</td>
<td>25.8</td>
<td>-0.345</td>
<td>-0.80</td>
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</tr>
</tbody>
</table>

Conversion: traditional units to SI—dehydroepiandrosterone: 1 ng/100 ml = 0.0347 nmol/l.