Hyperglycaemia in infantile gastroenteritis

Sir,

We read with interest the paper by Rabinowitz et al. Our own impressions were at variance with his figures. To verify our views we analysed 33 cases of acute gastroenteritis treated within our hospital in the last six months. All patients admitted with acute gastroenteritis have urea and electrolytes determinations before treatment is started. In our laboratory, blood glucose estimation is performed routinely on all blood samples sent for urea and electrolytes measurement. Blood glucose concentration is measured by glucose oxidase method.

The age range of our patients was between 1 and 22 months (mean 7.3 months). Twenty of these were below the 3rd centile of the Boston weight chart. One stool sample grew shigella but none of the others grew any pathogenic bacteria. The degree of dehydration was calculated from the weight of the child at admission, and weight when diarrhoea had stopped and the child was fully rehydrated. There was good correlation between this method and the clinical assessment of dehydration at admission.

Twelve children had severe dehydration with more than 10% loss of body weight and the rest had mild to moderate dehydration. Eleven children had high urea (more than 6-6 mmol/l), 14 had low potassium (less than 3-8 mmol/l), 10 had low sodium (less than 133 mmol/l), and 24 had low bicarbonate (less than 22 mmol/l) concentrations, but none had a blood glucose value of more than 10 mmol/l.

An occasional patient with high blood glucose concentration has been seen by all of us but we were sure that in our experience the incidence could not have been as high as 55%. All the clinical and biochemical parameters of our patients were quite similar to those reported by Rabinowitz et al. The differences between our and their experience and the similarities between their normal and hyperglycaemic children with acute gastroenteritis show that there must be some other factor(s) responsible for this phenomenon. More work needs to be done to find out whether it is the type of virus, the race of the patient, or over zealous use of oral rehydration powder that is responsible for this disparity.

Reference


B Singh, K Yip, S Bafakeer
King Khalid National Guard Hospital, Jeddah, Saudi Arabia

Dr Rabinowitz and co-workers comment:

We suggest that the following points may explain Singh et al’s failure to observe hyperglycaemia, compared with our high prevalence, in infantile gastroenteritis:

1. The nature of any underlying viral infection and ethnic group of the children, as acknowledged. Indeed, it is well established that certain viral infections may produce pancreatic beta cell destruction. None of our infants was given oral rehydration powder before admission.

2. A difference in seasonal incidence may also be relevant. Our patients presented in the late summer months but it is not clear when the Saudi Arabian children presented.

3. All the children in our study had severe disease, necessitating hospital admission. This evoked marked stimulation of stress hormones, one of the main pathogenetic factors in the evolution of hyperglycaemia.

4. Finally, the mode of collection of samples for blood glucose estimation appears to have differed. If their samples were sent to the laboratory in ‘U and E’ tubes without fluoride preservative, it is conceivable that substantial in vitro glycolysis occurred (especially in a hot climate), thereby spuriously lowering blood glucose readings.

Functional palatal incompetence and teratogenesis

Sir,

The interesting report by Dr Pearl and colleagues of palatal incompetence in the fetal anticonvulsant syndrome prompts us to describe two children who have similar problems.

The children are sisters born to first cousin Caucasian parents. Throughout each pregnancy their mother smoked 20 cigarettes and drank 5 or 6 ‘shorts’ of alcohol daily. At various times during the pregnancy of the elder girl she also took diazepam, nitrazepam, amitryptiline, and phenelzine.

Abnormal features present in both sisters include short stature and microcephaly, severe developmental delay, ataxia with an intention tremor, delayed bone age, and a similar facies with arched eyebrows, peri-orbital fullness, anteverted nares and thin upper lip. The elder girl has pulmonary hypertension with an atrial septal defect and patent ductus arteriosus. The younger girl has a normal heart and unilateral ptosis. Both girls have hypernasal dysarthria with nasal escape and very reduced palatal movement. Structurally their palates are intact with no submucous cleft.

The parental consanguinity makes it difficult to exclude an autosomal recessive disorder in these sisters, although their abnormalities do not readily conform to any inherited disorder known to us. They do, however, closely resemble the fetal alcohol syndrome in which speech problems including disorders of articulation and ‘voice dysfunction’ are common, although we are unaware of any report clearly documenting palatal incompetence in this condition.

In the patients described by Dr Pearl and colleagues, teratogenesis was attributed to anticonvulsants. One of the mothers also smoked heavily and abused alcohol. Alcohol
Correspondence

and anticonvulsants act synergistically and similarities between the fetal anticonvulsant and alcohol syndromes have been noted. Consequently, we wonder whether palatal function may be particularly susceptible to the teratogenic effects of alcohol or anticonvulsants, or both, and suggest that if such an effect were confirmed by others this would be a useful diagnostic pointer in these disorders.

I D Young and J R Moore
Leicester Royal Infirmary, Leicester

References


Clinical significance of gastro-oesophageal reflux

Sir,

Professor Carré is right to emphasise the frequency of unimportant gastro-oesophageal reflux in babies and children. It is possible to induce radiologically identifiable gastro-oesophageal reflux in almost any normal child. He is correct, therefore, in advising caution in attributing a whole gamut of symptoms to what may be a coincidental physiological event.

Unequivocal intrathoracic stomach is a rarity, and when it exists with reflux both are important and in need of management. Professor Carré states that the recognition of an intrathoracic stomach is all important and implies that in its absence reflux is of no significance. We would disagree with him on this. We have identified an intrathoracic stomach definitely in only four patients, and equivocally in a further one patient out of 18 who have needed recent fundoplication. In five patients there were severe strictures but in only one of these was there a partially intrathoracic stomach.

Eleven of the 18 patients had a funnel shaped gastro-oesophageal junction with no intra-abdominal oesophagus. This radiological sign may be important. From this sign some might infer that there is intrathoracic stomach mucosa and elevation of the lower oesophageal sphincter— as illustrated in Professor Carré’s Figs. 3 and 4. Failure of the oesophagus to empty itself of refluxed material may also be important. Funnel shaped gastro-oesophageal junction and persistence of refluxed material can be identified also in children whose vomiting resolves without developing severe complications of gastro-oesophageal reflux requiring surgery. They are not absolute signs of poor prognosis.

We suggest that there are factors other than a detected intrathoracic stomach which result in a poor prognosis for gastro-oesophageal reflux, and to try to identify these factors using new techniques is justifiable, and should be encouraged.

Reference


J F Ratcliffe, V Miller, and C Doig
Booth Hall Children’s Hospital, Blackley, Manchester M9 2AA

Ultrasound and the diagnosis of Wilms’ tumour

Sir,

We read with interest the paper on the usefulness of ultrasound in the management of abdominal malignancy by Kohler et al. They have assessed the performance of the ultrasound scan by calculation of its diagnostic accuracy and state that ultrasound is no more efficient than intravenous urography in the diagnosis of Wilms’ tumour and neuroblastoma. The efficiency of a test, however, is calculated in the following manner:

$$\text{TP} = \text{true positive}; \text{TN} = \text{true negative}; \text{FP} = \text{false positive}; \text{FN} = \text{false negative}.$$

<table>
<thead>
<tr>
<th>Patients with a positive ultrasound scan for Wilms’ tumour</th>
<th>Patients with a negative ultrasound scan for Wilms’ tumour</th>
<th>Total</th>
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<tbody>
<tr>
<td>Patients with Wilms’ tumour</td>
<td></td>
<td></td>
</tr>
<tr>
<td>36 (TP)</td>
<td>5 (FN)</td>
<td>41</td>
</tr>
<tr>
<td>Patients without Wilms’ tumour</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6 (FP)</td>
<td>56 (TN)</td>
<td>62</td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td>103</td>
</tr>
</tbody>
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The efficiency of any diagnostic test is the percentage of all results which are true results, whether positive or negative, and may be obtained by adding the number of true positive results to the number of true negatives and dividing by the total number of subjects tested. The diagnostic efficiency of ultrasound for Wilms’ tumour in the study population is, therefore, 36 + 56/103 = 89.3%.

The authors are not justified in stating that ultrasound is no more efficient than intravenous urography in the diagnosis of Wilms’ tumour and neuroblastoma without such efficiency calculations.

Similarly, the term diagnostic sensitivity has been used in a loose sense. The sensitivity of any diagnostic test is the percentage of true positive results obtained when a test is applied to patients known to have the disease and is obtained by dividing the number of true positive results by the total number of true positive and false negative results. For example, the diagnostic sensitivity of ultrasound for Wilms’ tumour in the study population is 36/41 = 87.8%.

In their paper the authors have equated diagnostic sensitivity with diagnostic accuracy which is not the case. The terms diagnostic accuracy, diagnostic sensitivity, and
Functional palatal incompetence and teratogenesis.

I D Young and J R Moore

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