cough". Three weeks later, she progressively developed a symmetrical eruption composed of 1–3 mm erythematous papules on the knees, ankles, elbows and forearms. She had mild fever and was quite miserable. Cervical and axillary lymphadenopathy was present. Liver and spleen were enlarged, being 2 cm and 1 cm respectively below the costal margins. A clinical diagnosis of Gianotti–Crosti syndrome was made. Laboratory studies showed mild microcytaemic anaemia (Hb 11.8 g/dl, MCV 75 μm³), lymphocytosis (leucocytes 15 300/mm³ with 15% neutrophils, 2.5% eosinophils, 1% basophils, 7.5% monocytes, 72.5 lymphocytes and 1.5% plasmocytes), and elevated liver enzymes (AST 74 U/l, ALT 67 U/l). Seroconversion to EBV was found with anti-viral capsid antigen IgM antibodies positive and anti-viral capsid antigen IgG titers raising from 1/40 to 1/320 in 2 months. Antibodies to human immunodeficiency virus 1–2, cytomegalovirus, hepatitis A–B–C, parvovirus and toxoplasmosis remained negative. The child was unwell for 3 weeks and the eruption then gradually disappeared over a 3-week period. No treatment was given.

Children may develop Gianotti–Crosti syndrome associated with EBV as the sole stimulatory factor, and it might well have been the case with this child. However, since Gianotti–Crosti syndrome can be considered the result of an individual immune response to a viral infection and not the direct consequence of the virus itself, and since it has also been described after immunization, it is reasonable to think that in this child, both the immunization and the EBV infection acted as triggers to the eruption. Such a sequence is in accordance with the recent suggestion that Gianotti-Crosti syndrome may need both a previous immune stimulation and a viral infection [1].

References

C. H. Schröder
Dorine W. Swinkels
Hans L. Willems
Leo A. H. Monnens

Studies with pre-treatment of milk with calcium acetate to reduce the phosphate content

Received: 26 July 1994
Accepted: 27 January 1995

Sirs: In the management of chronic and end stage renal failure the dietary intake of potassium and phosphate has to be reduced. Drugs that bind these compounds are prescribed on a large scale. These drugs have an unpleasant taste and have to be administered in relatively large volumes. Pre-treatment of drinks with calcium polystyrene-sulphonate leads to a 50% reduction in potassium content [1, 2]. The present study aims to reduce the phosphate content of milk, since this is the major compound of infant nutrition. The method must be simple enough to be applied at home by the patient, and must not negatively influence the taste.

Five ml of normal cows milk were treated with calcium acetate (CaAc) in different quantities (15, 30, 45, 60, 75, and 150 mg). Samples were mixed thoroughly, after filtration at 4°C pH as well as calcium, phosphate, and protein contents decreased markedly (from 6.71 to 6.05 and from 37 to 29 g/l with 45 mg of CaAc). The milk developed an unpleasant sour taste if more than 30 mg of CaAc was added. The influence of duration of incubation and of temperature is given in Table 2, using an amount of 30 mg of CaAc.

From these experiments it is clear that a simple precipitation reaction with CaAc is not a suitable method to reduce the phosphate content of milk. Other methods, such as dialysis may be applied, but these techniques are not suitable for routine domestic use.

Table 2 Influence of duration of incubation on calcium and phosphate concentration after the addition of 30 mg of CaAc, at two different temperatures. The first number gives the concentration at 4°C, the second at room temperature

<table>
<thead>
<tr>
<th>Incubation time</th>
<th>Phosphate (mmol/l)</th>
<th>Calcium (mmol/l)</th>
</tr>
</thead>
<tbody>
<tr>
<td>30 m</td>
<td>30/30</td>
<td>66/64</td>
</tr>
<tr>
<td>60 m</td>
<td>27/30</td>
<td>60/63</td>
</tr>
<tr>
<td>90 m</td>
<td>35/34</td>
<td>72/56</td>
</tr>
<tr>
<td>120 m</td>
<td>29/29</td>
<td>63/63</td>
</tr>
<tr>
<td>21 h</td>
<td>31/31</td>
<td>65/64</td>
</tr>
</tbody>
</table>

References

C. H. Schröder (✉) · L. A. H. Monnens
Department of Paediatrics,
St. Radboud University Hospital,
P.O.B. 9101, 6500 HB Nijmegen,
The Netherlands
Tel.: NL-80-616872
Fax: NL-80-540576

D. W. Swinkels · Roland Verschuur
H. L. Willems
Department of Clinical Chemistry,
University of Nijmegen, The Netherlands