Infantile colic and feeding

M-R STÅHLBERG AND E SAVILAHTI

Department of Paediatrics, Turku University Hospital, and Children's Hospital, University of Helsinki, Finland

SUMMARY In a double blind crossover study 10 children with infantile colic were fed breast milk and cow's milk formula, untreated and treated with lactase. Colic was present on 71% of breast milk and 89% of cow's milk days. Daily duration and severity of colic did not differ for milk preparations.

Infantile colic, excessive and unexplained crying by healthy infants, commonly in the evenings, has been found to associate with cow's milk given as a formula[1] or used by the lactating mother.[2] In other studies no such associations have been detected[3 4] nor did infantile colic seem to be a symptom of lactose malabsorption.[5] To explore the connection between infantile colic and feeding we conducted a double blind crossover study using four milk preparations.

Patients and methods

Subjects. Ten weaned children, with a mean (SD) age of 11·9 (8·4) weeks, were referred to us by health nurses from well baby clinics because of infantile colic, which had continued since a mean (SD) age of 4·2 (7·0) weeks. On physical examination all children seemed healthy. Their mean (SD) weight gain since birth had been 216 (82) g per week. One child had asymptomatic bacteriuria without urinary tract anomalies. Urinary cultures of the other children yielded normal results. No pathogenic bacteria, rotavirus, or adenovirus antigens were found in faecal samples.

Milks. Four milk preparations were used in random order. These were (1) pooled breast milk; (2) pooled breast milk treated with Maxilact LX 5000 (Gist-Biocade, Delft, The Netherlands), hydrolysing enzymatically over 90% of lactose contents; (3) commercial adapted liquid cow's milk formula (Tutteli, Valio Ltd, Finland); and (4) the same cow's milk formula treated with Maxilact LX 5000 as above. Milks 2–4 were prepared, packed in coded cans, and delivered frozen by Valio Ltd. Untreated breast milk was frozen in similar coded cans. The aim was to feed each child for one week with each of the four milks. Frozen milk was given for one week's estimated need at a time. No other milk products were given to the babies during the study.

Recordings. The parents recorded daily the time, duration, and severity of attacks of colic. Once a week the children visited the clinic where they were examined, recordings from the previous week were returned, and milk for the next week was delivered.

Statistical methods. We used two way analysis of variance and Student's *t* test for analysis of data.

Results

Three children on cow's milk formula treated with lactase, one on breast milk treated with lactase, and one on breast milk had colic that was so severe that it was impossible to continue feeding for the planned time. None of the four milks differed significantly from the others in the percentage of days with colic (Table 1). Combining the milks into lactose containing and lactase treated groups showed no difference ($p>0·05$). Children had colic more often on cow's milk formulas (regardless of lactose) than on breast milk ($p<0·05$).

Table 1 *Days with colic/days on different milks in 10 patients with infantile colic. Roman numerals refer to the order of administration (I=first, II=second, etc)*

Case No	*Milk feed*			
	Breast milk	*Breast milk treated with lactase*	*Formula*	*Formula treated with lactase*
1	5/6 I	6/7 II	7/7 IV	7/7 III
2	3/7 III	1/3 IV	6/7 II	5/6 I
3	5/6 I	3/7 II	9/9 IV	5/5 III
4	6/6 I	6/7 II	7/7 IV	3/3 III
5	4/5 II	5/5 I	4/5 IV	5/5 III
6	1/7 IV	2/7 III	6/7 I	6/7 II
7	7/7 III	7/7 I	7/7 IV	3/3 II
8	4/7 I	1/7 IV	4/7 II	1/7 III
9	6/7 I	7/7 II	7/7 III	7/7 IV
10	3/3 IV	4/5 I	6/7 II	3/3 III
Total	44/61	42/62	63/70	45/53
Mean %	74·7	67·1	89·4	88·3

Two way analysis of variance: $F=2·958$, $df=3/27$, $p>0·05$.
Student's *t* test: cow's milk formulas *v* breast milks, $p<0·05$; lactose containing milks *v* lactase treated milks, $p>0·05$.

Table 2 *Duration and severity of colic: total hours with colic/No of symptomatic days (difficult colic in parentheses). The longest duration of each child is in bold*

Case No	*Milk feed*			
	Breast milk	*Breast milk treated with lactase*	*Formula*	*Formula treated with lactase*
1	0·6 (0·2)	3·4 **(3·2)**	**5·2** (1·6)	1·2 (1·0)
2	0·6 (0)	1·5 (0)	1·5 (0)	**2·8 (1·6)**
3	0·5 (0·5)	0·1 (0)	0·9 (0·7)	**3·8 (3·8)**
4	8·1 (7·3)	4·3 (2·5)	6·9 (3·3)	**17·0 (17·0)**
5	**5·2** (1·6)	4·6 (1·0)	0·8 (0·8)	4·3 **(3·5)**
6	0·2 (0·2)	0·2 (0·2)	0·1 (0·1)	**0·4 (0·3)**
7	1·5 (1·5)	1·1 (1·0)	1·0 (0·6)	**2·2 (2·2)**
8	0·7 (0·6)	2·0 (2·0)	**2·9 (2·9)**	1·8 (0)
9	**2·8 (0·8)**	1·5 (0·4)	1·9 (0·6)	1·2 (0·4)
10	**5·5 (5·5)**	2·7 (2·2)	1·2 (0·9)	3·5 (3·3)

Two way analysis of variance: F for overall colic=1·8, df=3/27, p>0·05; F for difficult colic=1·9, df=3/27, p>0·05.
Student's *t* test: cow's milk formulas *v* breast milks, p>0·05; lactose containing *v* lactase treated milks, p>0·05.

There were no differences between the milks in daily duration of colic (Table 2). Not even regrouping cow's milk formulas versus breast milks or lactose containing versus lactase treated milks showed any differences. Severity of attacks was also unaffected. In five children the total duration of colic and in six children the duration of difficult colic was, however, longest on cow's milk treated with lactase.

Discussion

The aetiology of infantile colic may be multiple, different causes operating in different children and perhaps one child's colic even being a result of several factors. To minimise the effects of confounding factors we conducted this investigation as a crossover study.

Colic was present on 89% of the days on cow's milk feeding and on 71% of the days on breast milk feeding. This difference was significant ($p<0·05$), but 71% is still high: colic is not cured by breast milk. No differences were found regarding severity of colic on those days when the disease was observed. Healthy infants may have physiologic lactase deficiency during several weeks,[6] but infantile colic is not a symptom of lactose malabsorption. Other factors than milk must be crucial.

We are grateful to Valio Ltd for donation of formulas and treatment of milks with lactase.

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Correspondence to Dr M-R Ståhlberg, Department of Paediatrics, Turku University Hospital, SF-20520 Turku, Finland.

Received 7 August 1986

Somatomedin C deficiency in Asian sisters

M E MCGRAW, D A PRICE, AND D J HILL

Department of Child Health, Royal Manchester Children's Hospital, Manchester, and Department of Paediatrics, University of Sheffield, Children's Hospital, Sheffield

SUMMARY Two sisters of Asian origin showed typical clinical and biochemical features of primary somatomedin C (SM-C) deficiency (Laron dwarfism). Abnormalities of SM-C binding proteins were observed, one sister lacking the high molecular weight (150 Kd) protein.

In 1966 Laron *et al* first described a form of familial dwarfism characterised by the clinical features of severe growth hormone (hGH) deficiency and high concentrations of plasma immunoreactive hGH.[1] Low plasma somatomedin activities before and during treatment with hGH indicate that the primary defect is in the production of somatomedin.[2] Most reported cases of this autosomal recessive disorder are in oriental Jews,[3] although a few patients of European origin have been described.[2] We report two Asian sisters with Laron dwarfism