Fig. 2 Plasma terbutaline levels (ng/ml) expressed as mean $\pm$ SEM ($n=5$) for low dose and high dose terbutaline.

Discussion

This study demonstrates a highly significant improvement in peak flow rate when terbutaline is given in a higher dose adjusted for weight than that used in adults. Peak plasma levels within a range known to be non-toxic in adults⁴ were reached 2 hours after the dose and only one patient complained of slight headache during this part of the study.

The lower dose also improved PEFR but not significantly. This is contrary to the findings of Leegaard and Fjulsrud⁵ who used this dosage in a larger number of children. The failure to show significance in the present study may be related to the few children taking part and the fact that they were studied within 24 hours of stopping intravenous therapy for severe wheeze. Further studies are

required to assess the optimum absorption of the drug in such conditions.

Maximum bronchodilatation as measured by PEFR did not correspond directly to maximal plasma levels; this is not surprising since the response in the lung will relate more directly to the amount of free drug present locally and the state of reactivity of the β receptors, in addition to other mucosal factors within the airways.

This study demonstrates that it is safe and effective to give children terbutaline in a dose of 0.25 mg/kg (maximum 5 mg) in asthma.

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Chloride deficiency syndrome due to chloride-deficient breast milk

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SUMMARY A case of dietary chloride deficiency syndrome in a fully breast-fed infant is described. The mother's milk was found to be deficient in chloride and the infant's symptoms resolved on cows' milk formula.

The dietary chloride deficiency syndrome in infants is characterised by anorexia, failure to thrive, and hypokalaemic metabolic alkalosis.¹ First described

in 1979² the condition resulted from the ingestion of Neo-Mull-Soy subsequently found to be deficient in chloride. Recently a single case was reported in a breast-fed infant; the mother's milk was found to be deficient in chloride.³

This report describes a further case of dietary chloride deficiency syndrome in a fully breast-fed infant. The mother's milk was found to be deficient in chloride and the infant's symptoms resolved completely once the feeds were changed to cows' milk formula.

Case history

The patient, a 7-week-old boy, presented with a history of vomiting all feeds for one day. He had been born after a term uncomplicated pregnancy, weighed 2850 g at birth, and was the first child of a 17-year-old mother. The infant was exclusively breast fed and had previously been well. On examination he was 5% dehydrated and his weight was on the 3rd Boston centile. His initial serum acid base status showed a partially compensated metabolic alkalosis (pH 7.47, P_{CO_2} 55 mmHg, base excess +13.6 mmol/l, standard bicarbonate 36.3 mmol/l), with serum electrolytes as follows: Na 125 mmol/l, K 3.6 mmol/l, Cl 63 mmol/l. A diagnosis of hypertrophic pyloric stenosis was considered but a barium meal showed a normal stomach and gastric outlet with no evidence of pyloric stenosis. He was rehydrated with intravenous isotonic saline and given cows' milk formula feeds.

By the next day he was much better. His electrolytes had returned to normal and his acid base status was improved (pH 7.40, P_{CO_2} 54 mmHg, base excess +6.9 mmol/l, standard bicarbonate 29 mmol/l). He was discharged without a firm diagnosis having been made and the mother was advised to continue breast feeding.

Three weeks later he returned to hospital with a history of excessive vomiting for one day. He was again 5% dehydrated and had a severe metabolic alkalosis with very low serum electrolytes (pH 7.50, P_{CO_2} 56 mmHg, base excess +17 mmol/l, standard bicarbonate 40.2 mmol/l, Na 118 mmol/l, K 2.7 mmol/l). A chest x-ray film showed the features of a bilateral bronchopneumonia. He was admitted for rehydration with intravenous isotonic saline and antibiotics. His vomiting stopped within 24 hours, his acid base and serum electrolytes returned to normal within 48 hours, and his pneumonia resolved. Random urinary electrolytes determined before his serum electrolytes had returned to normal showed undetectable levels of sodium and chloride and a potassium concentration of 36 mmol/l. The mother denied having administered any diuretics to the infant. All investigations including a skull x-ray film, electroencephalogram, and isotope brain scan looking for evidence of an intracranial lesion as a cause of vomiting were negative. The patient thrived in the ward and had no further episodes of hypoelectrolytaemia while on a cows' milk formula. He was discharged and the mother asked to bring him to a follow-up clinic.

Two weeks later his mother complained that he had been anorexic for one day but had not vomited. He had been fully breast fed since leaving the hospital. His serum electrolytes showed severe hyponatraemia.

His urinary electrolytes on this occasion were Na 2 mmol/l, K 39 mmol/l, Cl 0. Tests of adrenal function showed a normal 17-hydroxyprogesterone and 11-hydroxylation index, a plasma cortisol of 567 nmol/l, and a very increased plasma aldosterone level (1193 nmol/l) indicative of hyperaldosteronism. Sweat electrolytes determined when the patient was well were not raised. Electrolyte determination on three separate samples of the mother's breast milk were as follows: Na 4, 4, and 9 mmol/l; K 12, 14, and 13 mmol/l; and Cl undetectable on all three occasions.

The infant was put on to cows' milk formula feeds. His symptoms resolved and his electrolyte disturbance corrected within 48 hours. On discharge his mother was advised to continue with the formula feed. He has remained well and at age 1 year all his growth parameters are just below the 50th Boston centile.

Discussion

Metabolic alkalosis with hypoelectrolytaemia and dehydration in infancy is unusual. It is seen most often in association with excessive vomiting secondary to a surgical condition such as pyloric stenosis. This diagnosis was considered initially but excluded on the basis of a normal barium study. Other conditions which can result in this combination of biochemical abnormalities include thiazide diuresis, Bartter's syndrome, pseudohypoaldosteronism with renal salt wasting,⁴ vomiting secondary to intracranial lesions, cystic fibrosis, and deficient dietary chloride. Hypochloraemic alkalosis in infants after exchange transfusion with blood containing acid-citrate-dextrose solution as preservative is also well described.⁵

Administration of diuretics to this child was considered but careful questioning failed to show a source of any such agent in the family. The urinary findings with low sodium and chloride concentrations and the prompt response to treatment in this patient effectively ruled out this possibility and that of Bartter's syndrome and renal salt wasting.⁶ The high aldosterone levels were believed to be a response to the low serum sodium concentration.

A normal skull x-ray film, isotope brain scan, and electroencephalogram were inconsistent with a diagnosis of an intracranial lesion, and the subsequent course of the infant finally ruled out such a possibility. Cystic fibrosis may present initially with metabolic alkalosis and hyponatraemia⁷ but was an unlikely diagnosis in view of the normal sweat electrolytes.

The diagnosis of dietary chloride deficiency was eventually considered. The undetectable chloride

levels in the mother's milk and the complete resolution of symptoms and biochemical abnormalities once the infant's feeds were changed to a formula preparation confirmed the diagnosis.

The characteristic clinical features, the laboratory findings, and the probable pathophysiology of the condition have been well described by Grossman *et al.*¹ In their series all infants manifested anorexia, failure to thrive, microhaematuria, and low urinary chloride concentrations and most had metabolic alkalosis and hypokalaemia. The infant reported here presented with vomiting and dehydration on two occasions, and anorexia on another. He had not thrived well and had the typical serum and urinary biochemical findings but did not have microhaematuria.

There are few data regarding the daily chloride requirements in infancy. The recommended minimum chloride concentration for infant formulae (11 mmol/l) is based on the average levels found in human milk.⁸ No explanation for the absence of chloride in this mother's breast milk was apparent. She appeared clinically healthy, was not on any medication (for example, diuretics) which might have affected the composition of her milk, and refused to allow further investigations to be undertaken on herself.

To our knowledge ours is only the second case of the dietary chloride deficiency syndrome so far reported in a breast-fed infant. We support the recommendation of Asnes *et al.*³ that the diagnosis be considered in all breast-fed infants who fail to

thrive. In addition it should be considered in all infants who have metabolic alkalosis and hypoelectrolytaemia particularly if the urinary chloride concentration is very low.

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Making heel pricks less painful

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SUMMARY A mechanical lancet, the Autolet, was compared with a manual heel prick in 36 newborn infants undergoing routine blood sampling for the Guthrie test and hypothyroid screening. Each method was equally effective in obtaining satisfactory blood samples but the Autolet was considerably less painful.

It is common practice to take blood samples for laboratory examination in the newborn infant by heel prick. Small volumes of blood can often be analysed by micro methods, making venepuncture, which may be difficult and requires skill, unnecessary.

The usual technique is to prick the infant's heel manually with a sterile metal stylet and then to squeeze gently until enough blood has been obtained. It is painful for the baby, and more than one prick may be needed to obtain enough blood, particularly if performed by an inexperienced operator. Many mothers also find the procedure distressing.

A mechanical device for capillary sampling, the Autolet (Owen Mumford, Woodstock, Oxford)¹ has recently become available. This device has been used successfully by diabetic children monitoring their own blood glucose at home.^{2,3} The stylet is placed in a spring-loaded cartridge which is held against the skin. When the spring is released, the stylet pierces