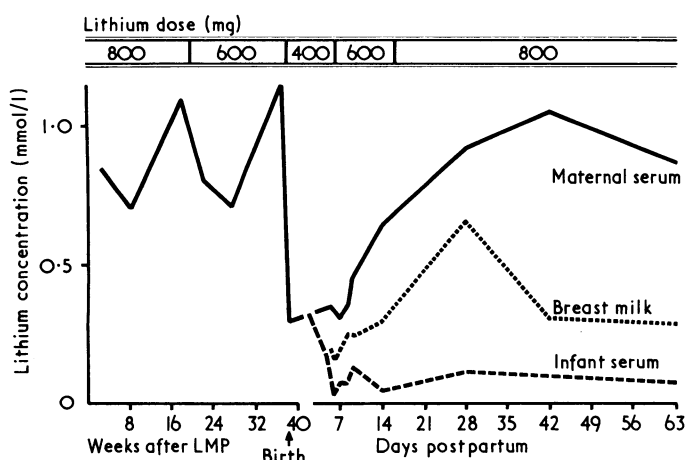


Lithium carbonate and breast-feeding

Lithium carbonate taken during pregnancy has been associated with neonatal hypotonia¹ and congenital heart disease.² There is little information on lithium concentrations in human breast milk or in the serum of breast-fed infants. We have studied such a case.

Case report

A 36-year-old woman, gravida 1, para 0+1, had been taking lithium carbonate for manic depressive psychosis for seven years when pregnancy was confirmed at eight weeks. The pregnancy was allowed to continue. Initially maintained at 800 mg daily, the dose was reduced twice during pregnancy to maintain therapeutic serum levels (see figure). Her mood was more stable than at any previous time, and she needed no other medication apart from routine haematinics. At 38 weeks she went into spontaneous labour lasting 12 hours and received protective forceps for suspected prematurity.



Concentration of lithium in maternal serum and milk and in infant serum.

Six hours before delivery she was given pethidine hydrochloride (Pethilorfan) 100 mg and promazine hydrochloride 50 mg. A boy was born weighing 3450 g. He was mildly hypotonic for the first two days. An electrocardiogram showed nothing abnormal, and the blood count and blood sugar level were normal; the lower femoral epiphysis was present.

The mother was anxious to breast-feed and this was established within six days. Lithium concentrations in the serum of the mother's pooled breast milk and the baby's urine were all monitored closely. The mother's serum level fell over the time of delivery, and the oral dose was doubled to achieve satisfactory serum levels. The baby's level was similar to the mother's at delivery but fell rapidly to 0.030 mmol/l by the sixth day and then rose slightly once breast-feeding was established. Despite a considerable rise in the mother's serum and breast milk levels there was no appreciable rise in the baby's serum level. He thrived and developed normally. Serial 12-hour collections of his urine on days six to nine inclusive gave lithium concentrations of 0.57, 1.20, 0.45, 0.64, 0.29, 0.30, 0.63, and 0.50 mmol/l. The mother became less anxious to breast-feed and stopped during the tenth week. Tests of thyroid function and bone chemistry were then normal.

Comment

The similar serum lithium levels for mother and baby at delivery confirmed that there is free exchange across the placenta.³ The baby's serum level of lithium fell rapidly in the first week of life as reported.³ The mean urinary concentration was 0.57 mmol/l, which was almost 10 times the mean serum level, and this shows that the neonatal kidney is capable of excreting lithium against a concentration gradient. Breast-milk lithium levels were about half maternal serum levels and rose with an increase in the oral dose. Despite the rise in concentration achieved in breast milk, the baby's serum levels remained constantly low—much lower than the level to which he had been exposed during pregnancy. Breast-feeding was discouraged and finally stopped at 10 weeks because of the known inhibition by lithium of cyclic 3'5' adenosine monophosphate⁴ and the theoretical risk to the developing brain.

Since the mother was being treated for manic depressive psychosis we thought that the act of breast-feeding might be therapeutic. The baby will require further close follow-up, but the benefits from breast-feeding appeared to outweigh any possible risk from lithium in the neonatal period.

We thank Sister L Curtis, Mrs A Sanderson, and the biochemistry laboratory for their help.

¹ Strothers, J K, Wilson, D W, and Royston, J, *British Medical Journal*, 1973, **3**, 233.

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³ Mackay, A V P, Loose, R, and Glen, A I M, *British Medical Journal*, 1976, **1**, 878.

⁴ Singer, I, and Rotenberg, D, *New England Journal of Medicine*, 1973, **289**, 254.

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Meningitis caused by group R haemolytic streptococci

We report a case of meningitis caused by infection with group R haemolytic streptococci, probably the first case to be described in Britain.

Case report

A 62-year-old man cut his hand while operating a machine smeared with bacon fat at a pork pie factory. That evening he felt unwell, began to sweat, and developed rigors. Next morning he complained of pain in both hips, and was admitted to a local cottage hospital. The same day he became drowsy and developed meningism. He was transferred to the district general hospital. Lumbar puncture confirmed bacterial meningitis (white cell count $1.5 \times 10^9/l$ (1500/mm³), mainly neutrophils, with Gram-positive diplococci, proteins 2.1 g/l (210 mg/100 ml), and glucose 2.69 mmol/l (48 mg/100 ml)). Culture of the cerebrospinal fluid grew a beta-haemolytic streptococcus on blood agar. The organism was resistant to bacitracin and grew on MacConkey's medium. The isolate could not be grouped with antisera for groups A, B, C, D, E, F, or G streptococci. The Cross-infection Reference Laboratory reported the organism to be a member of group R which had failed to grow in the presence of 10% bile or 4% NaCl at pH 9.6 or at 45°C and did not resist heat at 60°C for 30 minutes. Arginine and esculin were hydrolysed, the Voges-Proskauer test result was negative, and polysaccharide was not formed from sucrose. Acid was produced from trehalose, lactose, raffinose, salicin, inulin, sucrose, and melibiose but not from sorbitol, mannitol, arabinose, melezitose, or dulcitol after five days. A similar organism was isolated from the blood. Purulent fluid was aspirated from the left hip joint, but no organism was cultured probably because the patient had already received several doses of penicillin.

He was treated with penicillin G 10 000 units intrathecally and 2 million units intravenously every two hours for the first 12 hours, then 1 million units intravenously four hourly for seven days. He improved considerably within 48 hours of starting treatment. His final recovery was complicated by a deep vein thrombosis. On discharge from hospital the only residua were high-tone deafness in the left ear associated with some vertigo.

Discussion

Haemolytic streptococci were implicated in septicaemic infections in pigs in 1954 by Field *et al*¹ and by de Moor² in 1959. They continued to be isolated from pigs and piglets in England and elsewhere in Europe. In Denmark in 1968 Perch *et al*³ recorded two cases of