Short reports

Oral contraceptives and breastfeeding: haematological effects on the infant

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SUMMARY Severe clinical and haematological manifestations of folate deficiency occurred in a previously healthy, fully breast fed, 10 month old infant whose mother took oral contraceptives.

Oral contraceptives may interfere with intestinal folate absorption, with resultant folate deficiency.¹ We describe folate deficiency in a 10 month old fully breast fed infant whose mother took oral contraceptives.

Case report

A 10 month old boy was admitted after three weeks of recurrent vomiting and diarrhoea and severe deterioration of his nutritional status. He was born at term (birthweight 3600 g) and until his present illness he had been a healthy and thriving infant growing on the 90th centile. He was fully breast fed. On hospital admission, the baby was pale, listless, and had generalised oedema. He had angular stomatitis and a beefy red tongue with atrophy of the papillae. The skin had a yellowish tint, and there were areas of hyperpigmentation, mainly over the legs. The hair was sparse, reddish, and lusterless. There was evidence of recent loss of subcutaneous fat, and the abdomen was distended. Neurological and developmental evaluation was normal. A blood count showed macrocytic anaemia: haemoglobin 7 g/dl, mean corpuscular volume 101 fl, with macrocytosis of the erythrocytes and hypersegmentation of the neutrophils in the peripheral blood smear. A bone marrow aspirate showed active haemopoiesis, with appreciable megaloblastic changes and increased iron deposition. Serum lactic dehydrogenase activities were very high at 2270 U. Serum iron concentration was 78 µg/dl and total iron binding capacity was 336 µg/dl. The serum vitamin B₁₂ concentration was 176 pg/ml, and the red cell folate concentration was 40 ng/ml (normal 120 ng/ml). Stool examinations for bacterial and

parasitic pathogens were negative, and roentgenographic studies of the gastrointestinal tract were normal.

The baby's mother was in excellent nutritional condition, and had a normal blood count. Her peripheral blood smear, however, showed hypersegmented neutrophils, and her red cell folate concentration was 94 ng/ml. She had begun taking oestrogen-progestin pills three months after delivery.

Treatment of the baby consisted initially of intravenous nutrition supplemented with folic acid. Five days later there was reticulocytosis (14%) and the patient's clinical condition was substantially improved. Oral feeding could gradually be instituted, with addition of oral folic acid 5 mg daily. After 28 days in hospital the baby was sent home, having attained a remarkable clinical recovery. There was no evidence of macrocytic anaemia, but oral iron was prescribed because of a drop in serum iron to 36 µg/dl, and the occurrence of microcytosis and hypochromia on the blood smear.

Discussion

While iron deficiency is recognised as the most common nutritional deficiency and the main cause of anaemia in infancy, a finding of macrocytic anaemia in infants beyond the first 3 to 4 months of life requires careful investigation. Folate deficiency is considered the major cause of megaloblastic anaemia in the young,² and requires a search for the underlying causative or contributory factors.² In our patient, no such factors were found. He was born at term and had been a healthy and normally growing infant until his episode of gastroenteritis three weeks before admission. Also, he had received no drugs that could have interfered with folate absorption and utilisation.¹⁻³ He presented, however, with the full clinical and haematological picture of severe folate deficiency. This is unlikely to have developed during his relatively mild illness of three weeks' duration, because a much longer period is required

for folate deficiency to become overtly manifest.⁴ We must, therefore, assume the existence of an aggravating factor which could cause a longstanding depletion of folate in this infant. The marginally low red blood cell folate concentration with incipient haematologic manifestations of folate deficiency in the mother cannot by itself be regarded as the cause of folate deficiency in her infant.⁵

Our patient's mother had taken an oestrogen-progestin oral contraceptive regularly since her baby was 3 months old; these hormones are excreted in breast milk, and may impair folate absorption in the gut of the infant. A rapidly growing, fully breast fed baby may thus be deprived of his barely adequate folate input, and the stage is set for frank folate deficiency. This sequence of events may explain the development of severe folate deficiency in our patient. There are a good number of published reports on the relation between oral contraception and folate deficiency, but the association of oral contraception in the lactating mother and folate deficiency in her infant is presented here for the first time.

'Contraceptive pills with estrogen/progesterone' are listed among the drugs that are 'usually compat-

ible with breast feeding'.⁶ With the present day trend towards prolonged breast feeding, the concurrent use of oral contraceptives is likely to become more prevalent. Our report points out a potential hazard of maternal oral contraception to the breast fed infant.

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Should hepatitis B surface antigen positive mothers breast feed?

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SUMMARY Breast fed infants may be at greater risk of mother to infant hepatitis B virus infection compared with formula fed infants. We studied 85 infants born to 84 hepatitis B surface antigen positive mothers (only two of whom were hepatitis B e positive), and who had received immunisation against hepatitis B virus. Our results indicate that breast feeding does not increase the risk of developing hepatitis B virus infection in infants born to these mothers if immunisation is carried out.

A vaccine against hepatitis B virus is now available, and infants born to carrier mothers are one of the groups with immunisation priority as perinatal infection frequently results in the carrier status.¹⁻³ Previous studies have shown that this vaccine is safe and effective in these infants;⁴ however, whether the efficacy of the vaccine differs in breast and formula

fed infants born to infected mothers is not known. This is possible as breast feeding may be a mechanism for mother to infant transmission.^{2 5 6} In this study we show that passive-active immunisation allows infants born to mothers who are hepatitis B virus surface antigen positive, hepatitis B e negative to be breast fed without any added risk.

Materials and methods

Mothers. Eighty five infants born to 84 asymptomatic mothers who were hepatitis B surface antigen positive on routine prenatal screening were studied. The presence of hepatitis B surface antigen was detected in mothers' sera in two occasions, two months apart. In addition hepatitis B e antigen and antibodies and hepatitis B core antibodies were investigated.

Infants. The term infants studied (47 boys and 38 girls) were all born after a normal pregnancy and