CASE REPORT

Hypnozoites in Bradford

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SUMMARY

We present two paediatric cases of a condition that should be considered when a child of an immigrant family presents to the A&E department with a pyrexia with no obvious focus of infection.

CASE 1

A 3-year-old Asian boy presented to hospital in June 1987 with a 24 h history of anorexia and vomiting associated with a high temperature. The General Practitioner had diagnosed a chest infection and prescribed a course of amoxil. However, when the child continued to vomit the parents took the child to hospital. The child had a previous history of asthma and had last been abroad to Pakistan nine months previously, with no malarial prophylaxis.

On examination the child was alert, temperature was 38.8°C and there were petechiae over the left side of his neck. The spleen was 3 cm palpable. Haemoglobin was 10.8 g%, WBC 4.900/mm³ and platelets 52,000/mm³. Examination of the blood film revealed *Plasmodium Vivax*. Chloroquine sulphate 200 mg was given orally, then 100 mg six hours later and daily on the subsequent three days. This was followed by a two week course of primaquine 7.5 mg daily to destroy parasites in the liver (hypnozoites) and prevent relapses. Glucose-6-phosphate dehydrogenase assay was normal.

The child made an uneventful recovery and was discharged following the course of chloroquine.

CASE REPORT 2

A 3-week-old baby boy was admitted in June 1987 with a history of failure to gain

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weight since birth and pyrexia for four days. He was bottle fed and had not been feeding well for two days before admission. He was born at full term by normal vaginal delivery (birth weight 3.14 kg, 10th centile) in Bradford.

The parents were immigrants from Pakistan, his mother having come to Britain in July 1985. Since arrival in Britain she had not travelled abroad. In October 1986, the mother had a pyrexial illness for four days. On admission, the baby looked unwell and undernourished. Weight on admission was 3.17 kg (3rd centile). Axillary temperature was 39°C. There was mild icterus and pallor. Liver and spleen were both 2 cm palpable. He was irritable with a high-pitched cry. Investigations showed Hb 10.9 g%, WBC 10,500/mm³, platelets 43,000/mm³, normal urea and electrolytes. Total serum bilirubin was 70 μ mol/litre, due to haemolytic anaemia.

Lumbar puncture was not performed because it was found that the baby's peripheral smear was heavily infested with malarial parasites, identified as *Plasmodium Vivax*. Repeated examinations of thick and thin blood films from the mother were reported negative for malarial parasite. The baby was given oral chloroquine sulphate 75 mg then 37.5 mg six hours later followed by 37.5 mg daily on the subsequent three days. He started gaining weight after three days at a rate of 50–60 g/day. Irritability improved. A repeat blood film, after three days of treatment with chloroquine, was negative for malarial parasites.

The mother was given a 14-day course of primaquine (following a normal glucose-6-phosphate dehydrogenase assay). Primaquine was not required for the baby because congenital malaria is a transfusion malaria and the exoerythrocytic (hepatic) cycle is absent.

DISCUSSION

It is unusual in vivax malaria for relapse to occur more than three years after the patient has left the endemic area. However, relapse can occur up to eight years (Manson's Tropical Diseases, 18th Edition).

Although malaria is one of the commonest infectious diseases in the world, congenital malaria is uncommon even in areas where malaria is endemic. In 1986, 2309 cases of imported malaria were reported in Britain, of which only one was congenital malaria (Malaria Reference Office). So far only nine cases of congenital malaria have been documented in literature in Britain (Davenport, 1986).

The mechanism of transmission of congenital malaria is unclear. It is widely believed that in endemic areas congenital malaria is rare due to the presence of protective maternal IgG antibodies which cross the placenta. The natural course of malaria results in at least partial immunity developing by adulthood. Transplacental malaria primarily occurs in babies of poorly immune mothers (Logie & McGregor, 1970).

The features of congenital malaria in reported cases have included fever, jaundice, hepatosplenomegaly, pallor (due to haemolytic anaemia) and thrombocytopenia. Irritability, lethargy and anorexia are noted in the later stages. Birth weight and early feeding pattern are normal.

Convulsions occur rarely, except in cerebral malarial. Fever is reported in all cases.

Other complications include vomiting, diarrhoea, electrolyte imbalance, and nephrotic syndrome in plasmodium malariae infection. The usual age of presentation is 3–6 weeks at which time passive immunity from maternal IgG antibodies starts declining. Our case presented at the age of 3 weeks with failure to thrive and pyrexia. A high index of suspicion is necessary to make the diagnosis. In most reported cases there has been either a clear-cut maternal history of malaria during pregnancy or the diagnosis was made on routine examination of peripheral blood film (Marshall, 1983) as in our case. There have been reports of convulsions and death following intramuscular injections of chloroquine, this route should therefore be avoided (Quin *et al.*, 1982).

It is necessary to treat the mother with primaquine whether her peripheral blood smear is positive or not.

Both case reports emphasize the need to consider the diagnosis of malaria even if the child or mother have not been abroad for some considerable time. Although not occurring in these patients it is important to remember that malarial prophylaxis is only relative protection and that the disease can still occur in spite of adequate prophylaxis.

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