pancreatic ducts, and islet tissue supported by a stroma of abundant smooth-muscle fibres (see Figs. 1 and 2). The tumour was confined to the muscle coat of the pylorus, and the overlying mucosa was normal. The kidneys showed mild lymphocytic infiltration of the stroma and no evidence of renal tubular calcification.

Comment

The lesion under discussion is a heterotopia of pancreatic tissue. This is not an uncommon finding. In various series of necropsy material the incidence is estimated at from 0.04% to 15% of examinations (Feldman and Weinberg, 1952). Most cases have been reported in adults.

Pearson (1951) collected details of aberrant pancreatic tissue in all sites in 589 cases. Of these, 25% were in the stomach and 30% were in the duodenum. In most cases the abnormality was an incidental finding at necropsy.

Pancreatic heterotopia as a cause of pyloric obstruction in children would appear to be extremely rare. Collett (1946), in an extensive review of the literature, traced 471 reported cases of aberrant pancreatic tissue at all ages: 46 (10%) occurred in children; of these, five were situated in the stomach and one other was a polypus on the lesser curvature of the stomach which had prolapsed through the pylorus and caused obstruction (Mouat, 1925-6). Collett described a second case of pyloric obstruction in a boy aged 6 who had suffered from attacks of colicky abdominal pain and vomiting since birth. The symptoms were relieved after local excision of a prepyloric polypus which contained pancreatic tissue.

In the cases described by Mouat and by Collett the obstruction was caused by a polypus in the pyloric canal, unlike the case under discussion, in which the heterotopia was situated in the wall of the pylorus itself and caused obstruction largely by virtue of associated hypertrophy of smooth muscle. The occurrence of smooth-muscle hypertrophy in relation to aberrant pancreatic tissue has been noted on many occasions and is discussed by Krieg (1941).

There are many theories concerning the origin of pancreatic heterotopia, and the most generally accepted is that of Warthin (quoted by Pearson, 1951), who suggests that pancreatic ducts may be snared off by the upward and downward growth of the intestine.

In most cases pyloric pancreatic heterotopia is unsuspected in life. In those patients with symptoms, attention has been drawn to various syndromes, including vague abdominal discomfort or more definite symptoms suggesting peptic ulceration, gall-bladder disease, or pyloric obstruction. Radiological studies may be equivocal or may suggest carcinoma; in some instances a polyp in the region of the pylorus has been demonstrated. More rarely, the presenting symptoms may be the result of one or more of the complications which are known to occur in ectopic pancreatic tissue at any site—for example, acute pancreatic oedema, acute haemorrhagic pancreatitis, and neoplastic change. Rarely, an islet-cell adenoma in ectopic tissue has been responsible for hyperinsulinism and hypoglycaemia.

In the patient under discussion the exact mechanism of death is uncertain, and it is difficult to correlate the degree of pyloric obstruction with the relatively short history. The clinical history also serves to illustrate the important point that a patient suffering from a marked functional disability may, in fact, have a serious organic lesion.

Summary

Clinical and pathological details are recorded of a 5-year-old child who at necropsy was found to have pyloric obstructions due to pancreatic heterotopia. References to previous causes in the literature are briefly discussed.

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CONGENITAL MALARIA IN ENGLAND— PLASMODIUM OVALE

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Congenital malaria in England is rare, the last case being reported by Dimson (1954). A review of the literature shows that only five cases have been reported in this country, three (Jones and Brown, 1924; Tanner and Hewlett, 1935; Gammie, 1944) being caused by *Plasmodium vivax* and two (Moffat, 1907; Dimson, 1954) by *Plasmodium falciparum*. There is no case attributed to *Plasmodium ovale*.

A further case is reported, that of an infant delivered by caesarean section and infected with *P. ovale*. The diagnosis was made 21 days after birth.

Mother's History

The mother of the patient first attended the antenatal clinic on May 6, 1955. She was a gravida-two, aged 25, and was expecting her baby on September 24.

For the previous two years she had been living in East Africa—from January 9, 1953, to January 29, 1955—and had been delivered on October 21, 1953, of a full-term healthy female child weighing 7 lb. 14 oz. (3.6 kg.) by lower segment caesarean section. Apparently the operation had been carried out because of uterine inertia and the possibility of mild pelvic disproportion. The child developed normally and returned to this country with her parents.

The mother and father had been living in a malarious zone, but the mother did not develop a malarial attack until January 21, 1955, eight days before she returned to the United Kingdom. She had been an inconsistent taker of proguanil, the probable reason for her infection. Her husband and her child, both of whom had been maintained strictly on a suppressive dose of proguanil, did not develop malaria. The attack in January, 1955, was confirmed at Amani, the headquarters of the East African Institute of Malaria and Vector-borne Diseases, by finding trophozoites in the peripheral blood films. The type of malarial infection was identified as malignant tertian; *ovale* parasites were not seen in these films. She was treated with a therapeutic course of chloroquine sulphate ("nivaquine")—200-mg. tablets being given daily for six days in the dosage of 6, 5, 4, 3, 2, and 1. This was followed by a suppressive course of proguanil (100 mg. daily for one month); she had no more attacks until September 25, eight days after she had been admitted to hospital.

On her first antenatal examination no abnormality was found except a slightly raised blood pressure (130/80) and a suggestion of pitting oedema of the ankles. The fundal height was consistent with a pregnancy of 20 weeks. As the pregnancy advanced the blood pressure became stabilized at 120/80, the ankle oedema did not increase, and the urine remained clear. No major pelvic abnormality was revealed by x-ray pelvimetry.

She was admitted to hospital on September 17, after she had experienced a few contractions. Examination of the abdomen on this date showed the head to be high, and pelvic examination revealed that the cervix was not taken up and did not admit a finger.

Whilst in hospital the presentation changed repeatedly, and on September 26, owing to continued slight irregular contractions and an unstable presentation, a lower segment caesarean section was carried out.

During the evening of the 25th, a day before delivery, her temperature rose to 102° F. (38.9° C.). Previously it had been normal except for two minor increases on September 18 and 23. No abnormal physical signs were to be found to explain the pyrexia other than a herpetic rash on the upper lip. On this occasion the white-cell count was normal and the urine sterile. The peripheral blood was not examined for parasites.

The operation on September 26 was straightforward. It was noted that the placenta was posterior in position and in no way damaged by the uterine incision. At the time malarial infection of the mother had not been suspected: had it been, blood films from the placenta and cord might have proved most informative. Following delivery the baby responded rapidly after the mouth and nasopharynx had been cleared by suction. The child was a male weighing 8 lb. 13 oz. (4 kg.) and was apparently healthy.

The mother's temperature rose to 103.5° F. (39.7° C.) the day after delivery. It did so again two days later, the interval between these two pyrexial attacks being 48 hours. Blood films were taken and malaria was confirmed by the presence of ring forms and disintegrating trophozoites. The temperature peaks suggested benign tertian malaria. Therefore she was given a therapeutic course of chloroquine sulphate followed by a suppressive one for a month. No further rise of temperature was recorded after chloroquine sulphate therapy had been started.

Case Report

The baby appeared well and for three days was breast-fed. Then he developed a pubic rash. For this he was treated with systemic penicillin, potassium permanganate baths, and local aqueous gentian violet 0.66%. It was decided to discontinue breast-feeding. On October 5 the baby's haemoglobin was 114%, the white-cell count was normal, and parasites were not seen in the peripheral blood films. Because of the rash the baby was retained in hospital after the mother's discharge on October 15.

On October 17, 21 days after delivery, when the rash had almost completely cleared, the baby developed more symptoms in the early evening. He refused his 6 p.m. feed, made grunting and whimpering noises, and appeared pallid. His temperature was 104° F. (40° C.), his pulse 140, and respiratory rate 50. No abnormality was found in the cardiovascular, respiratory, or central nervous systems, and the spleen was not palpable. His general condition indicated an early pulmonary infection. In spite of this, since malaria was now suspected, blood films were taken again and treatment was begun immediately with erythromycin and proguanil. The proguanil was to be discontinued if the blood films were negative. His temperature was normal and his condition satisfactory the following morning.

The peripheral blood films revealed malarial forms—trophozoites. Proguanil was continued (0.25 g. daily for five days) and followed by a therapeutic course of chloroquine sulphate (½ tablet twice daily for two days, then ½ tablet daily for two days). A suppressive course of chloroquine sulphate (½ tablet weekly for three weeks) was then begun. The day following the attack the haemoglobin was found to be 83%, falling to 68% in five days, but rising to 76% in a further five days. This increase was attributed to the administration of oral ferrous sulphate which was started the day after the attack. He was discharged on October 30, apparently well, and weighing 10 lb. 2 oz. (4.6 kg.). He has since been seen twice, and on the last occasion (December 12, 1955) he was in excellent general health and had gained weight steadily.

Diagnosis.—The films were later sent to Mr. P. G. Shute, assistant director of the Ministry of Health Malaria Reference Laboratory, who diagnosed a *P. ovale* infection and reported as follows: "Half-grown forms too compact for *P. vivax*. Host cells less swollen than usual for *P. vivax*. Prominent and coarse stippling typical of *P. ovale*. Male and female gametocytes seen."

Commentary

Although congenital malaria is a rarity in England, it was considered that, following the discovery of parasites in the mother's blood, transmission might be more likely because of possible operative trauma at the placental site. There is no information about the factors favouring transplacental transmission or at what time during gestation it occurs.

In this case the clinical picture simulated a pulmonary infection. That congenital malaria might present as a pulmonary disturbance has been shown by Jemma (1936) and Perves (1946). It was felt that, whilst we were awaiting blood film reports, treatment with both proguanil and erythromycin was justified. The erythromycin was prescribed for the possible pulmonary infection because the prolonged course of penicillin given for the skin condition made it unlikely that the invading organisms would be penicillin-sensitive. Here it should be stated that it would have been better to use another drug instead of proguanil for the infant's acute malarial attack, for, though an excellent prophylactic, it is a slow schizonticide.

Eight months earlier the mother had had a malarial attack which had been apparently efficiently treated. In spite of this she developed a further attack at full term. It is suggested that either she was reinfected before leaving the country or she was incubating a *P. ovale* infection when the malignant tertian became apparent in 1955. Chloroquine sulphate treatment would not eradicate this, for it does not destroy the pre-erythrocytic and exo-erythrocytic forms of the parasite developing in the liver.

Transplacental transmission does occur in exceptional cases (Covell, 1950). Whether the trauma of caesarean section or the previous uterine contractions caused some intermingling of the maternal and foetal red blood cells at the placental site, or whether the placental barrier was crossed before this, is a matter for conjecture. Since England is not an endemic area transmission must have been via the maternal sinuses.

Summary

A case of congenital malaria due to *Plasmodium ovale* in an infant delivered by caesarean section is reported.

Successful treatment with chloroquine sulphate and proguanil is described.

The history of malarial infection of the mother enabled the correct diagnosis of congenital malaria to be made.

The danger of the condition being mistaken for an early pulmonary infection is discussed.

I thank Mr. J. M. Bowen, consultant obstetrician, and Dr. T. A. Brand, consultant paediatrician, for their permission to publish this case and for their subsequent help; Dr. J. Fine, consultant pathologist, Mr. P. G. Shute, assistant director of the Ministry of Health Malaria Reference Laboratory, and Mr. G. Webbe, entomologist to the medical department, Tanganyika, for their help in the confirmation of *P. ovale* infection.

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As the war years recede, fewer war-blinded men and women come to St. Dunstan's for rehabilitation and training; yet 40 new cases were admitted to St. Dunstan's during the past 12 months. The majority were casualties from the second world war and subsequent engagements, but 19 were men who served in the first world war who had gone blind gradually through the years from the delayed effects of mustard-gas poisoning.