Malignancy and herpes zoster affect the same age groups. Herpes occurs with greater frequency than does cancer.

The series of 54 cases of malignancy and 81 of herpes zoster do not show any tendency for malignancy to develop following herpes. In the two cases where herpes and cancer occurred in the same person the herpes followed the diagnosis of cancer and suggests that the herpes is caused by irritation of the posterior nerve root either by the original cancer or by concealed secondaries in the spinal column. It could also, of course, be unrelated.

Conclusion

A study of 81 cases of herpes zoster and 147 cases of chicken-pox occurring over a period of seven years failed to show any epidemiological association between the two diseases. While neither showed substantial sex or urban/rural variations, the diseases differed widely in age groups involved, in susceptibility, and in the annual and seasonal variations observed over this time.

The study of 54 cases of cancer and 81 cases of herpes zoster over a similar period does not suggest that a herpes zoster infection has any carcinogenic properties. In fact, in the only two cases in which both diseases occurred the cancer antedated the herpes.

My thanks are due to my assistant, Dr. T. C. Morton, for help in getting this information out and to my secretary, Mrs. Potts, for care with my notes.

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PYLORIC OBSTRUCTION DUE TO PANCREATIC HETEROTOPIA IN A CHILD

BY

D. MacKINNON, M.D.
Consultant Pathologist

AND

F. W. NASH, M.D., M.R.C.P.
Consultant Paediatrician

Merthyr and Aberdare Group of Hospitals

Pyloric obstruction in childhood is rarely encountered except within the first few months of life. The following is an account of a child aged 5 years who died and in whom pyloric obstruction due to pancreatic heterotopia was discovered at necropsy.

Case Report

A girl aged 5 years 2 months was admitted to hospital in December, 1954. There was a history of recurrent attacks of vomiting for approximately one month, and at the onset of her illness she had suffered from dysuria and frequency of micturition. Since infancy the child had been undernourished and caused her parents considerable anxiety on account of frequent refusal to take food. This was regarded as part of a functional disturbance—for during a previous prolonged hospital admission for atopic dermatitis she was emotionally very upset and suffered from mutism.

On admission the patient was underweight (28 lb. 2 oz.—12.75 kg.) and was slightly dehydrated. She appeared extremely nervous and had badly bitten fingernails. There was an extensive monilia infection of the mouth; the urine contained numerous pus cells, and *E. coli* was cultured from the urine. There was a mild normochromic anaemia.

The thrush and urinary infection responded rapidly to treatment and the general condition improved. As the child was emotionally disturbed it was decided to discharge her home. However, before this could be done she developed a relapse of the urinary infection and the vomiting recurred. In the early hours of January 10, 1955, the child vomited copiously, collapsed, and died quite suddenly.

Post-mortem Examination.—The external appearances were those of a poorly nourished female child. There was bilateral pulmonary oedema. The air passages were clear. The stomach was dilated and there was an obvious tumour at the pyloroduodenal junction. On opening the stomach it was found that the pyloric canal would only admit a probe, 3 mm. in diameter. The pyloric tumour, which almost completely encircled the pyloric canal, measured 1 cm. in thickness and 1.6 cm. in its long axis. It was a hard tumour, and the cut surface resembled that of a leiomyoma. The other organs appeared normal to the naked eye. Microscopy showed the tumour to consist of pancreatic glandular tissue,

FIG. 1.—Section of the pyloric tumour which illustrates the marked muscle hyperplasia. ($\times 10$)

FIG. 2.—Higher-power view of the pyloric tumour showing pancreatic tissue supported by a muscle stroma. ($\times 50$)

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

BY

H. G. JENKINS, M.B., B.S.

Late Senior House Officer to the Obstetric and
Gynaecological Departments, the County
Hospital, Griffithstown, Mon

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.