accuracy depends on maintaining the ultrasound equipment in optimal condition, paying great attention to anatomical detail, and considerable experience. Experience is required both in obtaining appropriate scans (by active "interrogation" of the patient) and in interpreting the images. The relative importance of these different aspects of experience and the difficulties in teaching them have not yet been established.

This study has shown that grey-scale ultrasonography has important diagnostic and management roles in patients with chronic pancreatic disease, as has been established in patients with acute pancreatitis and evolving pseudocysts.^{13 14}

Where do these results leave other diagnostic techniques? Pancreatic function tests are occasionally important, but imaging methods are more discriminating. Pancreatic isotope scanning has been discarded in this unit,¹ and angiography is used only in highly selected cases. Computerised axial tomography provides good images of the pancreatic outline¹⁵ and is preferable to ultrasonography in obese patients, in whom ultrasound is particularly difficult,¹⁶ but preliminary comparisons have shown no other convincing advantage over ultrasonography.¹⁷⁻¹⁹ We find that ultrasonography gives better overall diagnostic discrimination; in our unit computerised tomography of the pancreas is reserved for a few problem patients, often those with mass lesions who have undergone surgery.

Ultrasonographic diagnosis is based on the appearance of the organ outline and its parenchyma. It is therefore well complemented by invasive techniques that outline the duct system, such as ERCP²⁰ and percutaneous transhepatic cholangiography in patients with jaundice. Like others who have studied this topic,²¹⁻²⁴ we now recommend the combination of grey-scale ultrasonography and ERCP.²⁵ The ultrasonography is performed first, and this diagnosis may be sufficient if it is confidently normal in a patient in whom the clinical suspicion of pancreatic disease is low. In most other cases ultrasonography is followed by ERCP to complete the documentation and give information about the duodenal loop and papilla of Vater.

Ultrasound technology is advancing rapidly, and high-resolution "real-time" (moving-picture) systems will soon be in general use. Images of diagnostic quality are becoming easier to achieve, and developments should decrease the degree of operator dependence. These advances should consolidate the position of pancreatic ultrasonography and allow the technique

to be widely adopted as an easy and early diagnostic method in the study of pancreatic problems.

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SHORT REPORTS

Delayed haemorrhagic disease of the newborn with extradural haematoma

In breast-fed newborn infants there is an increased risk of haemorrhagic disorders associated with vitamin K deficiency.¹ Beyond the neonatal period the aetiological role of breast-feeding is less clear. In those cases where symptomatic vitamin K deficiency has occurred there have often been other predisposing factors such as previous antibiotic treatment or prolonged diarrhoea.^{2 3} We describe a breast-fed infant who had received no vitamin K after delivery and who developed a bleeding tendency at four weeks, later resulting in an extradural haematoma.

Case report

A 6-week-old baby girl presented with a two-week history of bruising and bleeding from the palate. Her mother was a 38-year-old Greek Cypriot, gravida three, with thalassaemia minor. She was induced at 37 weeks gestation for mild hypertension. Delivery was normal, and the birth weight 2.2 kg. The baby was not given vitamin K after delivery. Apart from mild transitory jaundice, the neonatal period was uneventful. She had been entirely breast-fed up to the time of presentation. There was no history of antibiotic treatment or episodes of diarrhoea. She was a pale baby, with

bruising of her left hand and right side of her face. Apart from an umbilical hernia, there were no other positive findings. Investigations gave the following results: haemoglobin concentration 4.9 g/dl, reticulocytes 16%, WBC 9×10^9 /l (9000/mm³), normal platelets, prothrombin time >5 minutes, kaolin cephalin clotting time >5 minutes, and normal liver function tests. Clotting factor assays were performed: II <1%, V >150%, IX <3%, X <1%. These results confirmed a diagnosis of vitamin K deficiency leading to haemorrhagic disease. She was treated with fresh frozen plasma, whole blood, and vitamin K. Repeat Hb was 13.9 g/dl and the clotting screen 24 hours later was normal.

Over the next 48 hours she became febrile and irritable. A bacteriological screen, including lumbar puncture, was negative. Within a further 24 hours she had developed acute neurological symptoms—bulging fontanelle, widening of the sutures, conjugate deviation of her eyes to the left, and, finally, generalised seizures. A repeat lumbar puncture obtained uniformly blood-stained fluid, but bilateral subdural taps were negative. An electroencephalogram showed greatly reduced activity on the left side. Computerised axial tomography (CAT scan) four days after admission showed a large left occipital extradural haematoma (figure a). This was drained surgically and she made an uneventful postoperative recovery. She had no further seizures and her clotting screen remained normal. She has been followed up at regular intervals. At the age of 5 months her general level of development appeared normal. It was possible, however, to demonstrate a right homonymous hemianopia and difficulty in conjugate deviation of her eyes to the right. Spontaneous movement was reduced in the right arm and leg. A repeat CAT scan (figure b) showed atrophy in the posterior half of the left hemisphere.

(a) CAT scan showing large left-sided haematoma. (b) Repeat scan four months later showing cerebral atrophy on left.

Comment

Haematological investigations confirmed a diagnosis of vitamin K deficiency in this infant. She was a breast-fed, low birth-weight infant who had not been given vitamin K after delivery. There was no history of either diarrhoea or antibiotic treatment. Acute neurological complications have been reported² but we can find no mention of any similar long-term neurological deficit. Maternity units vary greatly in their practice of giving prophylactic vitamin K, but there are strong grounds at least for giving it to infants who will be breast-fed. This infant's extradural haematoma and subsequent neurological handicap might have been prevented had vitamin K been given soon after birth.

We thank Dr A Piesociwz, Mr C E Polkey, Dr R D Hoare, and Professor R J Robinson for permission to report this case.

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Positive antinuclear factor tests with prazosin

Prazosin is a vasodilator and potent hypotensive agent.¹ Postural hypotension after the first dose² and occasional dizziness on long-term treatment¹ are the most important symptomatic side effects. We have

investigated 57 hypertensive patients treated with prazosin and report for the first time a high incidence of positive antinuclear factor (ANF) tests in patients given this drug.

Patients, methods, and results

A total of 107 hypertensive patients were tested for ANF. Forty-seven of them had been treated with prazosin, a thiazide diuretic, and beta-blocking agent; 25 others had taken first a thiazide diuretic and beta-blocker for over a year; and 35 had previously been untreated. Ten of these 35 were given prazosin alone. ANF was measured by an immunofluorescence technique in all the patients who had taken prazosin. The measurement was repeated in follow-up. In those with positive tests DNA binding and serum complement levels were measured and LE cells sought. The sera from 10 patients with positive and 10 with negative tests were submitted with code numbers to the Department of Haematology, the Royal Postgraduate Medical School.

The first ANF test was positive in 12 (7 men, 5 women aged 55-73) of the 47 patients who had taken prazosin with a diuretic and beta-blocker (table). The duration of treatment with prazosin and dose ranged from 6 weeks to 25 months (mean 8.5 months) and 6-15 mg daily (mean 12.5 mg). At the time of this first assessment nine of the 12 with positive tests were still taking prazosin but three had stopped taking it seven weeks, 12 weeks, and 12 months previously, respectively. ANF titres ranged from 1/10 to 1/320. Four patients had titres of 1/10, four of 1/20, two of 1/40, one of 1/160, and one of 1/320. DNA antibodies ranged from 1-23% (mean 6.7%) (normal range 0-20%). Prazosin was withdrawn from all 12 with positive tests and the ANF titre measured again four months later. In six it was unchanged, in two it was negative, and in two it had increased from 1/20 to 1/40 and 1/160 respectively. Out of 35 patients who continued with prazosin four developed positive ANF tests for the first time. One of them had been negative after 19 months' treatment, and again after 22 months, and had a positive titre of 1/80 after 27 months' treatment. None of the 25 patients treated with a beta-blocking agent and thiazide diuretic alone and none of the new hypertensive patients had a positive test. It had become positive in three of the 10 new patients beginning prazosin treatment. Their titres were 1/20, 1/40, and 1/80 respectively. Conversion occurred after three to four months of treatment with prazosin 6-15 mg daily. In all patients with positive ANF tests serum complement levels were normal and LE cells were not found. Our positive tests were confirmed by the Royal Postgraduate Medical School laboratory.

Comment

Out of the 57 patients who took prazosin 19 developed a positive antinuclear factor. In each case it was the speckled type and not associated with high DNA binding, abnormal serum complement, or LE cells. These features are unlike those of systemic lupus erythematosus and, indeed, no patient developed an SLE-like syndrome. Cairns and Jordan³ reported one patient treated with prazosin who developed acute febrile polyarthritis which reappeared on restarting the drug, but the ANF test was negative. Our discovery of positive ANF tests in patients who were taking prazosin in combination with a diuretic and beta-blocker first suggested the possible association with prazosin. No positive tests were found in a control group taking a diuretic and beta-blocker. The association was confirmed by the development of a positive test in three of 10 patients treated with prazosin alone. Conversion of a negative to a positive ANF test was seen as early as three months and as late as two years after beginning treatment.

An LE-like syndrome is associated with other peripheral vasodilator drugs, notably hydralazine and procainamide. LE cells appear before the onset of symptoms and may persist after drug withdrawal.⁴ Although none of our patients had symptoms of an autoimmune illness we recommend that any new symptoms in patients taking prazosin should be investigated.

We thank Mr R I Palmer, Mr J Edwards, and Dr G L Scott for their assistance, and Miss E Rowbottom for measurements of antinuclear antibodies done in the Department of Immunology, Royal Postgraduate Medical

Antinuclear factor (ANF) in patients taking prazosin

Treatment group	Patients (No)	ANF + ve (No)	Continued treatment	Patients (No)	Follow-up ANF + ve (No)
Diuretic + beta-blocker + prazosin	47	12	{	Prazosin stopped	12
Diuretic + beta-blocker	25	Nil		Prazosin continued	35
Untreated	35	Nil		Unchanged	25
				Prazosin alone	10
				Other treatment	25
					Not repeated
					3
					Not repeated

ANF + diuretic + beta-blocker + prazosin compared with diuretic + beta-blocker $P < 0.005$ (Fisher's exact test).

*Four months after stopping prazosin.