tions in the appearance or function of a modified spray that had been exposed to 150 cycles of a 150°C autoclave as well as 50 cycles of a 134°C autoclave.

Probably many other hospitals may need to pay close attention to this unnecessary, potentially dangerous source of hospital-acquired infection.

We thank Miss Anna King for technical help, and Dr G T Spencer and Dr W D Wylie for their advice.

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(*Accepted 18 April 1977*)

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Acute pancreatitis after translumbar aortography

Investigative procedures should ideally have minimal attendant risks to the patient, but acute pancreatitis is an occasional (2-4%) complication of endoscopic retrograde pancreatography and a less well-recognised complication of translumbar aortography (TLA). We describe here the diagnosis and management of three patients who developed acute pancreatitis after this angiographic investigation and present the results of screening 50 consecutive patients for clinical and biochemical evidence of pancreatitis after TLA.

Incidence of pancreatitis and management

TLA is a necessary investigation in some patients with peripheral vascular disease and is performed under general anaesthesia during a 48-hour admission. In 1974 we encountered one patient with acute pancreatitis after TLA, and the next case occurred early in 1976. In April to October 1976 50 consecutive patients were routinely screened for hyperamylasaemia after TLA and carefully observed for any clinical signs of acute pancreatitis. None developed either clinical or biochemical evidence of the disease, but in the eight weeks after this survey one further patient suffered from acute pancreatitis within a few hours of TLA. In the three years under review about 700 patients have undergone TLA. The incidence of acute pancreatitis complicating this investigation is therefore less than 0·5%.

All three patients suffered severe abdominal pain and vomiting within hours of completing TLA. The first two patients were managed conservatively throughout their illness but concern that aortic dissection might account for the hyperamylasaemia in the third patient resulted in laparotomy. An inflamed pancreas with associated fat necrosis was found with no significant damage to the aorta. No definitive surgery was performed but peripancreatic drains were placed. Only one patient (case 1) showed the flank staining indicative of aortic extravasation, which has a more medial location than the Grey Turner's sign of acute pancreatitis.

Hyperlipoproteinaemia was not a causal factor. One patient had biliary disease and another a heavy alcohol intake. Hypoalbuminaemia, hypocalcaemia, and arterial hypoxaemia were features in each patient (see table). All three patients received humidified oxygen as part of their management because of their arterial hypoxaemia, and in other respects they were managed conservatively without the addition of aprotinin or glucagon.

Comment

TLA probably induces acute pancreatitis by direct damage to the body of the pancreas where it crosses anterior to the aorta at the level of L 1. In two of our three patients the aortic entry was at the level of T 12-L 1, which is consistent with the needle having traversed the pancreas. In our unit we now prefer to puncture the aorta at the level of L 2-3, using a 16-18 BWG steel needle to inject Angiografin or Conray 480 dye at an injection pressure of 0·35-1·1 kgf/cm^3.

Hypocalcaemia was associated with considerable hypoalbuminaemia in each case, indicating that the fall in total calcium was directly related to the fall in serum albumin values.[1] The hypoxaemia was corrected to satisfactory levels by humidified oxygen via a Hudson mask in all three patients without the need for assisted ventilation.

Patients being investigated for peripheral vascular disease often suffer from widespread atheroma with associated myocardial ischaemia. It is therefore gratifying that all the patients in this rather poor risk group survived. This type of pancreatitis was probably iatrogenic in origin (despite the presence of other causal factors in two patients) because of the short time interval from aortography to the clinical presentation of acute pancreatitis. The low incidence of this complication of aortography suggests that it is sufficient to check serum and urine amylase concentrations in the few patients who become symptomatic in the hours after aortography.

We acknowledge with thanks the invaluable help of Sister Coull (peripheral vascular unit, Belvidere Hospital) in completing this study.

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(*Accepted 22 April 1977*)

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Values in patients with acute pancreatitis

Case No	Age	Peak serum amylase (U/l)	Lowest values			Fasting		Hospital stay (days)
			Calcium (mmol/l)	Albumin (g/l)	Pao_2 (kPa)	Cholesterol (mmol/l)	Triglyceride (mmol/l)	
1	63	6500	1·95	28	8·0	4·3	1·45	19
2	55	2200	1·90	32	7·6	7·7	1·30	17
3	57	4000	2·05	29	6·7	5·3	2·0	25

Conversion: SI to traditional units—Calcium: 1 mmol/l ≈ 4 mg/100 ml. Pao_2: 1 kPa ≈ 7·5 mm Hg. Cholesterol: 1 mmol/l ≈ 39 mg/100 ml. Triglyceride: 1 mmol ≈ 88 mg/100 ml.

Haemochromatosis presenting as angina and responding to venesection

Congestive cardiac failure, pericarditis, arrhythmias, and conductive defects are well-recognised cardiac manifestations of idiopathic haemochromatosis. Angina pectoris may be the presenting symptom but is less well recognised. A case is described with the results of venesection and a seven-year follow-up period.

Case report

A 38-year-old butcher presented in 1969 with a six-month history of angina on moderate exercise, palpitations, and dyspnoea on severe exercise. He did not smoke and drank two pints of beer per week. His family history

had no relevance to his illness. He was a non-pigmented normotensive man with an enlarged (6 cm below costal margin) firm non-tender liver, and was otherwise normal. Apart from minor abnormality of liver function tests the results of other relevant laboratory investigations were normal. The serum iron was 57·7 μmol/l (322 μg/100 ml) and 100% saturated transferrin. The resting electrocardiogram (ECG) showed S-T depression segments in standard leads II and III, and leads V5 and V6 with symmetrical T wave inversion in V5 and V6. Histological examination of a specimen obtained by liver biopsy showed established cirrhosis with heavy iron deposition within the hepatic cells and fibrous septa.

Regular phlebotomy was undertaken. At 12 months (25 1 venesected) the patient continued to note angina on moderate exercise. His liver enlargement had decreased, the serum iron was 44·7 μmol/l (250 μg/100 ml), and the resting ECG and liver function tests (LFT) were normal. At 30 months (60 1 venesected) he had no symptoms even on strenuous exercise, but mild hypochromic microcytic anaemia was present (Hb 10 g/dl, serum iron concentration 7·1 μmol/l (39·7 μg/100 ml). This improvement has continued, apart from a period when he defaulted from treatment for 18 months, at which stage angina returned and again responded to venesection.

Discussion

Death from cardiac causes is a common outcome of untreated haemochromatosis.[1] This is usually due to congestive cardiomyopathy, which may respond to venesection.[2] Angina as a presenting complaint has rarely been recognised,[3 4] and no reports have dealt with the effect of long-term treatment. In this patient the resting electrocardiogram and abnormalities of liver function returned to normal after 12 months' venesection, while the angina did not completely respond until venesection had been carried out for a total of 30 months. The necessity of regular continuous venesection is emphasised by the return of symptoms after default.

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(*Accepted 3 May 1977*)

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Decreased urinary oestriol concentrations in pregnant women during hexamine hippurate treatment

Hexamine hippurate (Hiprex) and hexamine mandelate (Mandelamine) have long been widely used in treating urinary tract infections, but are now mainly used for long-term treatment or for preventing recurrent urinary tract infections. The drugs have few side effects and may be used safely during pregnancy and the puerperium. We have observed that oestriol concentrations in the urine of pregnant women fall sharply soon after starting treatment with hexamine hippurate, although plasma oestriol concentrations and fetal well-being are not affected by the drug.

Methods and results

We studied five pregnant women, who were in hospital throughout the investigation until delivery. We collected 24-hour urine samples daily before and during treatment with hexamine hippurate. Urinary oestriol was measured by Oakey's modified technique.[1] Venous blood samples were taken daily, and serum human placental lactogen (HPL) was determined by radioimmunoassay using the Radiochemical Centre HPL immunoassay kit (Amersham, Buckinghamshire, England). Daily measurements of serum unconjugated oestriol were also performed by radioimmunoassay using a CIS kit (Département des Radioéléments, Gif-sur-Yvette, France).

Oestriol concentrations in the mothers' urine fell to below normal values (28-170 μmol/24 h (8-50 mg/24 h)) immediately after starting hexamine hippurate treatment, whereas plasma oestriol and HPL concentrations showed no important changes. The patients were treated until delivery, and all the newborn infants were healthy.

Comment

Measurement of urinary oestriol is widely used for assessing fetoplacental function and fetal well-being, especially in complicated or high-risk pregnancies. Some factors, however, may cause diminished oestriol values without being indicative of impending fetal death.

Ingestion of corticosteroids[2] by the mother probably suppresses oestrogen excretion in pregnancy by inhibiting corticotrophin secretion from the fetal pituitary gland. Ampicillin[3] and phenoxymethylpenicillin[4] may reduce urinary excretion and the plasma concentration of oestriol either by causing oestriol biosynthesis in the fetoplacental unit to diminish, or by impairing the permeability of the placenta to oestrogens. Possibly ampicillin also interferes with the enterohepatic circulation of oestriol resulting in increased excretion in the faeces.

Both hexamine hippurate and hexamine mandelate[5] interfere with measurements of urinary oestriol but not with plasma oestriol determinations. Formaldehyde, a breakdown product of hexamine hippurate due to hydrolysis in an acid urine, combines with the phenolic oestrogens to form a substance which is nonreactive in the colorimetric procedure of Oakey.

Because of its simplicity and reliability, Oakey's method is still suitable for the routine evaluation of fetoplacental function. To eliminate misleading results, however, maternal factors should be carefully evaluated before collecting urine samples.

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(*Accepted 20 April 1977*)

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Urinary and plasma oestriol and human placental lactogen concentrations in five pregnant women before and during treatment with hexamine hippurate. Ranges of values from onset of treatment until delivery are given in parentheses

Patient No	Duration of pregnancy (weeks)	Urinary oestriol (μmol/24 h)		Plasma oestriol (nmol/l)		Human placental lactogen (mg/l)	
		Before	During	Before	During	Before	During
1	28	50	0 (0–12)	64	109 (109–315)	4·1	5·5 (5·5–6·8)
2	35	48	3 (1–23)	90	46 (47–86)	4·5	3·9 (3·6–5·0)
3	34	75	3 (3–9)	55	49 (66–89)	5·0	6·4 (6·2–6·7)
4*	36	56	5	86	80	4·1	3·9
5†	31		4–29		63–216		4·9–7·2

*Delivery occurred immediately after start of treatment.
†Treatment started on admission to hospital.
Conversion: SI to traditional units—urinary oestriol 1 μmol/24 h ≈ 0·29 mg/24 h; plasma oestriol 1 nmol/l ≈ 28·84 ng/100ml.