The liquor was offensive and the placental swab subsequently grew *Proteus* spp. and *Escherichia coli*. The I.U.D. was expelled with the fetus.

Two hours after delivery the blood pressure fell to 70/50 mm Hg, despite the blood transfusion. She was centrally cyanosed; the pulse was rapid and feeble. The central venous pressure (C.V.P.) was plus 12 cm water and the urine output for the preceeding 12 hours had been only 400 ml. The antibiotic was changed to intravenous cephaloridine together with intravenous hydrocortisone. The patient's condition then rapidly improved. The blood pressure, C.V.P., and urinary output all returned to normal levels after 10 hours. Cephaloridine was continued for eight days. She was discharged home, well, nine days after admission.

Discussion

Pregnancy associated with an I.U.D. is a potentially serious condition. If the pregnancy is to continue the F.D.A. recommends that the device should be removed if the strings are visible. Removing the device may decrease the incidence of spontaneous abortion.⁴ Furthermore, the insidious onset of symptoms is impressive² and the incidence of other complications is also increased.⁵ There is a great need for more information on I.U.D.s, and a registry system for I.U.D.s such as is being started in the U.S.A. should be considered in Britain.

The value of C.V.P. monitoring in a hypotensive patient is further emphasized here. A C.V.P. reading slightly above normal established that the cause of hypotension was "pump failure" rather than under-transfusion. It was thus the septicaemia which was treated urgently rather than the further administration of intravenous fluids.

I thank Mr. S. Madden for his assistance and permission to publish this case.

¹ U.S. Food and Drug Administration, Press Conference on I.U.D.s and Supporting Documents, Rockville, Maryland, 20852 20 December 1974.

² Christian, C. D., *American Journal of Obstetrics and Gynecology*, 1974, **119**, 441.

³ Hurt, W. G., *Obstetrics and Gynecology*, 1974, **44**, 491.

⁴ Alvoir, G. T., jun., *Obstetrics and Gynecology*, 1973, **41**, 894.

⁵ Stevens, J. D., and Fraser, I. S., *Journal of Obstetrics and Gynaecology of the British Commonwealth*, 1974, **81**, 282.

Royal Infirmary (Sutton), Hull HU8 9HE

A. K. THOMAS, M.R.C.O.G., Registrar in Gynaecology

Pancreatitis in the Hard Water Syndrome

If dialysis fluid used in haemodialysis is made up of hard water unpleasant complications may develop. They include headache, sweating, tachycardia, hypertension, vomiting, and sometimes gastrointestinal haemorrhage. These have been attributed to a rise in plasma calcium and magnesium concentrations.¹ Therefore in hard water areas the tap water required for preparing dialysis fluid should be softened or deionized. Failure of the softening or deionizing system may result in severe hypercalcaemia causing the hard water syndrome.

Case Report

A 23-year-old girl with terminal renal failure due to chronic pyelonephritis was established on home haemodialysis in December 1973. Warm, single-pass dialysis fluid was prepared in a Dylade proportionater by mixing softened water and a concentrated solution of salts.* The usual dialysis fluid calcium concentration was 1.88 mmol/l (3.75 mEq/l) and magnesium 0.5 mmol/l (1 mEq/l). Water was softened by a Permutit domestic water softener, which the patient was instructed to regenerate once a week. Access to blood was via a forearm arteriovenous fistula. For nine months she dialysed thrice weekly without trouble. On 23 October 1974 she was admitted urgently to hospital within a few hours of completing dialysis. She complained of severe headache, nausea, vomiting, central abdominal pain, generalized weakness, and sweating. These symptoms had developed while dialysing. During the penultimate dialysis she had experienced similar though less severe symptoms.

On examination she was afebrile, perspiring, and restless; pulse rate 88/min, regular; extremities warm; B.P. 210/130 mm Hg. Her arteriovenous

*Renalyte; McCarthy's.

Graph showing high plasma amylase associated with high plasma calcium and subsequent parallel reduction in both measurements. D represents haemodialysis.

Conversion: SI to Traditional Units—Creatinine: 1 mmol/l ≈ 0.0113 mg/100 ml. Calcium: 1 mmol/l ≈ 4 mg/100 ml. Phosphate: 1 mmol/l ≈ 3.1 mg/100 ml.

fistula had thrombosed. There were epigastric guarding and rebound tenderness, normal bowel sounds, and no abdominal distension or free fluid. All tendon reflexes were exaggerated. Fundi normal. Haemoglobin was 6.1 g/dl, white blood count $8.8 \times 10^9/l$, blood urea 11.6 mmol/l (70 mg/100 ml), serum creatinine 530 mmol/l (6.0 mg/100 ml), sodium 128 mmol/l, potassium 5.5 mmol/l, bicarbonate 36 mmol/l, calcium 4.9 mmol/l (19.6 mg/100 ml), serum phosphate 1.3 mmol/l (4.1 mg/100 ml), alkaline phosphate 42 IU/l, protein 72 g/l, albumin 39 g/l, amylase 2838 Somogyi units/100 ml, serum alamine aminotransferase 12 IU/l, bilirubin 12.0 mmol/l (0.7 mg/100 ml). Magnesium was not measured. E.C.G. showed much shortened QTc intervals. Erect and supine abdominal radiographs showed gaseous distension, no fluid levels, and no pancreatic calcification.

The patient admitted that during the past three to four weeks she had neglected to check the hardness of the water before dialysis and had not regenerated the water softener. Probably, therefore, she had been exposed to a high dialysis fluid calcium concentration during at least two dialysis periods. When checked, the calcium concentration of the unsoftened tap water was 3.1 mmol/l (12.3 mg/100 ml) and the magnesium 0.4 mmol/l (1.0 mg/100 ml). She was dialysed in hospital for the next week using a leg Scribner shunt. Plasma calcium and amylase levels rapidly fell to normal and the symptoms resolved over the next three to four days (see fig.). Barium meal examination and a cholecystogram were subsequently found to be normal.

Discussion

The symptoms caused by the hard water syndrome have been attributed to hypercalcaemia and hypermagnesaemia.¹ Mixter *et al.*² reported pancreatitis in 11 cases of hyperparathyroidism and reviewed 51 previous cases from the literature. Hypercalcaemia was present in nearly all their cases though it was not always a reliable clue to pancreatitis. Hypercalcaemia might induce hypertension, and Drukker³ considered this to be a possible cause of the headaches, nausea, and vomiting. In hypercalcaemia associated with hyperparathyroidism intravascular coagulation has been noted and microthrombi have been seen in pancreatic and renal vessels.⁴ Perhaps the clotting in our patient's AV fistula was related to a state of hypercoagulability. The effect of hypercalcaemia on the conversion of trypsinogen to trypsin may also be important.⁵

We thank Drs. A. R. Ford, M. R. Baker, and T. Duffy who assisted in the clinical care of the patient.

¹ Freeman, R. M., Lawton, R. C., and Chamberlain, M. A., *New England Journal of Medicine*, 1967, **276**, 1113.

² Mixter, C. G., jun., Keynes, W. M., and Cope, O., *New England Journal of Medicine*, 1962, **266**, 265.

³ Drukker, W., *Proceedings of the European Dialysis and Transplant Association*, 1968, **5**, 284.

⁴ Baer, L., and Neu, H. C., *Annals of Internal Medicine*, 1966, **64**, 1062.

⁵ Haverback, B. J., *et al.*, *American Journal of Medicine*, 1960, **29**, 424.

Addenbrooke's Hospital, Cambridge

D. B. EVANS, M.B., M.R.C.P., Consultant Nephrologist

M. SLAPAK, M.CH., F.R.C.S., Honorary Consultant Surgeon