Case 2—A 40-year-old man was given amiodarone because of recurrent supraventricular tachycardia. After two years the drug was stopped because the arrhythmia was well controlled. One month later he showed clinical signs of thyrotoxicosis and had a small non-tender goitre. In-vitro T₃ uptake was 50%. Despite three weeks' treatment with carbimazole 60 mg/day the patient lost 10 kg in weight. On admission to hospital he had tachycardia, was sweating profusely, and had myopathy. Antibodies against thyroglobulin were 1/16 (positive) but were negative against microsomal antigen. Serum T₄ and T₃ concentrations were very high (figure, bottom). Urinary iodine

Serum T₃ and T₄ concentrations: (top) during treatment with prednisone alone (case 1) and (bottom) after addition of prednisone to treatment with carbimazole and propranolol (case 2). As the values in the cases were measured with different radioimmunoassays the absolute figures are not comparable.

excretion was 66 μ mol/day (normal range 0.8–1.6 μ mol/day). The patient was given propranolol and prednisone; carbimazole was continued. During prednisone treatment T₄ and T₃ concentrations fell rapidly. They promptly rose again after the drug was stopped. A second course of prednisone was therefore given and the dose tapered when the hormone concentrations decreased. The goitre had disappeared five months after amiodarone treatment had been stopped; thyroglobulin antibodies were absent and the patient is clinically well.

Comment

Thyrotoxicosis occurred during amiodarone treatment in case 1 and after treatment in case 2. Both patients had a transient, non-tender goitre. During prednisone treatment T₄ and T₃ concentrations fell and the condition of both patients improved. This effect was confirmed when prednisone treatment was interrupted; hormone concentrations rose again and clinical deterioration was evident.

Apparently, prednisone shortens the duration of the rise in concentrations of T₄ and T₃; its action on serum hormone concentrations is much more rapid than that of the hormone synthesis blocking drug carbimazole. Prednisone is of particular value if propranolol fails to control the clinical signs of hyperthyroidism (case 2). In case 1 the patient tolerated prednisone without any recognisable side effects, whereas the patient in case 2 developed transient cushingoid facial signs.

As the use of amiodarone is becoming more and more widespread, a simple and effective treatment for this particular kind of thyrotoxicosis, which is resistant to standard treatment, is needed because

of the long-lasting iodine overload of the thyroid during and after amiodarone treatment.

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- ¹ Savoie JC, Massin JP, Thomopoulos P, Leger F. Iodine-induced thyrotoxicosis in apparently normal thyroid glands. *J Clin Endocrinol* 1975; **41**:685–91.
- ² Jonckheer MH, Broekaert I, Blockx P, Bernard R. Amiodarone et fonction thyroïdienne. *Arch Mal Coeur* 1976; **69**:1315–9.
- ³ Burger A, Dinicheret D, Nicod P, Jenny M, Lemarchand-Beraud T, Vallotton MB. Effect of amiodarone on serum triiodothyronine, reverse triiodothyronine, thyroxine, and thyrotropin. *J Clin Invest* 1976; **58**:255–9.
- ⁴ Blum M, Weinberg U, Shenkman L, Hollander CS. Hyperthyroidism after iodinated contrast media. *N Engl J Med* 1974; **291**:24–5.
- ⁵ Chopra IJ, Williams DE, Origiazzi J, Solomon DH. Opposite effects of dexamethasone on serum concentrations of 3,3',5'-triiodothyronine (reverse T₃) and 3,3',5-triiodothyronine (T₃). *J Clin Endocrinol Metab* 1975; **41**:911–20.

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Overt diabetes mellitus without glycosuria in a patient with cutaneous ureteroileostomy

Though a positive result to a urine test for glucose requires further investigation, a negative result by no means excludes diabetes. The classic cause of a false-negative result is a high renal threshold for glucose, which is present in advanced renal failure. Another cause that is not often remembered is a ureteroenterostomy. We report a case of overt diabetes mellitus without glycosuria in a patient with an ileal conduit and normal renal function.

Case report

A 59-year-old Caucasian man was admitted for investigation with a six-month history of unusual lethargy. Two years previously he had had a radical cystectomy with ileal loop urinary diversion for bladder cancer and had since remained well. He was not taking any drugs; his weight, appetite, urine output, and stoma function were normal. Examination of the heart, lungs, abdomen, stoma, eyes, and nervous system showed no abnormalities. Urinalysis one hour after eating showed a pH of 8, proteinuria +, and no glucose or ketones. Laboratory study showed a haemoglobin concentration of 14 g/dl, a leucocyte count of $5.8 \times 10^9/l$ with normal differentials, a normal platelet count, and an erythrocyte sedimentation rate of 30 mm in the first hour. Plasma sodium concentration was 135 mmol(mEq)/l, potassium 3.00 mmol(mEq)/l, bicarbonate 14 mmol(mEq)/l, urea 6.2 mmol/l (37 mg/100 ml), creatinine 0.11 mmol/l (12.4 mg/100 ml), and chloride 115 mmol(mEq)/l. Blood sugar concentration measured at random was 20.5 mmol/l (369 mg/100 ml), and two fasting concentrations were 17.8 and 16.5 mmol/l (320 and 297 mg/100 ml) respectively. Appearances on chest x-ray and electrocardiogram were normal. His 24-hour urine volume was 1400 ml, creatinine clearance 84 ml/min, and total proteinuria 0.2 g. Results of an excretory urogram were normal. Midstream urine culture grew no pathogens, and daily urine testing for glucose gave negative results throughout his stay in hospital.

He was treated with insulin and potassium supplements and subsequently discharged taking glibenclamide and sodium bicarbonate. Four weeks after discharge his fasting plasma glucose, bicarbonate, and potassium concentrations had returned to normal, and he remained well.

Comment

Though diabetics with an ileal conduit may have glycosuria, total reabsorption of glucose from the loop may sometimes occur.¹ This case is representative of the occasional patient whose urine may be negative for sugar and thereby mislead the unwary clinician. The appearance of glucose in urine normally depends on three factors: the plasma

glucose concentration, the glomerular filtration rate, and the capacity of the renal tubules to reabsorb glucose.² Glycosuria develops in the diabetic because raised blood glucose concentration leads to quantities of glucose in the glomerular filtrate that cannot be reabsorbed. When the ureter has been diverted into the bowel an additional factor operates. The enterocytes tend to reabsorb glucose from urine traversing the loop in proportion to the urinary glucose concentration, the length of time urine remains in contact with the absorptive surface, and the area of intestinal mucosa exposed to urine—the same factors that govern the trading of bicarbonate for chloride ions in the genesis of hyperchloraemic acidosis.

These variable factors may help to explain why glucose reabsorption is complete in some ileal loops but not in others. Furthermore, ileal glucose transport that is carrier-mediated and sodium-dependent³ is significantly enhanced in diabetic patients⁴ and by high luminal glucose concentrations in the rat.⁵

I thank Dr A R Harrison for permission to report this case.

¹ McGouran RCM. Glucose reabsorption from ileal loops. *Br Med J* 1977; ii:932.

² Mudge GH. Tubular transport of urea, glucose, phosphate, uric acid, sulphate, and thiosulphate. In: Orloffs J, Berliner RW, eds. *Handbook of physiology*. Washington, DC: American Physiology Society, 1977: 587-652.

³ Schultz SG, Fuisz RE, Curran PF. Amino acid and sugar transport in rabbit ileum. *J Gen Physiol* 1966;49:849.

⁴ Vinnik IE, Kern F, Sussman K. The effect of diabetes mellitus and insulin on glucose absorption by the small intestine in man. *J Lab Clin Med* 1965;66:131-6.

⁵ Sund RB. Glucose and cation transport in rat jejunum, ileum and colon in vivo. *Acta Pharmacol Toxicol (Kbh)* 1978;42:117-24.

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Strangulated hernias through Tenckhoff cannula sites

Continuous ambulatory peritoneal dialysis has become a well-established method of treating end-stage renal failure since 1978.¹ Though the commonest gastrointestinal problem is the development of peritonitis, abdominal hernias are a well-recognised complication.² We report here two cases of patients on continuous ambulatory peritoneal dialysis who developed strangulated hernias through Tenckhoff cannula sites, a complication we believe not to have been recorded.

Case reports

Case 1—A 64-year-old woman with chronic glomerulonephritis was started on continuous ambulatory peritoneal dialysis on 29 July 1980 and discharged on 30 August 1980 with dialysis running satisfactorily. She was admitted as an emergency on 22 November 1980 with a "blocked catheter," 3.2 l of dialysate having run in and only 300 ml drained; otherwise she was asymptomatic. On examination there was a small, firm, tender subumbilical swelling at the site of peritoneal entry of the Tenckhoff cannula. Bowel sounds were normal. Her catheter was flushed with heparinised saline and urokinase inserted but to no avail. Two days later she started vomiting and developed abdominal distension. By this time the subumbilical mass was tender and erythematous and bowel sounds were obstructive. Abdominal films showed dilated small-bowel loops with fluid levels. Laparotomy was carried out that day, and the small bowel and the cannula were found to be herniating through the peritoneal entry site. Bowel and the intraperitoneal portion of the cannula were lying in the proximal end of the subcutaneous catheter tunnel. The small bowel was necrotic. The hernia was reduced, small-bowel resection carried out, and the catheter resited in the pelvis. Her postoperative course was uneventful and she was discharged on 9 December 1980. She was, however, readmitted four days later with a candida peritoneal infection but failed to respond to treatment. She also developed a chest infection, deteriorated despite treatment, and died on 27 December 1980.

Case 2—A 61-year-old man was started on continuous ambulatory peritoneal dialysis on 14 April 1980 after having rejected a transplant and

being found incompatible with haemodialysis. After multiple peritoneal infections and having his cannula changed twice he was admitted as an emergency on 26 November 1981 with an eight-hour history of lower abdominal pain, nausea, and vomiting. On examination the abdomen was soft and not distended. At the lower end of a small infraumbilical scar was a tense swelling with a questionable cough impulse. Bowel sounds were normal. Abdominal films showed a few dilated small-bowel loops with fluid levels. At laparotomy a necrotic loop of small bowel was found to have herniated through the peritoneum 2 cm below the entry site of his present cannula, presumably through the entry site of a previous one. The hernia was reduced, small-bowel resection was carried out, and the cannula tip resited in the pelvis. His postoperative course was uneventful. He was discharged on 11 December 1981 and has remained well since.

Comment

Though the most common gastrointestinal complication in continuous ambulatory peritoneal dialysis is peritonitis, an appreciable number of patients develop abdominal hernias, of which the most common seem to be incisional.² The two patients discussed here had hernias through Tenckhoff cannula entry sites. The hernial orifices were small and thus not surprisingly strangulation of bowel was a feature in both cases. The only other strangulated hernias reported in patients undergoing continuous ambulatory peritoneal dialysis were umbilical³ and epigastric.²

In our experience of 28 patients undergoing a combined total of 237 months of dialysis, four have developed abdominal hernias—one inguinal, two incisional (in the same patient), and the two strangulated hernias reported here. Both our patients had had their cannulas implanted using the standard introducer for penetrating the peritoneum. We suggest that a non-absorbable purse-string suture should be placed around the cannula on insertion and that an attempt should be made to suture the peritoneum when a cannula is removed.

Though the signs and symptoms may be minimal obstructed hernia should be considered as a cause of abdominal pain in patients undergoing continuous ambulatory peritoneal dialysis, especially if a tender lump is present in a scar.

¹ Popovich RP, Moncrief JW, Nolph KD, Ghods AJ, Twardowski ZT, Pyle WK. Continuous ambulatory peritoneal dialysis. *Ann Intern Med* 1978;88:449-56.

² Khanna R, Oreopoulos DG, Dombros N, *et al.* Continuous ambulatory peritoneal dialysis after three years: still a promising treatment. *Peritoneal Dialysis Bulletin* 1981;1:24.

³ Chan MK, Baillod RA, Tanner A, *et al.* Abdominal hernias in patients receiving continuous ambulatory peritoneal dialysis. *Br Med J* 1981; 283:826.

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Neonatal convulsions caused by withdrawal from maternal clomipramine

Clomipramine is a tricyclic antidepressant particularly useful in treating depression with obsessive or phobic features. Although its use is not recommended in the first trimester, there has been no contraindication to its use in later pregnancy. We describe two infants who developed convulsions due to withdrawal from maternal clomipramine, the second of whom was treated with reducing doses of clomipramine.

Case reports

Case 1—A 22-year-old primigravida who had a depressive illness with obsessional features was treated with clomipramine for the last seven weeks of pregnancy. A male infant was born by spontaneous vertex delivery at term weighing 3420 g with Apgar scores of 7 and 8 at one and five minutes respectively. He had a convulsion at eight hours of age, and intermittent convulsions persisted until he was 53 hours of age despite treatment with parenteral phenobarbitone and paraldehyde. He remained hypertonic and