provides further evidence of the doubtful importance of hyperamylasaemia, even when associated with upper abdominal symptoms; certainly it does not always indicate pancreatitis. Furthermore, the case highlights our reservations towards increasing reports of drug-induced pancreatitis, particularly when the diagnosis is not substantiated by anatomical confirmation.

Regardless of the source of the amylase in this case, we postulate that it was a direct result of theophylline intoxication, and in view of the successful management of theophylline poisoning by resin or charcoal haemoperfusion^{4,5} it is important that this association should be borne in mind. The widespread prescribing of theophylline renders it liable to abuse in self-poisoning, and hence theophylline overdosage should be considered as a possible underlying cause of apparent pancreatitis in patients who also suffer from chronic obstructive airways disease.

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Royal Albert Edward Infirmary, Wigan WN1 2NN

T H S BURGAN, LMS, MRCP, registrar in general medicine (now at: King Khaled Hospital, University of Riyadh, Saudi Arabia)
I GUPTA, MB, MRCPATH, consultant pathologist
C M BATE, MD, MRCP, consultant physician

Acute renal failure in dense deposit disease: recovery after plasmapheresis

Mesangiocapillary glomerulonephritis type II or dense deposit disease may occasionally present as acute renal failure. The prognosis for recovery of renal function in such cases is poor,¹ and to date no therapeutic regimen has proved consistently effective.

Plasmapheresis has been used in the treatment of many glomerular disorders with varying degrees of success,² but to our knowledge use of this technique in the acute forms of dense deposit disease has not been described. We report a case of dense deposit disease associated with partial lipodystrophy that presented with acute renal failure. Serum creatinine concentration returned rapidly to normal after intensive plasma exchange.

Case report

A 29-year-old woman with partial lipodystrophy gave birth to a healthy child after a full-term uneventful pregnancy. Five days after delivery she developed oedema of the eyelids and legs. Two weeks later she noticed dyspnoea, weakness, and decreased urine output and was admitted to hospital. Physical examination showed blood pressure 200/100 mm Hg, pallor, generalised oedema, retinal haemorrhages and exudates, jugular venous distension, a fourth heart sound, liver enlargement, and hepatojugular reflux. Laboratory investigations showed packed red cell volume 25%, white cell count $9.1 \times 10^9/l$ with normal differential, blood urea nitrogen 20 mmol/l (56 mg/100 ml), serum creatinine 388 $\mu\text{mol/l}$ (4.4 mg/100 ml), serum albumin 17.1 g/l, 24-hour proteinuria 8.6 g, microscopic haematuria, and hypocomplementaemia with a pattern suggestive of alternate pathway activation (low C3, normal C4, low total haemolytic complement). A test for C3 nephritic factor gave a positive result. A renal biopsy specimen showed changes consistent with mesangiocapillary glomerulonephritis type II. Eighty per cent of glomeruli showed cellular crescents.

Plasmapheresis (4 l exchanges) was started on the fifth hospital day. She was given prednisone 1 mg/kg/day, and cyclophosphamide 2.5 mg/kg/day. Plasmapheresis was stopped after the 10th session, when the serum creatinine concentration had decreased to 133 $\mu\text{mol/l}$ (1.5 mg/dl). Prednisone and cyclophosphamide were continued. Five months after the initial episode she was asymptomatic, her serum creatinine concentration was 88.4 $\mu\text{mol/l}$ (1 mg/100 ml), and she still had proteinuria (8.8 g/24 h) and microscopic haematuria.

Comment

Plasmapheresis has been used in the treatment of a few patients with the more usual, chronic forms of mesangiocapillary glomerulonephritis. Overall, it has been difficult to substantiate any benefit derived.^{3,4} Our experience suggests, however, that plasmapheresis may be useful in acute forms of dense deposit disease accompanied by extracapillary proliferation. The mechanism by which plasmapheresis improved our patient's renal function is uncertain. Possibly it removed complement, clotting factors, or other mediators of inflammation and thus lessened the kidney's acute proliferative response. Although attractive, this hypothesis cannot be proved in the absence of a follow-up renal biopsy. What seems clear is that plasmapheresis did not affect the basic underlying pathogenesis of the disease, since our patient continued to have haematuria and distinct proteinuria after her serum creatinine concentration had returned to normal.

Obviously no definite conclusions can be drawn from experience limited to a single case. Nevertheless, considering the generally poor prognosis of extensive crescent formation in acute renal failure, it might be worth while trying plasmapheresis in selected patients who have this syndrome associated with dense deposit disease.

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Nephrology Service, Hospital Clinic i Provincial, University of Barcelona, Spain

JESÚS MONTOLIÚ, MD, attending physician
EDUARDO BERGADÀ, MD, attending physician
PILAR ARRIZABALAGA, MD, resident physician
LUIS REVERT, MD, professor of medicine

C-peptide suppression test and sulphonylurea-induced factitious hypoglycaemia

Factitious hypoglycaemia presents particular diagnostic difficulties but may occur as frequently as insulinoma.¹ Failure of suppression of plasma C-peptide concentration during insulin-induced hypoglycaemia is characteristic of insulinoma,² C-peptide being a marker of endogenous insulin secretion.³ This report contrasts the results in two patients who underwent C-peptide suppression tests; one had an insulinoma and the other factitious hypoglycaemia due to glibenclamide ingestion.

Case reports

Case 1—A 44-year-old police officer collapsed at work, having eaten 3½ hours earlier. He was sweating and his blood glucose concentration was 1.9 mmol/l (34 mg/100 ml). He had had two episodes of dizziness and sweating over the previous four months; these occurred at least five hours after food. A vagotomy and gastrojejunostomy had been performed five years earlier for peptic ulcer. An oral glucose tolerance test showed reactive hypoglycaemia; blood glucose concentrations at 0, 30, 60, and 90 minutes were 4.4, 11.7, 5.8, and 2.6 mmol/l (79, 211, 104, and 47 mg/100 ml) and plasma insulin concentrations 10, 188, 29, and 14 mU/l respectively. The patient felt light-headed at the time of the lowest blood glucose concentration. After a 20-hour fast, with exercise, his blood glucose concentration fell to 2.4 mmol/l (43 mg/100 ml) when the plasma insulin concentration was 12 mU/l. In insulin-induced hypoglycaemia plasma C-peptide concentration was not suppressed (figure). Pancreatic arteriography showed a vascular tumour in the tail of the pancreas. This was removed and found to be an islet-cell

tumour; a second oral glucose tolerance test 21 months later again indicated reactive hypoglycaemia.

Case 2—A 43-year-old man was admitted to hospital in a coma and sweating profusely. His blood glucose concentration was 1.9 mmol/l (34 mg/100 ml) and he responded to intravenous dextrose. His wife had diabetes and was taking glibenclamide; he strenuously denied taking any of her tablets. He had had four milder episodes of drowsiness and sweating over the previous seven weeks. After overnight fasting on two occasions he remained normoglycaemic, but after two further fasts his blood glucose concentration fell to 2.9 mmol/l (52 mg/100 ml) and 1.9 mmol/l (34 mg/100 ml) with a plasma insulin concentration on the second occasion of 10 mU/l (assay sensitivity 3 mU/l, 95% confidence limits). Insulin-induced hypoglycaemia indicated abnormal C-peptide suppression (see figure). Several episodes of drowsiness with sweating occurred over the next month. Subselective arteriography of the pancreatic arteries showed a tumour blush in the body of the pancreas which measured 2.5 × 4 × 4 cm. No insulinoma was found at laparotomy, and distal pancreatectomy was performed; no tumour was identified in the resected pancreas. A postoperative C-peptide suppression test again suggested autonomous insulin production (see figure). Repeat pancreatic arteriography

Plasma C-peptide responses to bolus intravenous insulin (at 0 minutes) in case 1 (●---●) and in case 2 before (●—●) and after (●---●) distal pancreatectomy. Hatched area indicates normal response (n=8).

showed no evidence of a tumour. A further hypoglycaemic episode responded to 1 mg glucagon iv with normal blood glucose and insulin responses. A calcium infusion, however, was associated with a fall in blood glucose concentration to 1.9 mmol/l (34 mg/100 ml) and a rise in plasma insulin concentration from 16 to 24 mU/l, thus suggesting an insulinoma.⁴ Radioimmunoassay for sulphonylurea-type drugs in samples from the various investigations showed plasma concentrations of glibenclamide ranging from 107 to 352 µg/l on five of six separate occasions. The patient then admitted to taking his wife's glibenclamide tablets.

Comment

The C-peptide suppression test was particularly useful in case 1, in which the patient had undergone gastric surgery and had evidence of reactive hypoglycaemia. Plasma insulin concentrations at the time of fasting hypoglycaemia suggested a tumour of low functional activity, and his plasma C-peptide concentration was unsuppressed by hypoglycaemia. The C-peptide response in case 2, however, was similar to that in case 1. The value of C-peptide measurements in the diagnosis of hypoglycaemia has recently been reviewed.⁵ Our second case shows that chronic glibenclamide ingestion may lead to failure of normal C-peptide suppression. This, together with difficulties in interpreting pancreatic arteriography,⁶ emphasises that the offending drug must be measured to confirm or refute factitious hypoglycaemia due to sulphonylureas. A high index of suspicion is essential when patients with hypoglycaemia and access to antidiabetic drugs are assessed.

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Derby City Hospital and Derbyshire Royal Infirmary, Derby

J S HARROP, MSC, MRCPATH, consultant chemical pathologist
P R GOLDING, MD, FRCP, consultant physician
P GOODALL, MCHIR, FRCS, consultant surgeon
V M LEVEAUX, MD, FRCP, consultant physician
G A STEELE, MB, CHB, DMRD, consultant radiologist
A R INGLE, MSC, senior biochemist

Department of Medical Biochemistry, Welsh National School of Medicine, Cardiff

S RAINBOW, PHD, biochemist

Importance of thyroxine in suppressing secretion of thyroid-stimulating hormone after thyroidectomy

Serum concentrations of triiodothyronine (T3) correlate well with clinical indices of thyroid hormone activity¹ and are generally considered to be of greater functional importance than thyroxine (T4) concentrations. The inhibition of thyroid-stimulating hormone release by thyroid hormones, however, correlates better with serum concentrations of T4 than with those of T3 in hypothyroid patients receiving T4 replacement therapy.² We have observed two cases which indicate that T3 alone, in serum concentrations close to the upper normal limit, may not be sufficient to suppress the secretion of thyroid-stimulating hormone.

Case reports

Both patients were women who had had total thyroidectomies because of follicular or papillary cancer of the thyroid gland. Owing to supposed side effects their T4 replacement therapy was changed to T3 in daily doses of 60 µg (case 1) and 40 µg (case 2). This treatment produced clinical euthyroidism in both patients. The first patient was investigated with the thyrotropin-releasing hormone test (200 µg thyrotropin-releasing hormone intravenously) after 38 months of receiving T3 replacement therapy and the second after 19 months. The results are summarised in the table.

Determination of thyroid function in two patients receiving T3 replacement therapy after thyroidectomy

	Serum concentrations of thyroid-stimulating hormone (mU/l) after administration of thyrotropin-releasing hormone*			Serum concentrations* (nmol/l)	
	0	20 minutes	30 minutes	T4	T3
Case 1	28	100	100	1	2.8
Case 2	75	390	350	1	3.2
Normal range	1-7			5-154	0.9-3.2

*Samples drawn before daily administration of T3.

When the replacement therapy was changed back to T4 both patients had basal serum thyroid-stimulating hormone concentrations of 1 mU/l or less, which did not increase after administration of thyrotropin-releasing hormone. With T4 replacement therapy the serum T4 concentrations were 163 nmol/l (case 1) and 194 nmol/l (case 2) and the serum T3 concentrations 1.8 nmol/l (case 1) and 2.0 nmol/l (case 2). Thus, T4 replacement therapy suppressed the secretion of thyroid-stimulating hormone more effectively despite a lower serum T3 concentration.

Comment

Total thyroidectomy is a special situation in which the effect of T3 on the secretion of thyroid-stimulating hormone can be studied