study showed a significant reduction in lower oesophageal sphincter pressure after intramuscular administration of emepronium bromide. In the nine patients who completed the study no significant change in sphincter pressure was found after four weeks' treatment with a standard oral dose of emepronium bromide. This may have been due to the much lower serum concentrations seen after oral medication than after intramuscular injection, confirming the findings of Ritch *et al.*⁵

A local irritative effect of the tablets is probably the most common cause of oesophageal injury after oral medication, as no decrease in sphincter pressure was shown during oral treatment with emepronium bromide. Since, however, higher plasma concentrations of emepronium bromide decrease the sphincter pressure and the absorption of the drug from the gastrointestinal tract varies considerably, occasional high serum concentrations may be seen. This, combined with an individual variability in parasympathetic receptor sensitivity, might cause the lower oesophageal sphincter to relax, causing more severe cases of reflux oesophagitis in the lower oesophagus.

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Acute severe intravascular haemolysis: an unrecognised cause of pancreatitis

Pancreatitis or histologically proved pancreatic damage is common in chronic renal failure¹ and complicates 3-7% of renal transplant operations.² Hypercalcaemia as a result of hyperparathyroidism, hyperlipidaemia, and viral infections may be contributing factors in uraemic patients.³ Occasionally drugs—corticosteroids, cytotoxic agents, or thiazides—may be a factor.

We describe two patients receiving haemodialysis in whom acute pancreatitis developed in association with acute intravascular haemolysis owing to the use of hypotonic dialysate.

Case reports

CASE 1

A 42-year-old woman who had been receiving regular hospital haemodialysis for three months owing to end-stage renal failure secondary to reflux nephropathy complained of being unwell with central chest pain and diaphoresis 100 minutes into a dialysis session. Dark red blood was evident in the dialysis lines. She had no history of any viral illness or excessive ethanol ingestion. Her only medication was aluminium hydroxide gel 950 mg thrice daily. Physical examination was unremarkable except for moderate epigastric tenderness. Laboratory investigations showed packed cell volume 21%, serum sodium 109 mmol(mEq)/l, dialysate sodium 35 mmol/l, serum amylase activity 2000 U/l, and serum calcium 2.5 mmol/l (10 mg/100 ml). The plasma in a centrifuged specimen was pink. Dialysis was stopped immediately.

She was initially treated with 3% sodium chloride solution and dialysate containing 145 mmol sodium/l. She complained of increasing epigastric pain radiating into her back over the ensuing 24 hours. Physical examination

showed increasing epigastric tenderness with paralytic ileus. Dilated loops of small bowel were seen on an abdominal x-ray film; serum amylase activity had increased to 10 000 U/l. Clinical and biochemical variables gradually resolved over the next five days, treatment comprising nasogastric aspiration, intravenous fluids, and cimetidine. She remained well over the next six months and resumed hospital dialysis.

CASE 2

A 30-year-old man who had received home haemodialysis for four years was admitted because of failing vascular access. He complained of feeling hot, chest tightness, and dyspnoea 210 minutes into a dialysis session. Dark red blood was noted in the dialysis lines. A history of recent viral infection or ethanol ingestion was absent. His only medication was aluminium hydroxide tablets 375 mg thrice daily. Physical examination showed epigastric tenderness. Laboratory investigations showed serum sodium 115 mmol/l, serum chloride 83 mmol(mEq)/l, dialysate sodium 30 mmol/l, serum amylase 6600 U/l, and serum calcium 2.25 mmol/l (9 mg/100 ml). Centrifugation of a blood sample yielded pink plasma. Dialysis was immediately stopped. He was treated with 3% hypertonic sodium chloride solution, and dialysis was restarted with dialysate containing 145 mmol sodium/l.

He complained of increasing epigastric pain over the next 24 hours, developing paralytic ileus. Serum amylase activity was 17 600 U/l. His symptoms resolved over the next eight days with nasogastric aspiration, intravenous fluids, cimetidine, and analgesia. He remained well four months later, having resumed home haemodialysis. Barium-meal examination was normal.

Comment

Pancreatitis may be hard to diagnose in uraemic patients as serum amylase activity is commonly increased, usually less than threefold. Confirmation may be achieved by using the amylase to creatinine clearance ratio.⁴ The absence of a history of recent viral infection and ethanol ingestion, with normocalcaemia and satisfactory clearance on dialysis, exclude these as aetiological factors. The pathophysiology of haemolysis-induced pancreatitis is due to local release of intracellular lysosomal enzymes, especially cathepsin B, resulting in activation of trypsinogen and other zymogens with the sequelae of autodigestion and inflammation. Nevertheless, a further underlying risk factor may also be necessary, which in our cases was the uraemic state.

Although the intravascular haemolysis in our patients was due to human error in a recirculating single-pass positive-pressure system, its association with pancreatitis has not to our knowledge been reported before. We emphasise the importance of this complication, which may be prevented by strict attention to proper dialysis procedures and adequate treatment of water.⁵

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Comparative risks of rhesus autoimmunisation in two different methods of mid-trimester abortion

The introduction of anti-D immunoglobulin has greatly reduced the incidence of rhesus haemolytic disease, though cases still occur.¹ Autoimmunisation after spontaneous or induced abortion is an important factor and can probably be prevented: anti-D immunoglobulin is not always given to Rh-negative women.² Autoimmunisa-