0.060 (normal < 0.022), and malabsorption of radiocalcium. We concluded that the hypercalcaemia was resorptive although there was no radiological evidence of bone lesions. A saline diuresis was started using 7 l of 0.9% saline and 2 l of 5% dextrose over the next 24 hours. Despite maintenance of fluid balance and a urinary loss of 27.3 mmol (1092 mg) of calcium (normal < 9 mmol/day) plasma calcium and renal function remained unchanged (fig).

Peritoneal dialysis was then started using 2-l exchanges and a dwell time of four hours. The dialysis fluid, which was calcium-free, was composed of combinations of 0.9% saline, 5% dextrose, and small amounts of 50% dextrose, diluted if necessary with sterile water to achieve an appropriate sodium concentration. The patient also received 4 l of 4% dextrose, 0.18% saline intravenously daily with potassium supplementation. Within 17 hours the plasma calcium concentration had fallen to 4.23 mmol/l (16.92 mg/100 ml) and the patient became responsive and rational. Plasma creatinine also fell. Dialysis was continued for three days maintaining a total calcium output of more than 20 mmol/day (800 mg/day) despite a falling plasma level. Half of this was urinary loss and the remainder peritoneal. Plasma calcium fell to 2.90 mmol/l (11.60 mg/100 ml) and the patient was well enough to tolerate endoscopy. This revealed a squamous carcinoma of the oesophagus. In view of the hepatic metastases active management was withdrawn and the patient died 14 days later.

Comment

There are few reports of the use of calcium-free peritoneal dialysis in the management of severe hypercalcaemia.² In this patient it helped to reduce the plasma calcium and improve responsiveness to allow a definitive tissue diagnosis to be made. The associated renal failure and electrolyte imbalances were also easily corrected. Equilibration between the dialysate and the intravascular compartment was almost complete using four-hourly exchanges. Probably a more rapid reduction in plasma calcium could be obtained with a shorter dwell time allowing more frequent exchanges.

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Acute renal failure after a beer-drinking binge

It is not widely known that excessive ingestion of ethanol may precipitate acute renal failure in healthy individuals. Six patients who drank large amounts of vodka over several hours developed acute renal failure, occasionally with hepatocellular damage.¹ Oliguria persisted from 16 to 75 days. All patients required dialysis, and one patient died. We now report two similar cases of acute renal failure after heavy beer drinking with subsequent recovery after peritoneal dialysis.

Case 1

A 41-year-old motor fitter drank over two gallons (9 l) of beer that he had made himself from a kit. He drank this beer between 4 pm and midnight and complained of severe diffuse lower abdominal pains throughout the night. At 8 am he awoke with headache, neck pains, abdominal muscle pain, and general muscle stiffness. From this time his urine output fell. He was referred to hospital after three days. Examination showed a drowsy but otherwise fit man with no evidence of dehydration.

Blood pressure was 130/80 mm Hg. Urine volume was 300 ml in 24 hours, and urine osmolality 376 mmol (mosmol)/kg. Blood urea concentration was 22.5 mmol/l (135.5 mg/100 ml). Serum bilirubin concentration was 13 µmol/l (0.8 mg/100 ml), alkaline phosphatase activity 7 KA units, and aspartate transaminase activity 53 U/l. Cystoscopy showed no abnormalities, and ureteric catheterisation was unsuccessful. Plain radiography of the abdomen

showed two kidneys of normal size. On conservative treatment the patient remained oliguric and his blood urea concentration rose to 59.6 mmol/l (359.0 mg/100 ml). Peritoneal dialysis was started, and diuresis occurred on his sixth day in hospital. Six months later his glomerular filtration rate was 97 ml/min/1.7 m² and effective renal plasma flow rate 361 ml/min/1.7 m².

The patient had been a weekend beer drinker from the age of 16 and was accustomed to drinking eight pints (4.5 l) of beer a day at weekends. He had never been ill before or lost time from work.

Case 2

A 28-year-old carpenter drank over 30 pints (17 l) of beer at a pop-music festival and developed diffuse upper abdominal pain and dull constant back pain throughout the night. His urine output fell. These symptoms persisted for three days, after which he was referred to hospital. On examination the patient was drowsy and jaundiced but with a strong physique. There was no sign of dehydration.

Blood pressure was 120/89 mm Hg. Bladder catheterisation produced 50 ml of dark brown urine. Urine volume was 400 ml in 24 hours, and urine osmolality 311 mmol (mosmol)/kg. Serum urea concentration was 22.9 mmol/l (138.0 mg/100 ml), serum bilirubin concentration 133 µmol/l (7.8 mg/100 ml), aspartate transaminase activity 100 U/l, and alkaline phosphatase activity 18.6 KA units. After five days serum urea concentration rose to 65 mmol/l (391.6 mg/100 ml), but serum bilirubin concentration fell to 40 µmol/l (2.3 mg/100 ml), aspartate transaminase activity to 146 U/l, and alkaline phosphatase activity to 11.7 KA units. Oliguria persisted for 10 days and peritoneal dialysis was started on his sixth day in hospital. A renogram taken during the oliguric phase showed severe tubular dysfunction. Obstruction of the lower urinary tract was excluded by ureteric catheterisation. In the oliguric phase glomerular filtration rate was 4 ml/min/1.7 m² and effective renal plasma flow 23 ml/min/1.7 m²; three weeks later, after diuresis, these had risen respectively to 76 ml/min/1.7 m² and to 366 ml/min/1.7 m². The results of renal biopsy performed in the diuretic phase showed normal histological appearances (Dr O G Williams). The patient made an uneventful recovery. He drank up to 10 pints (5.7 l) of beer a day at weekends and smoked 30 cigarettes daily.

Comment

The sequence of events in our patients was similar to that described by Szepietowski *et al.*¹ Healthy individuals, after ingesting large quantities of ethanol over several hours, complained of severe abdominal pains with muscle stiffness, followed by suppression of urine. Hepatocellular damage occurred in some of these patients. There was some delay in admitting our patients to hospital since they had never been ill before and their symptoms were thought to be due to severe hangover. Admission to a surgical ward was eventually arranged because oliguria, combined with lower abdominal pain, suggested obstruction of the urinary tract.

Szepietowski *et al.* suggested that acute renal failure may result from nephrotoxic damage by ethanol.¹ This is unlikely because the results of renal biopsy, performed on one of our patients, showed normal histological appearances. Acute renal failure has been observed after a bout of heavy drinking in chronic alcoholics in association with myoglobinuria and accompanying muscle tenderness and swelling.^{2,3} Furthermore, myoglobinuric acute renal failure has been caused by alcohol in six out of 20 patients.⁴ The mechanisms by which rhabdomyolysis and myoglobinuria produce acute renal failure are not known.

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