hyperkalaemia (serum potassium, 7.5–9.1 mmol/l) [6]. The mechanism behind this apparent thalidomide-induced hyperkalaemia remains unclear. Therefore, the authors recommend caution when thalidomide is used in patients with moderate to severe renal failure (serum creatinine >300 µmol/l) or in patients on dialysis. To go a step further, we think that thalidomide should in all cases be started at a dosage that should not exceed 100 mg/day. It is also important to keep in mind that, even at this low dosage, thalidomide side effects may appear and that they may be severe.

Furthermore, thalidomide has been used to treat uraemic pruritus at a dosage of 100 mg/day in a study enrolling 210 patients with 29 cases of refractory uraemic pruritus and 11 were noted, with the mean reduction in patients' pruritus scoring. In this study by Silva et al., no side effect or biochemical abnormality has been reported [7].

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Acute pancreatitis during haemodialysis

Sir,

Acute pancreatitis tends to develop more frequently in patients with end-stage renal disease (ESRD) than in the general population, even in those managed by haemodialysis [1]. A case of acute pancreatitis in a patient on haemodialysis is presented herein to remind the nephrology community of this critical association.

This 74-year-old woman had been on haemodialysis treatment using cellulose dialyzers and heparin anticoagulation for the last 6 years due to ESRD as a consequence of diabetes mellitus and hypertension. She had no history of alcohol use. Medications included enalapril. While undergoing haemodialysis at a local hospital, blood pressure fell to 64/32 mmHg shortly after commencing the session and she complained of severe epigastralgia. Since serum amylase level was found to be significantly increased (1413 U/dl), she was transferred to our hospital. Laboratory findings on admission were as follows: lipase, 675 U/l; calcium, 8.4 mg/dl; and triglycerides, 89 mg/dl. Pancreatic isoamylase accounted for 96% of total serum amylase. Imaging studies revealed pancreatic enlargement with inflammatory exudates, and excluded the presence of biliary stones. The patient was managed conservatively under a diagnosis of severe acute pancreatitis. Her condition gradually improved and amylase levels on hospital day 36 were within normal range.

In the present case the angiotensin converting enzyme (ACE) inhibitor enalapril might have been one factor contributing to acute pancreatitis. ACE inhibitors are known to increase the generation of bradykinin, a vasoactive substance that induces angioedema, which in turn could cause pancreatic duct obstruction followed by enzyme leakage [2]. Bradykinin and subsequent nitric oxide release also could have been at least partially associated with hypotension during haemodialysis [3]. Hypotension together with underlying arteriosclerosis seen in many haemodialysis patients can induce mesenteric ischaemia represented by abdominal pain during the dialysis session, which has to be considered as another important risk factor for acute pancreatitis [1]. This is because reactive oxygen species such as superoxide released in the ischaemia/reperfusion process [4] can result in pancreatic damage [5].

In general, ESRD patients display multiple risk factors for acute pancreatitis, such as various medications or induced hyperkalaemia responsible for the activation of pancreatic enzymes [1]. Acute pancreatitis in ESRD patients must thus be considered as a multifactorial disease. Finally, whatever the precise pathophysiological mechanisms might have been in the present case, ischaemic events during the haemodialysis session appear to have triggered the process leading to acute pancreatitis in the light of the temporal course. Based on the present case, we would like to emphasize the need to avoid hypotension during the haemodialysis procedure. Removing as many potential risk factors as possible is also important, since acute pancreatitis tends to become life threatening in patients with ESRD [1].

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[None declared]
Sir,

Extreme hypernatraemia is rare in adults. Due to the fact that rapid correction of hypernatraemia may result in neurological complications, a gradual reduction of sodium concentration is generally advised. However, it is difficult in patients with extreme hypernatraemia, severe metabolic acidosis and renal failure. We describe a patient with extreme hypernatraemia (serum Na$^+$ 202 mEq/l), severe metabolic acidosis (HCO$_3^-$ 7.6 mEq/l) and renal failure who was successfully treated with continuous venovenous haemofiltration (CVVH) with a portion of commercial CVVH replacement fluid and additional sodium bicarbonate. We believe this is the first case report of such a novel treatment and may apply to other patients who suffer from extreme hypernatraemia, severe metabolic acidosis and renal failure.

A 69-year-old woman presented at our emergency room with progressive deterioration of consciousness for 4 days. Her past history included hypertension, chronic renal failure with serum creatinine of 3.7 mg/dl 1 month before admission, ischaemic stroke with left hemiplegia, and status post-craniotomy after stroke. Her consciousness was clear after ischaemic stroke with left hemiplegia, and status post-craniotomy after stroke. Her consciousness was clear after ischaemic stroke with left hemiplegia, and status post-craniotomy after stroke. Her consciousness was clear after ischaemic stroke with left hemiplegia, and status post-craniotomy after stroke. Her consciousness was clear after ischaemic stroke with left hemiplegia, and status post-craniotomy after stroke.

On arrival at the emergency department, her conscious status was E2M4V2. Vital signs revealed a blood pressure of 137/71 mmHg, heart rate of 62 b.p.m. and respiratory rate of 18 breaths/min. The initial laboratory data showed blood urea nitrogen (BUN) 114.7 mg/dl; creatinine 8.69 mg/dl; serum sodium 202.4 mEq/l; glucose 91 mg/dl; potassium 4.6 mEq/l; and blood osmolality 455 mosml/kg. Under the impression of acute exacerbation of chronic renal failure, extreme hypernatraemia, 5% dextrose water was given at a rate of 120 ml/h. The serum sodium level decreased from 202.4 to 188.5 mEq/l (13.9 mEq/l) in the first 24 h. However, the serum sodium concentration in the next 24 h decreased only 2.5 mEq/l because of infusion of high volume sodium bicarbonate for correction of metabolic acidosis. The patient became more and more dyspnoeic, and the arterial blood gas at 60 h after admission revealed pH 7.34, HCO$_3^-$ 9.1 mEq/l, PaO$_2$ 241 mmHg (O$_2$ mask 101/min) and PaCO$_2$ 17 mmHg. BUN was 134.7 mg/dl. Creatinine was 7.96 mg/dl. Chest X-ray revealed cardiomegaly and pulmonary oedema. She was therefore intubated and dialysis was arranged for further fluid and electrolyte management. The serum sodium concentration increased to 190.5 mEq/l after infusion of 7% NaHCO$_3$ for metabolic acidosis.

For the purpose of gradual correction of the sodium concentration, CVVH was the chosen mode of dialysis for this patient. We used an AK 10 blood pump (Gambro, Lund, Sweden) and a Hemofilter 6S membrane (Gambro, Hechingen, Germany) through a 12,12-Fr dual lumen catheter (Arrow, Erding, Germany), which was placed in a femoral vein. The blood flow rate was 200 ml/min during the dialysis procedure. Commercial CVVH replacement fluid solution A and CVVH replacement fluid solution B (Taiwan Biotech Co., Ltd., Taoyung, Taiwan) were mixed with a flow rate of 800 ml/h. The sodium concentration of the mixture is 142.3 mEq/l. The net ultrafiltration rate was adjusted according to her volume status. The serum sodium level dropped unexpectedly from 190.5 to 174.8 mEq/l in 6 h after starting CVVH. As her sodium reduction rate was above the target of 1–2 mEq/l/h, the sodium concentration of the replacement fluid was then adjusted by adding 7% NaHCO$_3$. We added 1.45 ml of 7% NaHCO$_3$ to 1 liter of commercial replacement fluid mixture for each 1 mEq/l elevation of sodium concentration. By this method, the replacement fluid was adjusted every 6 h with a 3 mEq/l targeted reduction of sodium concentration. As the serum sodium concentration was 174.8 mEq/l, the replacement fluid sodium concentration was set at 172 mEq/l. The serum sodium concentration then decreased gradually to 151.8 mEq/l by 54 h after commencement of CVVH. CVVH was changed to intermittent haemodialysis afterwards. The patient’s consciousness improved to E4M5-6Vt on the 6th day after admission. She was extubated on the 8th hospitalization day and was transferred to a general ward for further care.

Hypernatraemia is a common problem associated with high mortality and morbidity [1]. Managing extreme hypernatraemia is challenging. Though well-controlled studies to ascertain the optimal treatment of chronic hypernatraemia do not exist, gradual correction of hypernatraemia with a rate of no more than 1–2 mEq/l/h was suggested [1,2]. Free water hydration is the most commonly used method for hypernatraemia. However, fluid overload might develop in patients with heart failure and renal failure, as occurred in our patient. Since conventional haemodialysis and peritoneal dialysis cannot fulfill the requirement for gradual reduction of serum sodium concentration, we chose continuous renal replacement therapy. Moss et al. reported the first case of hypernatraemia (serum Na$^+$ 189 mEq/l) corrected by continuous arteriovenous haemodiafiltration in 1990 [3]. The patient died of diffuse alveolar damage and cardiogenic shock. Jen-Jar Lin et al. used continuous venovenous haemodialysis to correct hypernatraemia (serum Na$^+$ 180 mEq/l), hyperglycaemia and other electrolyte imbalance in a 12-year-old female patient [4]. They used custom-made dialysate with adjustment of dialysate electrolyte every 6 h and controlled the serum sodium level in 4 days. The patient survived without neurological sequelae after the critical episode. Custom-made dialysate and replacement fluid were not available in our hospital. So we chose CVVH, which needs only replacement fluid but no dialysate. The adjustment of sodium concentration was by adding 3% NaCl to the replacement fluid. The concentration of other electrolytes did not change much with 7% NaHCO$_3$. For example, the ratio of [Na$^+$]/[Ca$^{2+}$] from 142.3 to 190 mEq/l requires 69.2 ml of 7% NaHCO$_3$ in 1 liter of fluid. The reduction of [K$^+$], [Ca$^{2+}$], [Mg$^{2+}$] and [Cl$^-$] is only 6.4%, which is insignificant clinically.