Hyperamylasaemia and bronchoaspiration associated with Lanthanum carbonate

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Dear Editor,

We report the case of a female patient who suffered from hyperamylasaemia and rectal bleeding, and died unexpectedly owing to a possible bronchoaspiration of lanthanum.

The case involves a 54-year-old female patient with a medical history of hypertension and chronic kidney disease, who had not followed a peritoneal dialysis programme for 8 months, and who was admitted to our department for diarrhoea with hypotension and an episode of rectal bleeding. The abdominal physical examination was irrelevant. The detailed findings showed hyperamylasaemia at around 900U/l (42-220U/l), with leukocytosis levels at 22.1 X 10^9l. The other laboratory parameters were at a normal level. The peritoneal fluid was analysed, ruling out peritonitis. An X-ray of the abdomen (figure 1) showed diffuse opacifications in the colon, which corresponded to the lanthanum carbonate tablets introduced 2 months prior to hospitalisation as a treatment for hyperphosphatemia (0.75g oral 3/day). Two colonoscopies were carried out without success, showing a poor level of preparation and blocking the progression of the colonoscope. The presence of a fecaloma impacting on the rectosigmoidal junction was detected in an abdominal computed tomography. There were no signs of pathological masses in the pancreas or the rest of the abdomen. The fecaloma could be extracted; however, on the same day the patient showed signs of sudden dyspnoea and a deterioration of her general state, with hypotension and oxygen desaturation. The thorax X-ray (figure 2) showed radioopaque stripes along the pulmonary alveoli. The patient's family was further questioned, confirming the administration of the lanthanum tablets that had been suspended at admission. The patient underwent haemodialysis without any improvement in her pulmonary condition and died.

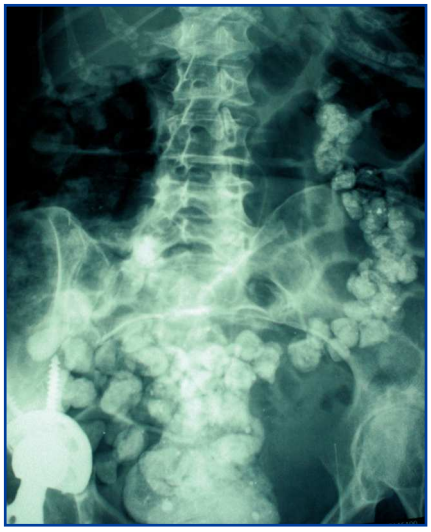

Figure 1. Abdomen. Radioopaque images in the colon, corresponding to lanthanum carbonate.

Lanthanum is a metal prescribed as a captor of aluminium- and calcium-free phosphorus that is visible in the intestine.[1-4] Its most frequent side effects include nausea, diarrhoea, hypotension, abdominal pain and constipation;[5] however, no symptoms of pancreatitis have been reported in the literature. Our patient probably aspirated lanthanum bronchially, causing her death. Patients treated with lanthanum should be closely monitored owing to the gastrointestinal side effects and their complications.

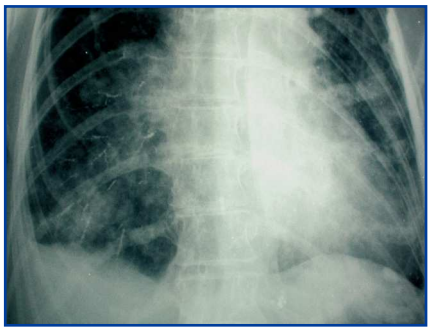

Figure 2. Thorax. Radioopaque stripes along the pulmonary alveoli.

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Progressive renal failure and nephrotic syndrome in a patient being treated with Lithium

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Dear Editor,

We report the case of a 48-year-old female patient, treated with lithium for bipolar disorder for more than 20 years, without other special health issues. In December 2008, she was examined for a deteriorating renal function (Cr 2.7mg/dl and FG according to MDRD of 20ml/min/1.73m),[2] her renal function of the previous year being Cr 1.4mg/dl. At the physical examination her blood pressure without any medical treatment was 170/95mmHg. The test carried out revealed Cr of 3.1mg/dl, Ur of 109mg/dl, K of 5.5mEq/l, P 4.7mg/dl, lithaemia of 0.9mEq/l and haemoglobin of 12.8g/dl;

the rest of the biochemical parameters being normal. Her urine showed proteinuria of 5.7g/24 h, with 10-12 red blood cells/field in the sediment. The immunological test showed: negative ANA and ANCA, normal immunoglobulin levels and a normal complement system. The ultrasound revealed normal-sized hyperecogenic kidneys. Due to these findings, it was decided to carry out a renal biopsy which showed the following: 5 glomeruli, one with segmental hyalinosis and another with complete sclerosis. Mesangial thickening and diffuse interstitial fibrosis with tubular atrophy and dilation present in those not suffering from sclerosis. Mesangial deposits of IgA (+++) and C3 (+++). Anatomopathological diagnosis of IgA nephropathy with chronic tubulointerstitial nephritis. The patient began treatment with calcium antagonists with an improvement in the pressure figures; however, renal function continued to worsen, which is why it was necessary to prepare for the substitutive treatment.

Lithium salts are widely used in bipolar disorder. The treatment of the chronic form of the disorder with these salts is associated with different types of renal damage, including nephrogenic diabetes insipidus, metabolic acidosis, chronic nephritis and hypercalcaemia. The most important predisposing factor is the time of exposure to lithium, while other factors include age, episodes of lithium poisoning and comorbidity. The anatomopathological substrate of the toxicity from lithium is interstitial fibrosis, which can appear 5 years following treatment. At the same time, focal segmental glomerulosclerosis is associated with tubulointerstitial changes. Renal tubular cysts are a sign of tubular damage, which is manifested as dilation of the distal segment and the tubular collector. The progression of nephritis brought about by lithium is slow, considering a drop in the glomerular filtration rate of 2.2ml/min per year of exposure, with a very low rate of terminal chronic kidney disease in the various published studies. Interruption of the treatment with lithium salts does not achieve the recovery of the patient's baseline renal function. Instead, in some cases, the deterioration continues at a similar rate following its interruption. It is thought that a point of no return exists, after which renal fibrosis continues despite discontinuing the aggression that triggered it in the first place. This depends on the anatomopathological substrate at that moment, with better response of the illness to small changes. In this instance, the renal biopsy allowed us to position the clinical condition, and it was decided to continue with the lithium treatment. This could not fully justify the patient's progress in full view of the biopsy result that was compatible with IgA nephropathy, which justified the clinical condition described.

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Previous ischaemic optic neuropathy in haemodialysis

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Dear Editor,

Optic neuropathy is a syndrome and not a mere common injury of the optic nerve; its aetiology is broad and greatly varies. Ischaemic optic neuropathies influenced by local and systemic factors are particularly noteworthy, many of which are difficult to understand. These processes occur in the field of other specialisations with surgical manoeuvres and/or aggressive diagnoses, in which the patient is compromised, as the initial diagnosis could be problematic.[1] Ophthalmologists are well aware of the process, however, doctors of other specialisations do not realise the possibility for this clinical condition, being familiar with its existence only when it appears. Various risk factors exist for ischaemic optic neuropathy (ION), however, we must emphasise sudden hypotension, which does not allow for the autoregulation mechanisms of the optic nerve to compensate, particularly if the patient suffers from previous hypotension, anaemia, sudden and/or recurrent haemorrhage, serious facial oedema, chronic kidney disease, bleeding surgeries and, generally, all situations associated with arteriosclerosis.

Based on the few reports in the literature of patients with chronic kidney disease (CKD), we report the cases of 2 patients in haemodialysis with a diagnosis of acute bilateral loss of vision due to a previous ischaemic optic neuropathy (PION).

Case 1

A 29-year-old woman with a diagnosis of CKD owing to GEFS was hospitalised for haemodialysis at the age of 25. Three years after the haemodialysis, she showed signs of severe secondary hyperparathyroidism (SHPT) with PTH i > 1,000pg/ml, ostealgia, asthenia, pruritus and persistent symptomatic hypotension. There were no arterial or cardiovascular calcifications. A diagnostic ultrasound of the parathyroid glands was carried out with no visualisation with ^{99}Tc and MIBI with diffuse increase in the fixation of the left and right inferior parathyroid lobes. Treatment for the SHPT was carried out with calcium binders and intermittent calcitriol IV for hyperphosphatemia. Subtotal parathyroidectomy was prescribed, taking into consideration the patient's age and transplant suitability. The previous laboratory