the infection had poor evolution of the dialysis technique, primarily due to adherences or problems with ultrafiltration, similar to the results from other studies.[4]

Although imaging tests prior to the second catheter insertion have low sensitivity, we believe that they are necessary, since an abdominal pathology secondary to the first case of peritonitis may be present, with no clinical symptoms, as occurred in our case of a patient with an abdominal abscess.

In the case of early dysfunction of the peritoneal catheter, a peritoneography is necessary for evaluating the presence of compartmentalisation (Figure 1).

In conclusion, a return to PD following CR due to peritonitis should be evaluated on an individual basis, paying special attention to those patients that had peritonitis refractory to treatment, with associated abdominal pathologies, and a high D/P creatinine level before the removal of the first catheter. The impact that the possible loss of residual diuresis would have on the evolution of the patient should also be taken into account.

Conflicts of interest

The authors have no potential conflicts of interest to declare.

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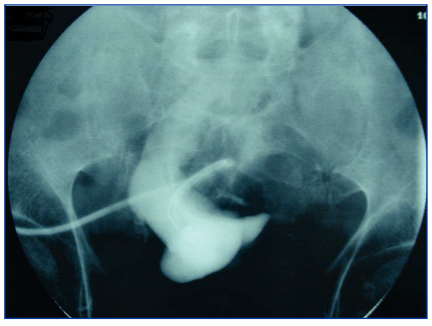

Figure 1. Image of a pseudocavity in the peritoneography.

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Salicylate poisoning management

Nefrologia 2011;31(6):751-2
doi:10.3265/Nefrologia.pre2011.Jul.11042

To the Editor,

Acute salicylate intoxication is one of the most common causes of intoxication from antipyretics. In fact, in recent years, the incidence of this condition has decreased due to a greater use of other drugs, such as paracetamol and non-steroid anti-inflammatory drugs.

Here we present the case report of a 60-year old woman with a background of depression that sought emergency treatment for mild cognitive impairment and consumption of multiple acetylsalicylic acid tablets. A physical examination revealed sustained arterial hypotension with a systolic blood pressure (SBP) of 80-90mm Hg and diastolic blood pressure (DBP) of 50-60mm Hg. Laboratory tests revealed urea: 81mg/dl, serum creatinine: 1.84mg/dl, pH: 7.39, HCO_3^-: 13.9mmol/l, and lactate: 1mmol/l. Serum salicylate levels were positive with concentrations of 65.68mg/dl. We performed a gastric lavage and started abundant hydration treatment and urine alkalization, as well as admitting the patient into the intensive care unit (ICU), where her low blood pressure values and oliguria continued, and her level of cognitive impairment increased.

Given the poor clinical evolution, with increased nitrogen retention values and altered haemodynamics, we decided to provide conventional haemodialysis for four hours, with positive balances (+2500ml) and high-flux polysulfone. The acid-base alterations were corrected following treatment, and drug concentrations decreased to 31.99mg/dl (51% reduction), with improved cognitive state and normalised blood pressure. The patient was discharged with no organ damage.

Therapeutic levels of salicylic acid range between 10mg/dl and 30mg/dl, and higher levels can produce moderate-severe intoxications, causing neurological deficits, coma, convulsions, pulmonary oedema, sustained hypotension, acute renal failure, and severe electrolyte imbalances,[1] although patient death is rare.[2]

Done normograms, which are widely used in several different types of intoxications, should not be used in acute salicylate intoxications because of the poor correlation between serum concentrations and the clinical and/or laboratory alterations produced. Any patient with high salicylate levels should be started on general support measures. A gastric lavage should also be applied in order to reduce the absorption of the toxin and the urine should be alkalised for increased excretion, at the same time as correcting the hydration state and controlling the hydroelectrolytic imbalances. The indications for starting haemodialysis for removing the salicylic acid vary according to author, but

the majority coincide that at concentrations greater than 100mg/dl, this treatment is warranted, although others reduce this value to 80mg/dl. In any case, clinical and laboratory alterations will indicate the need for haemodialysis in the majority of cases. In this manner, patients with haemodynamic alterations, acute renal failure, severe neurological alterations, and/or severe metabolic acidosis that do not respond to conservative treatment should be started on extra-corporeal depuration treatment.

There is currently no consensus regarding the type of dialysis that should be administered. Warthall, et al[3] described reduced salicylate concentrations by 77% to 84% using continuous veno-venous haemodiafiltration for a mean 11 hours, whereas Lund, et al[4] described similar results using conventional haemodialysis followed by continuous dialysis for 12 hours. In our case, we achieved a 51% reduction using conventional haemodialysis for four hours, which demonstrates the usefulness of this technique in the acute phase. We believe that more studies would be appropriate on this subject, although the results currently available seem to indicate starting treatment with conventional haemodialysis in severe cases or patients with important clinical/laboratory repercussions, since we can achieve a significant reduction in toxin levels within a short period of time, and afterwards the patient can be evaluated for continued depuration treatment with continuous techniques, according to the serum concentrations of salicylates and the previously mentioned alterations.

Conflicts of interest

The authors have no conflicts of interest to declare.

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B) BRIEF CASE REPORTS

Chronic hypercalcaemia in haemodialysis patients

Nefrologia 2011;31(6):752-4
doi:10.3265/Nefrologia.pre2011.Jun.10974

To the Editor,

The increase of adynamic bone disease in haemodialysis (HD) patients can hinder the diagnosis of other diseases that progress with latent hypercalcaemia. Here we present a n elusive relevant case.

A male patient of 73 years with terminal renal failure (TRF) secondary to diabetic nephropathy that started HD in June 2008. The patient smoked 10 cigarettes/day, and did not consume alcohol. The patient had long-term arterial hypertension (AHT), type 2 diabetes mellitus for 45 years on treatment with insulin, diabetic retinopathy, dyslipidaemia, chronic ischaemic heart disease tending towards acute myocardial infarction (AMI), stage III b-IV chronic ischaemia of the lower limbs requiring femoropopliteal bypass in December 2009, 80% stenosis of the left carotid artery, widespread vascular calcifications, chronic bronchopathy, obstructive sleep-apnoea syndrome, small (lacunar infarcts of the thalamus and hemiprotuberance) and large (previous stroke in the left hemisphere) vessel ischaemic brain disease, and polyarthrosis with severe degeneration of the lumbar spinal column.

Three years before starting HD, and with treatment including thiazide and oral calcitriol, the patient was hospitalised for severe hypercalcaemia (13.5mg/dl), with neurological and digestive symptoms. In the subsequent analysis, we observed hilar lymph nodes of a non-pathological size and isolated reticular pulmonary parenchymal opacities, homogeneous hepatosplenomegaly, angiotensin-converting enzyme (ACE) on the upper limit of its normal range (three measurements taken between 30U/l and 60U/l, under normal conditions, normal range: 8-55). All other tests were negative, and the patient was diagnosed with exogenous intoxication by vitamin D and thiazide, and was treated with parenteral pamidronate, with an excellent clinical evolution and no levels of 1,25 $(OH)_2$ vitamin D. Since then, the patient has been asymptomatic, with suppressed intact parathyroid hormone (iPTH) levels and a spontaneous tendency towards hypercalcaemia, for which he was diagnosed with adynamic bone disease (ABD). During the month of July 2009, the patient suffered a progressive condition of asthenia, anorexia, night sweating, disorientation, irritability, amnesic altera-