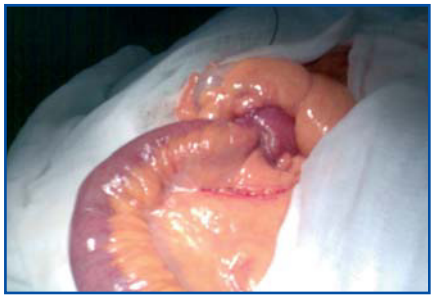

Figure 1.

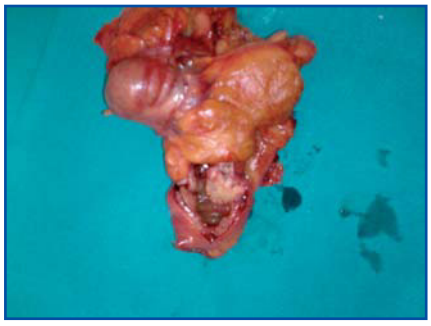

Figure 2.

infiltrating the adjacent muscle on anatomo-pathological examination; the tumour was serotonin-producing.

The most frequent location of the intestinal carcinoid tumours (ICT) is the caecal appendix (50%), followed by the ileum (25%), as in our case. The symptoms usually appear late and in a non-specific manner leading to a late diagnosis. 60% of the patients present hepatic metastases at the time of diagnosis. The carcinoid syndrome is found in only 5% of the patients and is related to the presence of hepatic metastases.[2]

In the diagnosis,[3] aside from the conventional imaging tests, the Octroscan is useful as it provides information on the localisation of tumours larger than 0.5cm and the expression or not of serotonin receptors. The differential diagnosis includes other intestinal tumours, ileo-caecal Crohn's disease and systemic mastocytosis.

Treatment should include surgical removal provided there are no metastases. Epsilon-aminocaproic Acid or somatostatin should be administered during the intervention to avoid carcinoid crisis. When there is metastatic dissemination, the patient should be managed conservatively. To reduce the metastases, 5-Fluorouracil or Adriamycin have been combined with Streptozotocin. Different drugs have been used for the treatment of the carcinoid syndrome with varied results including Chlorophenylalanine, serotonin antagonists, somatostatin, octeotride and interferon.

Although the prognosis for metastatic carcinoids is poor, survival is greater in patients with similar degrees of tumoural dissemination from other solid tumours.

1. Esteve V. Síndrome coronario, rectorragias y tumoración intestinal: a propósito de un caso. Nefrología 2006;26(4):507.
2. Gümüştaş OG, Gümüştaş A, Yalçin R, Savci G, Soylu RA. Unusual causes of small bowel obstruction and contemporary diagnostic algorithm. J Med Imaging Radiat Oncol 2008;52(3):208-15. Review.
3. Bornschein J, Kidd M, Malfertheiner P, Modlin IM. Gastrointestinal neuroendocrine tumors. Dtsch Med Wochenschr 2008;133(28-29):1505-10. Review.

A.I. Gómez Sotelo, B. Pérez Cabrera, C. González Puga, A. Palomeque
San Cecilio University Hospital. Granada.
Correspondence: Ana Isabel Gómez Sotelo
Hospital Universitario San Cecilio. Granada.
osiris986@hotmail.com

Haemodialysis management for salicylate intoxication

Nefrología 2009;29**(2)**:182-183.

Dear Editor,

Accidental overdose or suicide by salicylates provokes metabolic alterations and organ failure that can be fatal. [1,2] Haemodialysis must be used appropriately in these cases.[3,4] We present a case where haemodialysis was used in a potentially lethal overdose of Acetylsalicylic Acid (ASA).

We describe the case of a 24 year old female patient, with no morbid history, that presented in the Emergency Room 18 hours after having ingested 50 pills of ASA of 500mg, 25g total or 400mg per kilogram of weight, with suicidal intention. When hospitalized, she presented lethargy, confusion, vomiting, hypoacusia and tinnitus. Vital signs were normal and there were no positive findings in the physical examination. Laboratory tests on admission included: arterial gasometry: pH: 7.42, PO_2 115mmHg, PCO_2 14mmHg, HCO_3 9.2mmol/l. Creatininaemia: 0.89mg/dl, kalaemia: 2.1mEq/l, natraemia: 138mEq/l. The anion gap was 18. There were no alterations in liver function tests or in cardiac enzymes. A gastric lavage was performed, saline solution and sodium bicarbonate were administered. She was transferred to the Intermediate Care Unit with a salycilate level of 682mg/l.

Haemodialysis was carried out for four hours, with a polysulphone filter, and 39mEq/l bicarbonate, 3.5mEq/l of potassium and 140mEq/l of sodium bath. Fluid balance during haemodialysis was positive at 2,500cc. The patient was stable with progressive improvement of state of consciousness and slow correction of acid-base alterations. Salycilate levels of 99 and 1mg/l were obtained 12 to 20 hours after dialysis, respectively.

The ASA is absorbed in the stomach and small intestine as salicylic acid. It is conjugated in the liver and excreted in bile and urine. Therapeutic levels are of 100 to 300mg/l. The classic triad of intoxication by salicylate is hyperventilation, gastric irritation and tinnitus.[1] There may be liver, kidney, central nervous system and cardiovascular organ damage. Salicylate, above therapeutic levels, stimulates the respiratory centre and causes respiratory alkalosis.[5]

The toxic levels also produce a separation in the oxidative phosphorylation and accumulation of lactic acid, resulting in metabolic acidosis.[6] This mixed acid-base syndrome, with an increased anion gap, should lead one to suspect an intoxication by ASA in a patient where the antecedents of what has been ingested is unknown. The treatment of salycilate poisoning includes vital support, gastric lavage, the use of activated charcoal and alcalinization of the urine to encourage the excretion of the drug.[3] Salicylate has a distribution volume

of 150ml/k, a protein binding rate between 50 and 80% (which decreases when levels are high), and a molecular weight of 138.[3] These properties favour its elimination by haemodialysis, which is the indicated treatment in serious cases.

1. O'Malley G. Emergency department management of the salicylate-poisoned patient. Emerg Med Clin N Am 2007;25:333-46.
2. Chyka PA, Erdman AR, Christianson G, Wax PM, Booze LL, Manoguerra AS, et al. Salicylate poisoning: an evidence-based consensus guideline for out-of-hospital management. Clin Toxicol (Phila) 2007;45(2):95-131.
3. Wrathall G, Sinclair R, Moore A, Pogson D. Three case reports of the use of haemodiafiltration in the treatment of salicylate overdose. Hum Exp Toxicol 2001;20;491.
4. Locket S. Haemodialysis in the treatment of acute poisoning. Proc Roy Soc Med 1970;63:427-30.
5. Wood DM, Dargan PI, Jones AL. Measuring plasma salicylate concentrations in all patients with drug overdose or altered consciousness: is it necessary? Emerg Med J 2005;22:401-3.
6. Krause DS, Wolf BA, Shaw LM. Acute aspirin overdose: mechanisms of toxicity. Ther Drug Monit 1992;14(6):441-51.

N. Quintero Parra, A. Wurgaft Kirberg, Y. Orellana Araya, J. Arellano Lorca, L. Rojas Wettig, J. Pefaur Penna
Nephrology Department, Healthcare Complex Barros Luco, Santiago de Chile. Medical Sciences Faculty, University of Santiago of Chile.
Correspondence:
Yessenia Valeska Orellana Araya
Departamento de Nefrología. Complejo Asistencial Barros Luco. Santiago de Chile. Facultad de Ciencias Médicas. Universidad de Santiago de Chile.
yorellana20@yahoo.com

Candida-induced recurrent peritonitis after peritoneal catheter reinsertion

Nefrología 2009;29**(2)**:183.

Dear Editor,

Peritonitis caused by *Candida* is a rare but serious complication in patients on peritoneal dialysis, requiring the removal of the catheter in the majority of cases.

We present a case of relapsing peritonitis caused by *Candida parapsilopsis* after a prolonged period of peritoneal rest with adequate antibiotic coverage.

The patient is a 75 year old male with a past medical history of atrial fibrillation and chronic kidney failure (unknown aetiology) that began Kidney Replacement Therapy in November of 2003 with automated peritoneal dialysis.

This treatment was complicated by two episodes of peritonitis: the first episode, in December of 2005 caused by *Staphylococcus epidermidis*, and the second episode in July 2007 by *Klebsiella pneumoniae*, both resolved with antibiotic treatment given according to protocol. Antifungal prophylaxis was carried out in both cases with oral fluconazole.

In December 2007 he sought healthcare for abdominal discomfort, presenting cloudy peritoneal fluid, for which antibiotic treatment was prescribed. A yeast was identified in the peritoneal fluid 48 hours later, and antibiotic treatment with fluconazole and fluorocytosine was initiated. The peritoneal catheter was removed 24 hours after the diagnosis without complications. The patient replacement therapy was changed to haemodialysis, while continuing antibiotic treatment with fluconazole during three weeks.

After eight weeks of treatment and at the patient's request, a decision was made to re-insert a new peritoneal dialysis catheter, after performing an abdominal Computerised Axial Tomography (CAT), where no abnormalities were found except for a great amount of atheromatosis. The implantation of the catheter was performed by a surgeon that ruled out the existence of scar tissue.

15 days after the implantation, peritoneal dialysis treatment was started uneventfully. However after 10 days the patient presented with abdominal pain and cloudy fluid. Microbiology confirmed the new existence of *Candida parapsilopsis* in the peritoneal fluid, for which the catheter was removed and the patient was transferred permanently to Haemodialysis.

Fungal peritonitis in peritoneal dialysis is associated with a high percentage of failure.[1] The majority of the episodes are caused by *Candida species*, the optimal treatment remains unclear, but it requires the abandonment of the PD technique in the majority of cases.[2] Antifungal agents are recommended during a period of no less that 10 days after the removal of the catheter. The optimal time for the reinsertion of a new catheter after a fungal peritonitis is not clearly established. A period of at least 2-3 weeks is recommended.[3] These measures were not sufficient in our case. It may be that a longer period of antifungal treatment or a previous preventive treatment would be indicated in these cases prior to the insertion of a new catheter.

1. Prasad N, Gupta A. Fungal peritonitis in peritoneal dialysis patients. Perit Dial Int 2005;25(3):207-22.
2. Kleinpeter MA. Successful treatment of Candida Infections in peritoneal Dialysis patients: case reports and review of the literature. Adv Perit Dial 2004;20:58-61.
3. Beth Piraino, George R. Bailie, Judith Bernardini, et al. Peritoneal Dialysis-Related Infections Recommendations: Update. Peritoneal Dialysis International 2005;25:107-31.

C. Pérez Melón, O. Conde Rivera, E. Novoa Fernández, M. Borrajo Prol
Nephrology. Hospital Complex of Ourense. Ourense.
Correspondence: Cristina Pérez Melón
Nefrología. Complexo Hospitalario de Ourense. Ourense.
cristicpm@hotmail.com