- Herrero-Morín JD, Fernández N, Santos F, Rey C, Málaga S. Síndrome emolítico urémico asociado a neumonía neumococica. Nefrología. 2007;27(4):505-8.
- Copelovitch L, Kaplan BS. Streptococcus pneumoniae-associated hemolytic uremic syndrome. Pediatr Nephrol 2008;23(11):1951-6.
- Waters AM, Kerecuk L, Luk D, Haq MR, Fitzpatrick MM, Gilbert RD, et al. Hemolytic uremic syndrome associated with invasive pneumococcal disease: the United kingdom experience. J Pediatr 2007;151(2):140-4.
- Geary DF. Hemolytic uremic syndrome and streptococcus pneumoniae: improving our understanding. J Pediatr 2007;151:113-4.

A. Venuta, P. Bertolani
Paediatric Department. Modena University Hospital. Modena (Italy).
Correspondence:
Andrea Venuta
Dipartimento materno-infantile. Policlinico di Modema. Modena (Italy).
andrea.venuta@unimore.it

Syndrome of inappropriate antidiuretic hormone hypersecretion caused by pneumonia diagnosed using a CT scan

Nefrología 2009;29**(5)**:497-498.

Dear Editor,

Syndrome of inappropriate antidiuretic hormone hypersecretion (SIADH) should be suspected in patients with hypoosmolar hyponatraemia, elevated urinary osmolality, sodium concentration in urine above 40mEq/l, normal acid-base balance and blood potassium, and a low concentration of uric acid in plasma. Extracellular volume should be normal and the presence of kidney failure, hypothyroidism, cortisol deficiency and diuretic treatment should be ruled out.[1]

Among the causes of SIADH, those that are secondary to lung diseases have been described.[2-4] Possible mechanisms of induced vasopressin secretion are: hypoxaemia and hypercapnia, haemodynamic abnormalities, alterations in the regulation and release of desmopressin caused by tumours, different drugs and stress.[4]

We would like to describe the case of a 68-year-old male patient who was taken to the Emergency Department because of diffuse abdominal pain and vomiting, as well as alarming symptoms that included slow mental reactions and disorientation. The patient had a history of chronic obstructive pulmonary disease caused by severe asthma treated chronically with oral corticosteroids, non-insulin-dependent diabetes mellitus, arterial hypertension and a transurethral resection of the bladder because of a neoplasia four years before. The patient's usual treatment consisted of metformin, simvastatin, enalapril, alendronic acid, calcium carbonate, omeprazol, methylprednisone and inhaled bronchodilators.

A blood test was carried out which revealed severe hyponatraemia 115mmol/l with plasma hypoosmolality 243mOsm/kg and hypouricaemia 2.4mg/dl, with normal blood potassium and renal function. There was elevated sodium loss in urine of 148mEq/l. The presence of hypothyroidism and adrenal failure was ruled out. The patient appeared to present SIADH and so water was restricted and hypertonic intravenous saline solution was administered. The patient's hyponatraemia progressively improved and his cognitive state normalised. When searching for the cause of SIADH, a brain MRI scan was carried out but no significant findings were made and a chest CT was performed which showed increased density of alveolar characteristics limited to basal segments of the right upper lobe that was very suggestive of pneumonia (figure 1A). However, a chest x-ray had been carried out on admission that did not show significant changes with regard to previous tests (figure 1B), the respiratory auscultation was normal and there were no leukocytes or other values that indicated infection. During admission the only significant symptom was an occasional fever of 37.2-37.4º C. Therefore, oral levofloxacin treatment was started and six days later a new chest CT showed significant improvement in the pneumonia. The urinary antigen tests for *Legionella* and *Pneumococcus* were negative. Gradually, the withdrawal of hypertonic saline solution was possible. It was administered until discharge following 15 days in hospital with water restriction, 6g per day of salt and 10mg/day torsemide. Plasma sodium levels remained stable at

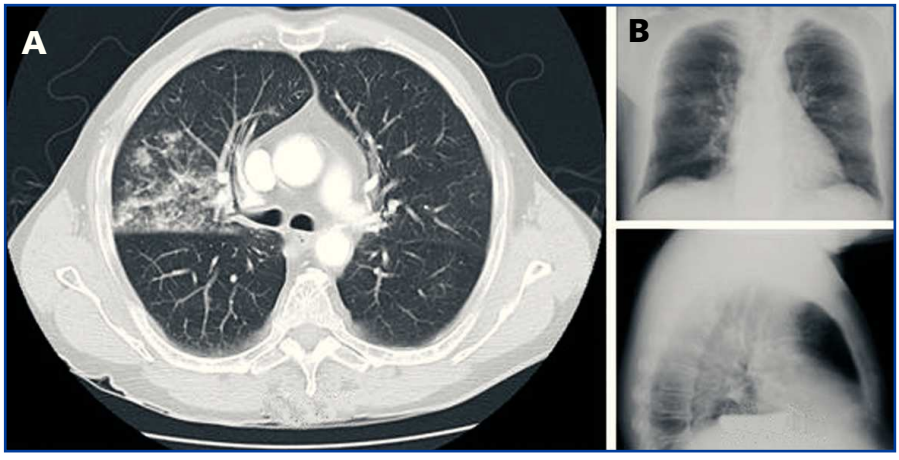

Figure 1. A y b.

131mmol/l. In the GP follow up, salt and diuretics were withdrawn and a month after admission the patient had fully recovered from SIADH, with plasma sodium levels 136mmol/l.

The following lung diseases have been described in the literature as potential inducers of SIADH: pneumonias, (viral, bacterial, tuberculous, mycotic), pumonary abscesses, asthma, atelectasis, pneumothorax and fibrocystic disease.[2-4] With regard to pulmonary tumours, small cell carcinomas are most likely to be involved, since induced SIADH is a paraneoplastic condition due to the ectopic secretion of vasopressin.[4] In the cases described, the symptoms of pulmonary diseases stand out first and foremost. In the case of our patient, it is interesting that the aetiological study of SIADH led to the diagnosis of pneumonia after the chest CT was carried out. Therefore we would like to highlight the importance of examining the lungs in cases of SIADH, especially when dealing with immunodepressed patients who are generally treated with corticosteroids and whose symptoms of infection may be latent, and thereby associated with very low clinical suspicion.

1. Rose BD, Post TW. Clinical physiology of acid-base and electrolyte disorders. 5th ed. New York: McGraw-Hill; 2001. p. 707-11.
2. Ellison DH, Berl T. The syndrome of inappopriate antidiuresis. N Engl J Med 2007;356(20):2064-72.
3. Adrogué H, Madias NE. Hyponatremia. N Engl J Med 2008;342(21):1581-8.
4. Anderson RJ. Hospital-associated hyponatremia. Kidney Int 1986;29:1237-47.

M. Picazo Sánchez, M. Cuxart Pérez, R. Sans Lorman, C. Sardà Borroy
Department of Nephrology. Empordà Health Foundation. Figueres Hospital. Figueres, Girona. Spain
Correspondence:
Montserrat Picazo Sánchez
Servicio de Nefrología. Fundació Salut Empordà. Hospital de Figueres. Figueres (Girona).
montserratpicazo@yahoo.es

Genital oedema in peritoneal dialysis

Nefrología 2009;29**(5)**:498-499.

Dear Editor,

Genital oedema is a relatively common complication in peritoneal haemodialysis. Different tests are used to diagnose the possible causes. We would like to present the case of an 80-year-old male patient with a history of colon cancer in 1995, monoclonal gammopathy of undetermined significance and chronic kidney disease of unknown origin, who began haemodialysis in 1999. After failing with several AV fistulas and being unable to create a new vascular access point, a permanent catheter was implanted in the right internal jugular vein in July 2004. In April 2009, an attempt was made to replace the permanent catheter but it was unsuccessful. Angiography of the vena cava revealed occlusion of the superior vena cava adjacent to the right auricle, with collateral circulation developing in the azygos vein.

Since vascular access for haemodialysis was impossible, a peritoneal catheter was implanted and the ventral hernia was repaired with mesh. This procedure was carried out by the Department of General Surgery and the removal of some loose adhesions was performed at the same time.

Seven days after the insertion of the peritoneal catheter, dialysis was started with a cycler and low infusion volumes. Forty-eight hours after starting the technique a very significant bilateral oedema affecting the scrotum and penis was observed.

Peritoneal dialysis was suspended for one week and the patient's condition progressively improved until the oedema disappeared. During this period haemodialysis was carried out using a femoral catheter with no complications.

Peritoneal dialysis was attempted once more using a cycler and low volumes but the scrotal oedema reappeared after the first session.

In order to establish the cause of the oedema, iodinated contrast was administered through the peritoneal catheter (iobitridol 300mg/l), regular abdominal control x-rays were carried out afterwards. At first 25ml of contrast was administered and the presence of the contrast was observed in the abdominal cavity (figure 1). Another 25ml was administered five minutes later and an x-ray in with the patient in the standing position was carried out, which showed the flow of the contrast from the peritoneal cavity to the scrotum in relation to the persisting peritoneo-vaginal canal (figure 2).

Genital oedema is well documented in peritoneal dialysis patients.[1] This phenomenon is associated with the flow of dialysis liquid from the abdominal cavity though inguinal hernias, a persisting peritoneo-vaginal canal, abdominal wall defects, etc.

The most commonly used method for diagnosis is a CT scan carried out after the infusion of two litres of dialysis liquid that contains contrast.[2-3] Another technique used is gammagraphy with Tc-99m.[4-5]

In the case of this patient, we have shown how a simple and easily accessible diagnostic procedure can help to identify the cause of genital oedema and establish the need for surgery.

1. Davidson PG, Usal H, Fiorillo MA, Maniscalco A. The importance of peritoneal imaging in the workup of genital edema in patients on continuos ambulatory peritoneal dialysis. Mt Sinai J Med 1999;66(2):125-7.
2. Leblanc M, Ouimet D, Pichette V. Dialysate leaks in peritoneal dialysis. Semin Dial 2001;14(1):50-4.
3. Litherland J, Lupton EW, Ackrill PA, Venning M, Sambrook P. Computed tomographic peritoneography: CT