

phenomenon in connective tissue disease, pulmonary hypertension and are currently recommended for scleroderma renal crisis. These have been used recently for treating cholesterol atheroembolisms as they quickly improve distal cyanosis, leg pain and kidney function.⁸⁻¹⁰

1. Thadhani RI, Camargo CA Jr, Xavier RJ, Fang LS, Bazari H. Atheroembolic renal failure after invasive procedures. Natural history based on 52 histologically proven cases. *Medicine* 1995;74(6):350-8.
2. Meyrier A. Cholesterol crystal embolism: diagnosis and treatment. *Kidney Int* 2006;69(8):1308-12.
3. Rodríguez Jornet A, Andreu FJ, Orellana R, Ibeas J, Vallvé M, Esteve V, et al. Diagnosis and prognosis of atheroembolic disease. *Nefrología* 2005;25(6):637-44.
4. Mignon F, Chaick A, Raynal P, Vereerstraeten A, Vanhaeverbeek M. A patient under hemodialysis with acute distal ischaemia: cholesterol cristal embolism. *Rev Med Brux* 2007;28(6):532-5.
5. Scolari F, Tardanico R, Zani R, Pola A, Viola BF, Movilli E, et al. Cholesterol crystal embolism: a recognizable cause of disease. *Am J Kidney Dis* 2000;36(6):1089-109.
6. Belenfant X, Meyrier A, Jacquot C. Supportive treatment improves survival in multivisceral cholesterol crystal embolism. *Am J Kidney Dis* 1999;33(5):840-50.
7. Stabellini N, Cerretani D, Russo G, Rizzioli E, Gilli P. Renal atheroembolic disease: evaluation of the efficacy of corticosteroid therapy. *G Ital Nefrol* 2002;19(1):18-21.
8. Grenader T, Lifschitz M, Shavit L. Iloprost in embolic renal failure. *Mt Sinai J Med* 2005;72(5):339-41.
9. Elinav E, Chajek-Shaul T, Stern M. Improvement in cholesterol emboli syndrome after iloprost therapy. *BMJ* 2002;324(7332):268-9.
10. adaceanu A, Avignon A, Ribstein J, Monnier L. Use of a prostacyclin analogue in cholesterol crystal embolism. *Diabet Med* 1998;15(3):262-3.

M.A. Rodríguez Gómez, M. Heras, A. Molina Ordas, M.J. Fernández-Reyes, R. Sánchez, F. Álvarez-Ude

Department of Nephrology. Segovia General Hospital. Segovia, Spain.

Correspondence:

María A. Rodríguez Gómez

Servicio de Nefrología. Hospital General de Segovia. Segovia.

asrodriguezgomez@hotmail.com

Streptococcus Pneumoniae infection and hemolytic uremic syndrome

Nefrología 2009;29(5):496-497.

Dear Editor,

Recently it has published in *Nefrología* a case of haemolytic uremic syndrome associated to pneumococcal infection (SP-HUS) in a 2-year-6-month old boy with pneumonia, that required veno-venous haemodiafiltration/haemofiltration during ten days. SP-HUS is an uncommon disease whose incidence, following invasive pneumococcal infection, is estimated at 0.4-0.6 %.

Its mortality rate is high, also in recent series, when compared with cases secondary to Shiga-like toxic-producing *E.Coli* infection (STEC-HUS). Exposition of Thomsen-Friedenreich cryptantigen (TF) present on the surface of erythrocytes, platelets and glomerular endothelial cells, by pneumococcal neuraminidase seems to trigger clinical manifestations.

Early recognition allows a proper treatment. Avoidance of plasma infusion and transfusions of unwashed blood products affects morbidity and mortality,¹ as IgM-containing blood derivatives may increase cellular damage.

We present a case of a 18-month old girl with high fever (40° C) and cough for five days; she was admitted for right pneumonia with pleural effusion. She was anaemic (Hb 5.6g/dL) with marked anisocytosis and schistocytosis and thrombocytopenic ($30 \times 10^9/L$).

Fibrinogen levels, and prothrombin and partial thromboplastin times were normal, while a direct Coombs' test was positive. Creatinine was mildly increased (61mmol/L) in presence of microhematuria and proteinuria. A rapid assay for detection of *Streptococcus pneumoniae* urinary antigen was positive. Subsequently *Streptococcus Pneumoniae* resulted from a haemoculture.

Intravenous antibiotic therapy (ceftazidime+vancomycin) was administered. The patient was transferred into a paediatric nephrology department. Six days after admission a drainage of the persisting pleural effusion was performed. Four transfusions of washed irradiated red blood cells were necessary to correct the severe anaemia.

Creatinine peaked at 79mmol/L, to return quickly toward normal values; diuresis and blood pressure were always normal. No dialytic treatment was required. One month after admission the patient was good with complete recovery; only microhematuria was persistent.

Our diagnosis was SP-HUS. The case in question differs from others described in literature for a very mild renal involvement that contrasts with the severe microangiopathic haemolytic anaemia.

It is hypothesized that various *Streptococcus Pneumoniae* serotypes with different neuraminidase activity can produce dissimilar manifestation of SP-HUS, ranging from isolated anaemia to full-blown HUS.² This wide spectrum of clinical presentations may cause an under-recognition of SP-HUS, with the risk of administering IgM containing blood derivatives. As in our case, invasive *Streptococcus Pneumoniae* infection associated with anaemia and Coombs' positive test, with no sign of DIC, can suggest the proper diagnosis.

- Herrero-Morín JD, Fernández N, Santos F, Rey C, Málaga S. Síndrome emolítico urémico asociado a neumonía neumocócica. *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. Bertolini

Paediatric Department. Modena University Hospital. Modena (Italy).

Correspondence:

Andrea Venuta

Dipartimento materno-infantile. Policlinico di Modena. 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 37.2-37.4° 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

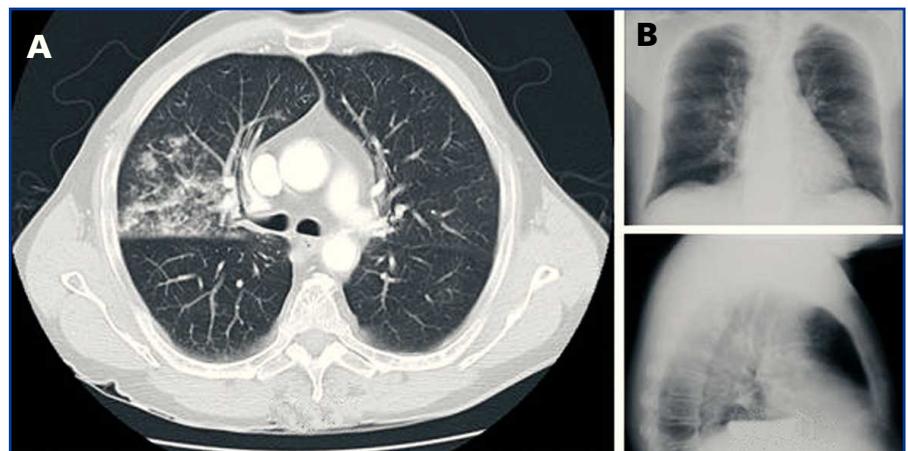


Figure 1. A y b.