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.1-10


Among the causes of SIADH, those that are secondary to lung diseases have been described. 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.

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