131mmol/l. In the GP follow up, salt and diuretics were withdrawn and a month after admission the patient had fully recovered from SIADH, with plasma sodium levels 136mmol/l.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

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Genital oedema in peritoneal dialysis

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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.

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Distal renal tubular acidosis with neurosensory deafness. Clinical evolution after 30 years of follow-up

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Dear Editor,

Primary distal renal tubular acidosis (dRTA) is a tubule disease characterised by metabolic acidosis with inappropriately alkaline urine, hypopotassaemia and hypercalciuria. It may be sporadic or hereditary, with a dominant or recessive autosomal pattern. The severity of the clinical spectrum may vary greatly, from slight, asymptomatic compensated acidosis with an occasional calculus to severe acidosis with delayed growth and early onset nephrocalcinosis caused by kidney failure. In general, patients with dominant-pattern dRTA have a milder phenotype than those with a recessive pattern.[1]

A subgroup of recessive dRTA patients suffers from progressive neurosensory deafness, caused by mutations in the gene that codifies sub-unit B1 in $H^{\pm}$ ATPase (ATP6V1B1).[2] The clinical profile for this tubular alteration, besides the deafness, is similar to that described for other types of dRTA.

We present the clinical evolution of a patient with dRTA with neurosensory deafness and chronic kidney disease secondary to nephrocalcinosis over a 30-year follow-up period.

Male patient aged 36. A few days after birth his clinical profile included frequent vomiting, polyuria, polydypsia, psychomotor delay, hyperchloraemic metabolic acidosis, hyperchloraemia, hypopotassaemia, alkaline urine and nephrocalcinosis, and he was diagnosed with dRTA. His family history included parents related by blood, one sibling with dRTA who died in a workplace accident and another healthy sibling.

The patient has been examined in our centre since he was six years old and receiving treatment with sodium citrate and oral potassium supplements.

Laboratory analysis data of interest: pH 7.07-7.33, bicarbonate 10-26mmol/L, Cl 113-124mEq/L, K 1.8-3.5mEq/L. Magnesium, calcium, phosphorus, ALP, PTH and Vitamin D levels are normal. Hb 12.2-18.7g/dl, Ht 38-55.3%. Urine: pH 7-8, anion gap 43mEq/l, Calcium 2.5-4.5mg/kg/day, Phosphorus 6-12mg/kg/day. Urine-plasma PCO_2 difference: 2mmHg (urine pH: 7.25 and plasma pH: 7.33), CaO/CrO index: 0.16-0.24.

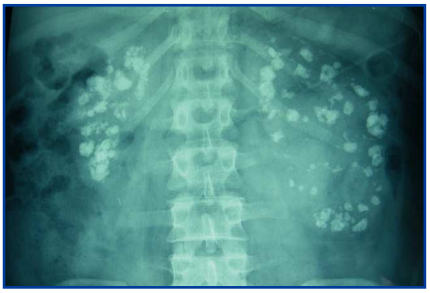

Figure 1.

The patient's treatment was irregular; he stopped taking his medication frequently, and often did not attend check-ups. This resulted in numerous admissions to hospital due to hypopotassaemic muscle paralysis and severe metabolic acidosis that would resolve quickly once bicarbonate and potassium treatment was administered.

When the patient was 16, a slow but progressive decrease in renal function began and the nephrocalcinosis became worse (figure 1). At 19, hearing loss was detected; the patient was diagnosed with bilateral neurosensory deafness, and needed a hearing aid.

At 25, he developed bilateral renal lithiasis and complications and hospital admissions have been common over the last 10 years. Most worthy of note are his bilateral renal colic episodes and the development of spontaneous steinstrasse, which on some occasions

Table 1.

Year	Hb (g/dl)	Ht (%)
1979	12.2	38
1981	13.9	38
1985	15	44.7
1990	15.2	44
1994	16.5	49
2000	17.1	49
2007	18.6	53.3
2009	18.7	55.3