resolved with spontaneous expulsion of the stones, and on others required urethral catheterisation with a double J stent and lithotripsy. Several episodes caused obstruction of the urinary tract with hydronephrosis and acute renal failure added to his chronic kidney disease; this resolved upon expulsion of the stones.

The latest check-ups show a progressive increase of nephrocalcinosis. There is also a parallel progressive increase of Hb and Ht levels currently with polycythaemia (table 1) and a normal EPO level (11.6mU/ml).

The patient currently maintains a stable creatinine clearance rate of about 45ml/min.

This case is important to us because our patient shows the main complications that can arise from this disease: 1) hypopotassaemic muscular paralysis as a result of discontinuing treatment frequently. 2) bilateral renal lithiasis, which in turn has caused obstructive acute renal failure requiring multiple urinary tract procedures, and has even provoked spontaneous steinstrasse, which is seldom described in this type of patient.[3] 3) polyglobulia, probably secondary to nephrocalcinosis, since the tissue hypoxia would induce an increase in EPO production which, although still in the normal range in our case, could be inappropriately high for such a high haemoglobin level. In the literature, this complication has very rarely been reported in association with dRTA;[4,5] 4) chronic kidney disease, which in our case is progressing very slowly, despite numerous complications (20 years have passed since the creatinine clearing rate began to decrease) and which has been stable over the last few years.

We conclude that with dRTA, supervising compliance with the treatment is very important for avoiding complications, some of which are potentially severe and completely avoidable. Paying attention to polycythaemia secondary to nephrocalcinosis is also crucial, as the condition could foster the development of thrombotic events in these patients. Despite everything, the renal failure has been progressing very slowly over 30 years of observation.

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Peritoneal dialysis after removing the catheter because of peritonitis

Nefrología 2009;29**(5)**:500-501.

Dear Editor,
Peritoneal dialysis (PD) after removing the catheter due to peritonitis in patients with chronic kidney disease is a treatment option that is certainly not free from subsequent complications and technique failures.

We present two clinical cases with early malfunction of the peritoneal catheter due to multiple adhesions.

Case 1: patient 67 years of age with a history of diabetes mellitus on automatic peritoneal dialysis (APD) who was admitted with a case of peritonitis and exit orifice infected with *Burckolderia cepacia* and an abdominal ultrasound image suggesting chronic cholecystitis. Given the poor clinical evolution and the persistent cloudy peritoneal fluid, we decided to remove the catheter and perform cholecystectomy in the same surgical procedure. Subsequently, the patient underwent haemodialysis (HD) and received antibiotic treatment (cyprofloxacine and meropenem) for two weeks after removal of the catheter according to the antibiogram.

The patient decided to have the new peritoneal dialysis catheter implanted a month and a half after its removal (abdominal CAT showed no changes). The procedure was performed by general surgery, during which the lax adhesions that were observed were liberated. One month after implantation, we observed that draining was difficult. A peritoneography was taken (50ml iobitridol 300mg/l), revealing the presence of contrast limited to a small cavity (figure 1).

In light of these results, we decided to transfer the patient to HD and remove the peritoneal catheter, which was completely clogged by omental adhesions.

Case 2: male patient aged 78 with a personal history of ischaemic-hypertensive cardiopathy, diverticulosis, chronic kidney disease (ischaemic type), undergoing APD since February 2005. He had suffered various peritonitis episodes: *Klebsiella* in March 2005, *E. coli* in August 2005 and *Serratia marcescens* and *E. faecalis* in September 2005. The abdominal CAT images were compatible with diverticulitis, and antibiotic treatment was administered. In October 2006, he presented a new episode of peritonitis with *Pseudomona aeruginosa*, and for this reason, it was decided to remove the peritoneal catheter, transfer the patient to

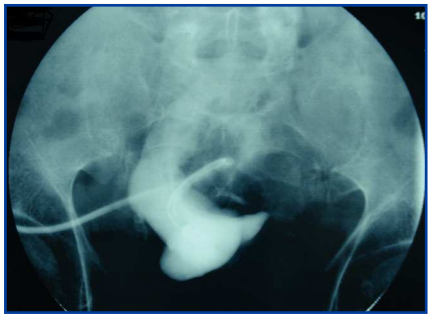

Figure 1.

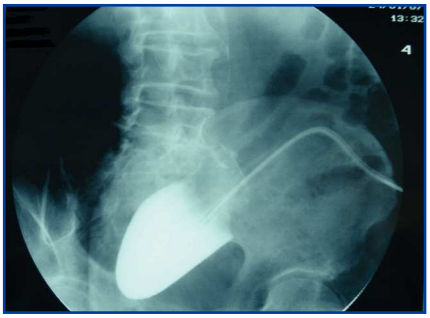

Figure 2.

haemodialysis and administer further antibiotic treatment according to the antibiogram.

In December 2006, at the patient's request and due to significant vascular access difficulties, we decided to implant a peritoneal dialysis catheter (abdominal CAT was normal), liberate lax adhesions during the procedure and verify catheter function whilst in the operating room.

At 15 days after implantation, the peritoneal catheter was malfunctioning and provoking difficulties during both infusion and drainage. The peritoneography showed an image similar to that described for the previous case (figure 2).

The patient made the definitive transfer to HD once the catheter was removed; multiple adhesions were observed.

Removal of the peritoneal catheter is necessary when treating certain types of peritonitis, principally those caused by funghi, enterobacteria, or where there is a coexisting subcutaneous tunnel infection.

There is no reliable objective method for identifying irreversible peritoneal damage prior to reinsertion of a new catheter. Ultrasound and abdominal CAT images are the most widely-used tests, but they have a low sensitivity.[1]

After a review of 189 cases of peritonitis in which a catheter was removed and subsequently replaced, Troidle et al. concluded that only 20% continue with that method one year after the removal.[2]

If the decision is made to return to PD, we recommend implanting the catheter using open surgery or laparoscopic surgery that allows us to obtain more information on the abdominal cavity condition.[3] This reimplantation should be performed at least 3-4 weeks after remission of the infection.[4]

In order to make this decision, we must take into account such factors as the severity of the peritonitis, residual diuresis, previous ultrafiltration capacity, aetiological agent, etc. With all of the above in mind, the decision must be a personalised one.

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Isolated tubulo-interstitial nephritis in a patient with systemic lupus erythematosus

Nefrología 2009;29**(5)**:501-502.

Dear Editor,

Systemic lupus erythematosus (SLE) is an inflammatory disease with systemic effects.[1] At least 50% of all patients present signs of nephropathy during their illness, and nearly half present diffuse proliferative nephritis.[2] Tubulo-interstitial nephritis as an isolated histological lesion is infrequent in SLE patients, and to our knowledge, few published cases exist in the literature.[1-2]

We present the case of a 67 year old female patient diagnosed with arterial hypertension. She was admitted with a sensation of nausea, urinary infection and anaemia. Laboratory analysis: Ht 27.8%; Hb 9.5g/dl. ESR 63mm. Urea 182mg/dl; creatinine 4.7mg/dl; calcium 9mg/dl; phosphorus 4.2mg/dl and total proteins 9g/dl. Creatinine clearance rate (Cockcroft-Gault formula): 17.73ml/min. Immunoproteins and complements were normal. Kappa chains 774mg/dl, Lambda chains 392mg/dl. In the proteinogram, we observed a wide-base peak in the Gamma region with increased IgG (193%) and light Kappa (191%) and Lambda chains (180%). K/L index = 1.97. Light chains in urine: Kappa chains 13.7mg/dl (0-0.7); Lambda chains 6.880 (0-0.39). A bone marrow aspiration and biopsy was performed, with a normal result. TSH: 4.85μUI/ml, free T4 1.03ng/fl; anti-TPO antibodies 22.5UI/ml; antithyroglobulin antibodies 115.3UI/ml. PTH: 110pg/ml. Urine (test strip): Proteins 25mg/l; sediment: abundant leukocytes. Tumour