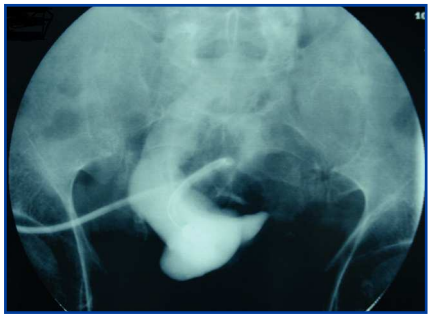

Figure 1.

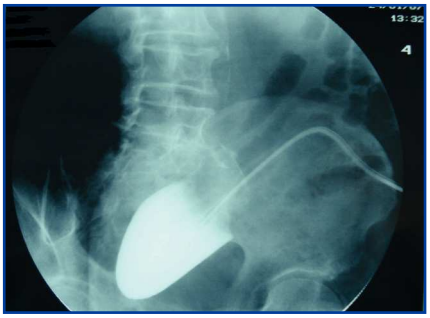

Figure 2.

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

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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

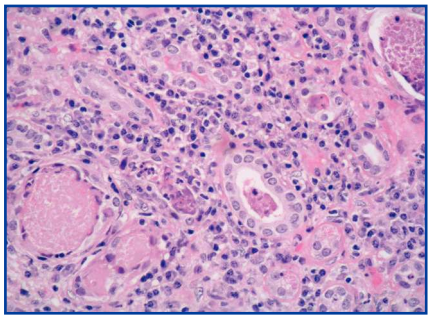

Figure 1. H-E (400x). Close-up of the lymphoplasmocytic infiltrate affecting tubular walls and granular cylinders with some polymorphonuclear cells in the tubular lumens

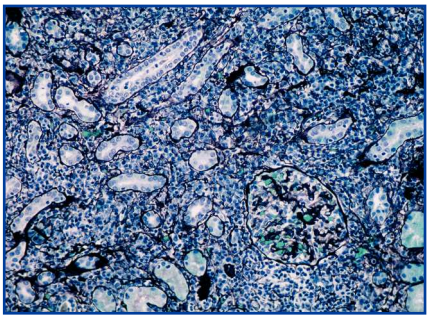

Figure 2. Jones' silver stain (x200). Hypercellular interstitium with a lymphocyte and plasmatic cell infiltration that provokes tubule erasing. Glomerules seen without alterations.

markers: normal. Viral serology: negative. Autoantibodies: ANA+, anti-DNA positive.

A percutaneous renal biopsy was performed (with 18 verifiable glomerules): No significant glomerular alterations. The tubules presented dilated lumens occupied by granular cylinders containing cellular detritus. Tubular atrophy foci and hyaline cylinders were present (figure 1). In the interstitium and vessels, abundant lymphocyte infiltrates, mostly from plasma cells that broaden the interstitium, provoke the collapse and disappearance of tubules as well as the erosion and epithelial infiltration of basement membranes (figure 2). With immunofluorescence, we observed granular deposits in the arterial walls of complement C'3 (++) and tubular cylinders of IgA (++). Using immunohistochemical techniques, we found linear deposits of light kappa chains on tubular basement membranes. Anatomical-pathological diagnosis was inflammatory tubulo-interstitial nephritis. This morphological profile appears in 10% of patients who suffer plasma cell dyscrasias, most frequently in myeloma with a predominance of light K chains.

In most cases of SLE, the glomerular condition is the main histological lesion. An isolated condition affecting the tubule and interstitium is rare.

We ruled out other causes of tubulo-interstitial nephritis, such as drugs or toxins. The intense presence of tubulo-interstitial damage in absence of significant glomerular damage supports the theory that immune complexes are bound to one or more tubulo-interstitial autoantigens that are not expressed in the glomerulus. The underlying mechanism represents *in situ* formation of immune complexes as a result of circulating autoantibodies binding to antigens.[3] Although the exact mechanism is unknown, it is possible that its virulence depends on the structural characteristics of the antigen-antibody union region, the isotope, and the isoelectric charge.[3] A recent study describes how the union of T CD4+ cells with antigens in the glomerular basement membrane can begin the glomerular damage that will trigger glomerulonephritis. It is possible that a similar mechanism might participate in the pathogenesis of tubulo-interstitial nephritis in such a way that cell immunity damages tubular antigens; this would trigger an interstitial condition, as in other interstitial nephropathies.[4]

The treatment of tubulo-interstitial nephritis in SLE is not well-established, but it seems not to require immunosuppressant therapy, and it might respond to low doses of oral steroids.[5]

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