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Partial recovery of obstructive kidney disease after 16 months on haemodialysis

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Dear Editor,

Urinary tract obstruction can cause a greater or lesser degree of kidney disease depending on its duration and severity. There is a lot of literature on the effects of kidney obstruction from an experimental point of view.[1-3] However, there is less experience in humans, although the published works agree that the progressive deterioration of kidney function after removing the blockage is unusual in most patients, especially during the first year.

We present the case of a patient with kidney disease secondary to obstructive prostate adenoma who required regular haemodialysis treatment. After 16 months, he showed partial kidney function recovery, which was sufficient to allow dialysis treatment to be stopped.

A 50 year-old man with a history of prostate syndrome was admitted into emergency due to progressive deterioration of his general condition. Physical examination revealed pale skin and mucous membranes and suprapubic dullness. The most relevant laboratory data showed Hb 5.9g/dL, urea/creatinine 425/19mg% and severe metabolic acidosis. The later immunological study (ANA, addition, etc) was normal or negative, proteinuria/24 hours 1.2g, and sediment with haematuria and pyuria with negative culture. An ultrasound showed a severe bilateral hydronephrosis with enlarged prostate, a bladder balloon with significant signs of postvoid residual bladder control. The patient brought an analytical examination performed 5 months earlier in which the only remarkable value was a creatinine of 1.6mg%.

The catheter was inserted and initial humoral regulation measurements were taken. Postobstructive polyuria was observed without improvement of kidney function. 2 packed red blood cell units were transfused and haemodialysis started. At 2 months the patient showed a marked clinical improvement with daily urine output of 1.5-2 litres, but with no evident improvement in the analytical results. A kidney biopsy was performed which showed the presence of 17 glomeruli of normal appearance with minor interstitial lymphocyte inflammatory accumulations, oedema of the tubular epithelium and isolated intratubular hyaline casts with a negative immunofluorescence study, all compatible with chronic moderate interstitial nephritis. Subsequently, retropubic prostate adenectomy was performed with a histological study of fibroadenoma nodular hyperplasia. A new kidney ultrasound was perfectly normal. After 16 months, residual clearances of 18ml per minute were observed in the analytical tests, so it was decided to stop dialysis. These values were stable 9 months after abandoning dialysis.

Obstructive nephropathy is a common cause of chronic kidney disease with a bimodal presentation affecting paediatric and elderly patients.[4] There are few studies on the development of obstructive nephropathy in humans, making it difficult to extrapolate experimental effects in clinical practice. In addition, the obstruction in humans is often incomplete and of a subacute or chronic course, and in most occasions it is very difficult to assess from the beginning. Generally the recovery of kidney function is observed between 7 and 10 days after the liberalisation of the urinary tract, although long recovery periods for kidney function have also been found.[5] There are few studies relating to the prognosis of obstructive kidney disease that requires dialysis treatment.[6,7] Ravanan et al[8] has the largest number of patients, with analysis of the behaviour of kidney function after unblocking in an initial group of 104 adult patients with severe and chronic obstruction. 28 of them required treatment with haemodialysis despite the unblocking. After the third year, only 9 patients needed replacement therapy and another patient underwent a transplant. Kidney function improved during the first 3 months, and remained stable in most patients after 3 years. A small number of them observed no improvement after the unblocking treatment and were more likely to require long term haemodialysis.

Thus, despite the fact that obstructive nephropathy can be accompanied by the emergence of an apparent terminal chronic kidney disease, partial improvement in kidney function may be achieved after long periods, even when the patient requires replacement therapy.

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Focal segmental glomerulosclerosis associated with polycythaemia vera

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Dear Editor,

Focal segmental glomerulosclerosis (FSGS) is characterised by the presence of nephrotic syndrome, hypertension and progressive deterioration of kidney function. Although in many cases its aetiology is unknown, it has been associated with inherited disorders, viral infections, induced by toxic substances and with hyperfiltration situations.[1]

Polycythaemia vera (PV) is a myeloproliferative disorder of unknown aetiology characterised by excessive production of normal erythrocytes, leukocytes and platelets.[2]

Glomerular involvement in PV is rare. We present a patient diagnosed with PV with nephrotic syndrome secondary to GSF and progressive kidney disease.

An 83 year-old woman, diagnosed with PV 4 years earlier, was admitted for the study of nephrotic syndrome and kidney disease of 2 years evolution.

There was no family history of polycythaemia. Among her background was: left nephrectomy due to hypernephroma at 63 years, bronchiectasis with recurrent bacterial superinfections, hypertension controlled with medication and celiac disease well controlled through diet. Four years earlier she was diagnosed with PV after a bone marrow biopsy, including details of polycythaemia and thrombocytosis, well controlled with hydroxyurea treatment (500mg/day).

In March 2007, she began with nephrotic range proteinuria (4.2g/day) with Crs 1.1mg/dL; antiproteinuric treatment was started with telmisartan 80mg/day and spironolactone 50mg/day. A month later the proteinuria dropped to 1.2g/day with no change in the creatinine. She was admitted to hospital in June 2007 for severe hyponatraemia (107mEq/L), symptomatic, with hyperkalaemia (5.7mEq/L) and metabolic acidosis (pH 7.34) secondary to treatment with spironolactone. Due to the persistence of nephrotic proteinuria and her history of bronchiectasis, a biopsy of rectal and abdominal fat was performed which discarded the existence of amyloidosis. Throughout its evolution, the proteinuria varied between 4-10g/day and started with lower limb oedema, decreased total protein and albumin (5.4/2.7g/dL) and a progressive decline in the glomerular filtration rate (Crs 1.6-1.7mg/dL).

In May 2009 she was admitted to hospital due to a deterioration in her general condition, oedema, Crs 3.6mg/dL and proteinuria 8.4g/day despite treatment with ARB. On admission, her blood pressure was 137/82mmHg; in the physical examination her systolic ejection murmur was heard II/VI in the left sternal border, and she had bilateral pitting oedema up to the root of her thigh. CBC: haemoglobin 16.8g/dL, haematocrit 56%, RBC 6,750,000/µL, 11,690/µl leukocytes with normal formula and platelets 460,000/µl. Crs 4.3, urea 102 (mg/dL). Total protein 5.9, albumin 2.5g/dL, cholesterol 188, triglycerides 260, uric acid 9.8mg/dL. Proteinuria 6.9g/d; 1.4 sediment erythrocytes/field and 5-10 leukocytes/field. Immunology: ANA, anti-DNA, ENA, ANCA and anti-GBM negative. Complement: $C_3$79, $C_4$30mg/dL. CRP 0.37mg/dL. Rheumatoid factor negative. IgG 820, IgA 185, IgM 188mg/dL. Kappa 635, lambda 515mg/dL. Kidney biopsy was performed with 8 glomeruli of which two were completely sclerosed, and the remaining six, one global mesangial expansion with increased mesangial cells could be seen and the other five had segmental proliferative lesions without necrosis accompanied by moderate epithelial proliferation. The interstitium showed moderate fibrosis with tubular atrophy and occasional chronic inflammatory infiltrates. Arterial and arteriolar vessels with hyperplastic lesions with occasional hyaline lesions. These findings were indicative of *cellular variant segmental proliferative glomerulonephritis*.

Treatment was initiated with three shocks of 125mg of 6-methylprednisolone followed by prednisone (1mg/kg/day) and mycophenolate mofetil (360mg/12 hours). No evidence of a decrease in proteinuria was seen and kidney function deteriorated progressively with clinical uraemia. So it was decided to make the right jugular catheter permanent and initiate treatment with periodic haemodialysis. A progressive improvement in the symptoms was seen. Treatment with mycophenolate mofetil was discontinued and prednisone was gradually withdrawn.

The patient developed a nephrotic syndrome secondary to FSGS 4 years after detection of the polycythaemia. It met the WHO criteria for diagnosis of