the rest of the biochemical parameters being normal. Her urine showed proteinuria of 5.7g/24 h, with 10-12 red blood cells/field in the sediment. The immunological test showed: negative ANA and ANCA, normal immunoglobulin levels and a normal complement system. The ultrasound revealed normal-sized hyperecogenic kidneys. Due to these findings, it was decided to carry out a renal biopsy which showed the following: 5 glomeruli, one with segmental hyalinosis and another with complete sclerosis. Mesangial thickening and diffuse interstitial fibrosis with tubular atrophy and dilation present in those not suffering from sclerosis. Mesangial deposits of IgA (+++) and C3 (+++). Anatomopathological diagnosis of IgA nephropathy with chronic tubulointerstitial nephritis. The patient began treatment with calcium antagonists with an improvement in the pressure figures; however, renal function continued to worsen, which is why it was necessary to prepare for the substitutive treatment.

Lithium salts are widely used in bipolar disorder. The treatment of the chronic form of the disorder with these salts is associated with different types of renal damage, including nephrogenic diabetes insipidus, metabolic acidosis, chronic nephritis and hypercalcaemia. The most important predisposing factor is the time of exposure to lithium, while other factors include age, episodes of lithium poisoning and comorbidity. The anatomopathological substrate of the toxicity from lithium is interstitial fibrosis, which can appear 5 years following treatment. At the same time, focal segmental glomerulosclerosis is associated with tubulointerstitial changes. Renal tubular cysts are a sign of tubular damage, which is manifested as dilation of the distal segment and the tubular collector. The progression of nephritis brought about by lithium is slow, considering a drop in the glomerular filtration rate of 2.2ml/min per year of exposure, with a very low rate of terminal chronic kidney disease in the various published studies. Interruption of the treatment with lithium salts does not achieve the recovery of the patient's baseline renal function. Instead, in some cases, the deterioration continues at a similar rate following its interruption. It is thought that a point of no return exists, after which renal fibrosis continues despite discontinuing the aggression that triggered it in the first place. This depends on the anatomopathological substrate at that moment, with better response of the illness to small changes. In this instance, the renal biopsy allowed us to position the clinical condition, and it was decided to continue with the lithium treatment. This could not fully justify the patient's progress in full view of the biopsy result that was compatible with IgA nephropathy, which justified the clinical condition described.

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Previous ischaemic optic neuropathy in haemodialysis

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Dear Editor,

Optic neuropathy is a syndrome and not a mere common injury of the optic nerve; its aetiology is broad and greatly varies. Ischaemic optic neuropathies influenced by local and systemic factors are particularly noteworthy, many of which are difficult to understand. These processes occur in the field of other specialisations with surgical manoeuvres and/or aggressive diagnoses, in which the patient is compromised, as the initial diagnosis could be problematic.[1] Ophthalmologists are well aware of the process, however, doctors of other specialisations do not realise the possibility for this clinical condition, being familiar with its existence only when it appears. Various risk factors exist for ischaemic optic neuropathy (ION), however, we must emphasise sudden hypotension, which does not allow for the autoregulation mechanisms of the optic nerve to compensate, particularly if the patient suffers from previous hypotension, anaemia, sudden and/or recurrent haemorrhage, serious facial oedema, chronic kidney disease, bleeding surgeries and, generally, all situations associated with arteriosclerosis.

Based on the few reports in the literature of patients with chronic kidney disease (CKD), we report the cases of 2 patients in haemodialysis with a diagnosis of acute bilateral loss of vision due to a previous ischaemic optic neuropathy (PION).

Case 1

A 29-year-old woman with a diagnosis of CKD owing to GEFS was hospitalised for haemodialysis at the age of 25. Three years after the haemodialysis, she showed signs of severe secondary hyperparathyroidism (SHPT) with PTH i > 1,000pg/ml, ostealgia, asthenia, pruritus and persistent symptomatic hypotension. There were no arterial or cardiovascular calcifications. A diagnostic ultrasound of the parathyroid glands was carried out with no visualisation with ^{99}Tc and MIBI with diffuse increase in the fixation of the left and right inferior parathyroid lobes. Treatment for the SHPT was carried out with calcium binders and intermittent calcitriol IV for hyperphosphatemia. Subtotal parathyroidectomy was prescribed, taking into consideration the patient's age and transplant suitability. The previous laboratory

results were as follows: HCT 37%, Hb 12.1, Ca 9mg/dl, P 6mg/dl, K 5 meq/l. Normal coagulation test results. Biopsy of the parathyroid glands: bilateral hyperplasia. During the surgery she developed hypotonia (average BP of 90/60). Six days following surgery, she experienced partial loss of vision in the RE and total in the LE, mild hypocalcaemia and symptomatic hypotension. Laboratory: HCT 36%, Hb 12, Ca 6.3, Ca i 0.2l, P 3.5 and Mg 1.9. The hypocalcaemia values were corrected.

Normal results of the collagen and coagulation tests. Results of anticardiolipin antibodies, lupus anticoagulant, ANCAp and c and serology for toxoplasmosis and syphilis were negative.

Neurological examination was carried out, showing right nasal hemianopsia and vision restricted to profiles on the left eye. Discus-shaped pupil in the RE and LE at 3 and 4mm, respectively. Ophthalmological examination: AV with vision, counted fingers at 50cm in the LE and slight drop in vision in the RE. A reactive mydriasis in the LE and reactive in the RE. Inner eye: attached retina, normal macula and congestion of the papilla with degenerative oedema (L and RE) and PO 10/10mmHg. Normal MRI of the brain and bulb. Evoked visual potential in the RE and LE of average range and prolonged central latency, compatible with moderately affected areas of the optical route that is consistent with optic neuropathy. Visual sharpness: 0.5 in the LE and 0.8 in the RE. Campimetry: outwith the normal limits for the LE, with an alteration in the relative feeling and a restriction of the blind spot (increased). There was a total decrease in feeling in the LE.

Corticosteroids (prednisone) were prescribed as a treatment at a dose of 1mg/kg/day for 30 days.

The papillary oedema increased in both eyes, with a vision restricted to profiles in the RE and amaurosis in the LE.

Within two months, an almost total atrophy of the right optical nerve was detected in the inner eye, with dilation and venous tortuosity, while there was total atrophy of the left optical nerve.

Case 2

In 1993, a 25-year-old man with CKD owing to obstructive nephropathy was hospitalised to receive haemodialysis. In November 1995, he was given a renal transplant from a compatible donor. In December 1995, he experienced FAV and received blood transfusions at various times. In September 2003, he was once again admitted to hospital for haemodialysis due to the transplant's chronic nephropathy. In the period from 2004-2009 he suffered from multiple blockages of the vascular access.

Anaemia with partial response to EPO. Severe SHPT with persistent hyperparathyroidism. Did not follow the diet and took in an excessive amount of fluids, leading to a significant interdialytic excess weight and severe hypotension. Deficient dialysis suitability with minor KTVsp at 1.4. Haematological tests were carried out for his medical history with thrombophilia, consistent with the inhibitory lupus effect and a slightly increased plasma homocysteinemia. Treatment with acetylsalicylic acid, folic acid + vitamin B complex and anticoagulation was administered. A short anticoagulant treatment was administered for lack of performance.

In March 2009, the patient showed symptoms of loss of vision in the RE and partial loss of vision in the LE. Inner eye: with bilateral papillary oedema. Normal ocular pressure. There is no medical history of headaches. Normal MRI of the brain. Neurological examination: negative AV light, abolished reflexes (areactive mydriasis). Inner eye: diffuse-edge papilla with papillary oedema + (no haemorrhage) (LE), diffuse-edge papilla with papillary oedema+++. Visual PE with severely affected areas of the optical route that is consistent with optic neuropathy. Results of laboratory tests: HCT 26, Hb 8.1, Upre 129,Upost 18, P 6.9, Ca 8.2 and PTH 2.431. Normal results for connective disease tests. Normal values for anticardiolipin IgG and IgM. Results of ANCAp and c and serology for HBs Ag, HCVAc, HIV, toxoplasmosis and syphilis were negative.

Beginning of treatment with: corticosteroids (prednisone) at a dose of 1mg/kg/day, with no response at 30 days. Inner eye test (2 months), bilateral atrophy. The patient is clinically damaged with bilateral amaurosis.

Discussion

We have come across 16 cases of PION in patients with substitutive renal treatment, 10 of which correspond to patients following a programme of periodic haemodialysis [2-13] and the rest to DPCA.[11,12] In general, all cases shared a medical history of hypotension episodes in dialysis,[2,8,13] one of which showed no hypotension,[9] while another was associated to Sildenafil.[10] The visual deficit was bilateral, and in 15 of the cases it manifested as anterior ischemic optic neuropathy, with only one case showing it afterwards. They showed a partial response to a corticosteroids dose. In the cases presented, we could not avoid thinking about the possible association of severe secondary hyperparathyroidism with calcific uremic arteriolopathy (calciphylaxis) in arterioles that moisten the optic nerve. Korzets et al[13] observed an acute loss of vision in 2 patients with a diagnosis of PION, a biopsy of the choroid of the temporal artery of whom showed signs of hypotension and calcification, taking into account that hypoperfusion could have occurred from calciphylaxis of the arterioles moistening the top of the optic nerve. Furthermore, the association of the ischaemic ocular pathology with the antiphospholipid syndrome is well known,[14,15] as shown in the second case, where the patient has clinical and laboratory symptoms of primary antiphospholipid syndrome (PAPS), and in which the ocular affectation associated with the presence of these symptoms includes *v*aso-occlusive retinopathy, estimated to be present in 29% of the patients with PAPS, and optic neuropathy of a likely ischaemic nature.

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Use of Levosimendan in acute heart failure and its effect on renal function

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Dear Editor,

Levosimendan is a relatively new inotropic agent, used in cases of decompensated heart failure, which has shown, in some reported cases, improvement in the patients' renal function.

We report the case of a 70-year-old male patient, monitored for grade III chronic kidney disease with no affiliated aetiology, with CCr of 45ml/min in the last examination. His medical history includes the following: monoclonal gammopathy of an uncertain significance; femoral cutaneous neuropathy; and multifactorial heart failure (ischaemic heart failure, non-ischaemic dilated cardiomyopathy, moderate pulmonary hypertension, heart disease with a pacemaker), with several episodes of decompensation causing his hospitalisation. He followed a regular treatment of Acenocoumarol, Carvedilol, Enalapril, Torasemide, Digoxin, Atorvastatin, Alopurinol, Folic acid, Vitamin B_{12}, Ferrous sulphate and Neorecormon.

His hospitalisation was caused by a new decompensation of his heart failure, a deterioration of his dyspnoea, important levels of orthopnoea, episodes of paroxysmal nocturnal dyspnoea, ascites and oedematisation of limbs with a progressive decrease of diuresis. Moreover, he mentioned profuse haemorrhage due to a haemorrhoidal condition, and the examination confirmed a deterioration of the renal function.

During examination the patient experienced tachypnoea, with labial cyanosis, jugular ingurgitation and bad tolerance to the supine position. BP: 116/54. The cardiopulmonary auscultation showed rhythmic tones at 69 bpm, with R3 and a systolic heart murmur of 1/6 in the mitral and aortic areas; presence of rhonchi and diffuse wheezing, with crepitations in both pulmonary bases. The patient showed signs of hepatomegaly and inferior limbs oedematised until the knee with fovea. Diuresis remained at a daily level of around 2 litres. The examination revealed a deterioration of his renal function with Cr 3mg/dl, CCr estimated at: 24ml/min (baseline levels of 2mg/dl and 45ml/min, respectively). Urea 173mg/dl, Na 132mg/dl and K 6.4mg/dl. Hb: 5.7g/dl, requiring blood transfusion. The ECG showed a biventricular pacemaker rhythm at 70 bpm, and the thorax X-ray revealed an image of severe cardiomegaly with signs of vascular redistribution. Colonoscopy was carried out with anal fissure being the sole finding. The ECG showed severely dilated left cavities with severe global hypokinesia and FE estimated at 21%.

Intensive diuretic treatment was carried out with a poor response, leading to the decision to treat with Levosimendan in perfusion at a dose of 12.5 mg IV for 24 hours. The patient progressed satisfactorily for his congestive condition: dyspnoea disappeared and renal function