Nephropathy induced by BKV or co-infection?

Nefrología 2009;29**(1)**:92.

Dear Editor,

The prevalence of BKAN varies between 1 and 5.5% and leads to the loss of graft in 65% of those affected.[1] The active viral infection can be confused with or even coexist with cell rejection processes.[2] Diagnosis is based on the detection of Decoy cells in the urine, demonstration of viraemia and viruria via CRP and histological studies.

Adequate screening and a reduction in immunosuppressive treatment are currently effective strategies for preventing and managing BKAN.

Co-infection due to Cytomegalovirus (CMV) and polyomavirus is possible, although uncommon. The introduction of potent immunosuppressive drug such as tacrolimus and the use of endoluminal catheters[3] seem to contribute to the greatest incidence of these infections.

We present the case of a 50 year old male patient with advanced Chronic Renal Failure of uncertain origin who received a cadaveric kidney transplant seropositive for CMV. Cold ischaemia time: 18 hours. Ureteral anastomosis and insertion of double-J catheter. Induction and maintenance were performed with tacrolimus, MMF and prednisone. High levels of FK were maintained for the first two months (average: 17.9ng/ml.) Delayed graft function, with Cr 1.2mg% at 36th day. After the third month Glomerular Filtration (GF) began to deteriorate (Cr 2.45mg%), coinciding with the finding of CRP in the urine positive for BK virus (50 x 10^6 copies) and Decoy cells. Plasma BK CRP CMV antigenaemia and viraemia were negative. Microalbuminuria (561mg/24 h), proteinuria (1.26g/24 h) and haematuria were found. A biopsy was performed under the suspicion of BKAN. This revealed interstitial infiltration of T lymphocytes and macrophages with significant tubulitis. Several nuclear and cytoplasmic viral inclusions were observed in the cortical and medullary tubes and in the vascular endothelium, with marked cytomegalia. Immunohistochemistry was positive for CMV and SV40. HLA-DR expression was restricted to lymphoid cells.

The diagnosis was interstitial nephritis associated to CMV and the BK virus. Mycophenolate mofetil (MMF) was suspended, the dose of tacrolimus was reduced and ganciclovir was initiated. The double-J catheter was removed. The patient made satisfactory progress and the number of BKV copies in the urine decreased (10.)[6] Microalbuminuria, proteinuria and haematuria disappeared. Renal function improved and two months after diagnosis, reached Cr de 1.6mg%.

In the case in question, deterioration of renal function associated with the appearance of viruria due to BKV led us to initially consider BKAN. The findings of nuclear and cytoplasmic inclusions, typical of CMV, in the renal biopsies, presented the possibility of co-infection which was confirmed via positivity for SV40 and the anti-CMV monoclonal antibody.

Although the majority of the series relates the existence of nephropathy with high viral loads in the plasma,[4] in our case repeated negativity for viraemia for BK and CMV is to be noted despite using highly sensitive techniques. Moreover, it was thanks to the renal biopsy that we were able to reach the correct diagnosis and perform the correct treatment.

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Keywords: *BKV-nephropathy. Co-infection. Cytomegalovirus. Kidney transplantation*

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Treating distal calciphylaxis with therapy associated with sevelamer and bisphosphonates

Nefrología 2009;29**(1)**:92-93.

Dear Editor,

Calcific uremic arteriolopathy (calciphylaxis) has an incidence of 1% and prevalence of 4% in the dialysis population. The pathogenesis is not clear and the different treatments (control of Ca-P metabolism, parathyroidectomy, adequate management of wounds, hyperbaric chamber, non-calcium based binders, bisphosphonates, sodium thiosulfate, etc.) did not show any improvements in the prognosis.

The clinical case of a male patient aged 55 diagnosed with Chronic Renal Failure (CRF) secondary to diabetic nephropathy and arterial hypertension is presented. The patient was admitted to Haemodialysis in May 2003.

He had antecedents of diabetes type 2 treated with oral hypoglycaemic drugs and then NPH insulin.

Nonproliferative diabetic retinopathy. Obesity (Body Mass Index [BMI] 36.) Dyslipidaemia. Ex smoker (2 packets a day.) Long-term AHTN (20 years) treated with Angiotensin Converting Enzyme inhibitors (ACE inhibitors) and calcium channel blockers. Peripheral vasculopa-