an increase in permeability and oedema in the vascular wall mediated by mastocyte degranulation and release of mediators, which were probably the cause of the premature fistula thrombosis. The patient retrospectively reported a history of oedema from balloons and preservatives, and pruritis of the tongue from some fruits. There is a high association (30-80%) between latex allergy and sensitivity to certain fruits, particularly those of tropical origin, coming from plants that are botanically unrelated to the plant from which latex is extracted.[1] An association has also been described between latex and ethylene oxide, and, even though the role of ethylene antioxidant antibodies in the pathogeny of latex allergy is unknown, it seems prudent to avoid the use of products sterilised with ethylene oxide in patients who are at risk.

Those who pose a higher risk of latex allergy include workers constantly exposed to latex, people with a tendency to multiple allergic conditions and children with urological malformations who have been subjected to numerous manipulations since infancy, which was the case with our patient, who presented with a chronic secondary obstructive uropathy of the urethral valves that required a number of interventions during infancy.[2] The number of operations to which a child has been subjected is clearly related to the presence of specific IgE antibodies against latex.

The population in dialysis with no previous history of allergic reactions should not be considered as at risk, despite their frequent exposure to latex.[3] However, one quarter of dialysis patients may at least show a positive allergy test. These allergic reactions[4,5] can have significant implications in this population, amongst which include the possibility of repeated fistula thromboses, and the consequential difficulty in attaining a functioning VA or risk in achieving a successful kidney transplant.

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Interferon-alpha and its deleterious effects on kidney transplant: regarding one case

Nefrología 2009;29**(4)**:366-367.

Dear Editor:

The prevalence of chronic infection due to the hepatitis C virus in kidney transplant patients ranges from 5 to 40%.[1] Hepatitis C increases the morbi-mortality in both haemodialysis and kidney transplant patients. Hepatitis C should be treated prior to transplant, since post-transplant treatment with interferon-alpha[2] increases the risk of acute humoral rejection, particularly during the immediate post-transplant phase.

We present the case of a 55 year old male with a history of chronic terminal renal failure secondary to mesangial glomerulonephritis IgA, who began haemodialysis in October 1989, arterial hypertension and chronic infection due to hepatitis C virus (genotype 1). The patient received a cadaver kidney transplant in 1997 and began immunosuppressant therapy with OKT3, corticosteroids and cyclosporine. Development following the renal transplant was without incident, with stable renal function (urea 45mg/dL, Cr 0.8mg/dL), negative proteinuria, chronically elevated transaminases, positive RNA-HCV with no evident clinical signs of cirrhosis or advanced hepatopathy, and cyclosporine levels within the therapeutic range. The patient regularly attended digestive reviews, where it was decided to recommend treatment with interferon-alpha and ribavirin for twelve months. His renal function remained stable during this period. Three months following completion of the treatment, renal function deteriorated with 133mg/dL urea, 1.8mg/dL Cr and 1.8g/24h proteinuria, which worsened with later tests (urea 203mg/dL and Cr 2.7mg/dL). Ig and cryglobulin levels and an autoimmune study were all normal. It was decided to admit the patient in order to perform a diagnostic renal biopsy. The differential diagnosis included relapse of mesangial glomerulonephritis IgA, membranous or mesangiocapillary glomerulonephritis caused by the hepatitis C virus, chronic graft nephropathy or acute secondary rejection on treatment of interferon. The renal biopsy revealed acute humoral rejection with C4d+, and Ab anti-HLA levels were positive (22%) against the donor. It was decided to begin treatment with three 250mg 6-methylprednisolone tablets and conversion to tacrolimus. The response to treatment was good, with an improvement of renal function: 150mg/dL urea, 2mg/dL CR and a reasonable decrease in proteinuria (1.2g/24h).

The optimum treatment for hepatitis C in renal transplant patients today is controversial. Use of interferon is not advised, since it increases the chance of episodes of acute humoral rejection (15-64%) three to six months after beginning treatment,[3] and its use is indicated only in patients with fibrosing cholestatic hepatitis, where there is a significantly increased morbi-mortality. The incidence of acute humoral rejection is lower in patients with long-developing transplants, owing to immunological accommodation. The mechanism for inducing acute rejection is unclear, but it is thought that the drug increases the release of HLA antigens in the cellular surface and induces the release of cytokines, consequently stimulating the production of antibodies.[4] To minimise the risk of rejection, patients should have stable immunosuppression and should be closely monitored.[5]

The case in question concerns a patient with a normally functioning kidney transplant and stable renal function who received, twelve years after transplant, treatment with ribavirin and interferon-alpha, with a subsequent episode of humoral rejection. The importance of this case lies in the fact that the acute humoral rejection appeared during the late post-transplant period, and three months after having completed interferon treatment, which is uncommon.

In conclusion, it is of vital importance that nephrologists and digestive specialists know the indications of interferon in the transplant population, weighing up its potential benefits against the risk of rejection, and ensure patients on antiretroviral treatment are more closely monitored, even once this treatment has ended. Safer and more effective treatments are needed for treating renal transplant patients with infection from the hepatitis C virus.

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Acute ischaemia as a consequence of arteriovenous fistula massage in haemodialysis

Nefrología 2009;29**(4)**:367-368.

Dear Editor:
Massage of an arteriovenous fistula following thrombosis can restore blood flow and avoid the comorbidity associated with catheters. However, this practice carries significant risks. We present the case of a male with thrombosis of the humero-cephalic arteriovenous fistula who, following massage, showed an acute ischaemia of the left upper extremity due to embolisation of the humeral artery.

The patient is 53 years old with chronic renal failure secondary to IgA nephropathy, in substitutive renal treatment for the last 19 years: 10 in haemodialysis and 9 in renal transplant. At present, the patient is on a regime of four haemodialysis sessions per week. The patient has chronic obstructive pulmonary disease and obstructive sleep apnoea syndrome. A diagnosis of myocardial ischaemia (acute myocardial infarction six years previously) and advanced peripheral vasculopathy was made. The patient had undergone multiple failed vascular accesses: right and left radiocephalic thrombosed fistulae, right humerocephalic fistula and a thrombosed polytetrafluoroethylene (PTFE) humerobasilic graft. At present, dialysis is carried out by means of a left humerocephalic fistula, created two years ago.

The patient showed elevated haematocrit levels (44%). Phlebotomies were periodically performed to reduce the risk of thrombosis, accentuated by low arterial pressures and regular systolic pressures of around 85 or 90 mmHg.

He attended his haemodialysis session with low arterial pressure, and a short time following connection, the fistula thrombosed. On examination neither thrill nor bruit was found; the fistula had been functioning normally during the dialysis session one day previously. A fistula massage was performed, without recovering function. Immediately following the treatment the patient began complaining of pain in his hand and feeling cold, as well as cutaneous pallor. A colour Doppler sonography showed an absence of distal flow.

Both humeral and distal vessel thrombectomies were performed and a large amount of thrombotic material was extracted. The radial and humeral