interstitial pneumonitis, and non-necrotizing granulomas.[2,4] For this reason, a lung biopsy is not essential, and the diagnosis of this condition must fulfil the following criteria: presence of lung disease on imaging tests, exclusion of pulmonary infection or other lung disease, and clinical improvement after drug withdrawal, independent of the pulmonary biopsy.[4,5] All these criteria were fulfilled in the patient presented here, who showed significant imaging and clinical improvement after withdrawal of sirolimus.

Due to the increased use of sirolimus, radiologists interpreting imaging studies of transplant patients using this drug should be aware of the imaging features associated with this potentially treatable complication. Although some previous authors reported the use of CT,[2,3,5] to our knowledge, there are no reports illustrating and discussing the high-resolution CT findings of pulmonary toxicity due to sirolimus.

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B) BRIEF PAPERS ABOUT BASIC RESEARCH OR CLINICAL EXPERIENCES

Guillain-Barré syndrome in kidney transplant

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Dear Editor,

Guillain-Barré syndrome (GBS) is the most frequent cause of acute polyneuropathy in adults. It manifests with progressive symmetrical motor weakness in the limbs and areflexia. Its most severe complication is acute respiratory failure, which has a mortality rate of 15 to 30% if it requires mechanical ventilation.

It may be associated with respiratory or gastrointestinal infection (cytomegalovirus [CMV], *Campylobacter yeyuni*) in two thirds of all cases.[1,2] It has been described in bone marrow transplants, but it is very rare in solid organ transplants.[3] It has also been associated with neurotoxicity caused by calcineurin inhibitors.[4] We present two cases of GBS in patients undergoing kidney transplants.

The first case is that of a woman who underwent a kidney transplant at age 22 with chronic renal failure (CRF) secondary to neurogenic bladder. She received steroids, tacrolimus and mycophenolate mofetil in addition to a prophylactic treatment against CMV, hyperimmune gamma-globulin (recipient IgG negative/donor IgG positive). Evolution of renal function was excellent. Five weeks after the transplant, the patient became ill with CMV (severe leucopoenia, hypertransaminasaemia, gastritis, AgP65 positive). She received intravenous gancyclovir during 15 days and made satisfactory progress. She was readmitted three weeks later due to progressive, symmetrical weakness of the lower limbs, paraesthesia and dysphagia, and in the next few hours she developed acute respiratory failure requiring mechanical ventilation.

The second case is that of a 62 year old man with unexplained chronic renal failure who underwent renal transplantation. He was treated with steroids, tacrolimus and mycophenolate mofetil. His renal function progressed very well. However, 2.5 months after the transplant he was admitted for severe paralysis of the lower limbs which quickly progressed to tetraparesis. His AgP65 was checked and found positive, and he was treated with IV gancyclovir during 18 days. He did not require mechanical ventilation, but did need prolonged physical therapy.

In both patients, the cerebrospinal fluid (CSF) and the electromyography offered a diagnosis of GBS (demyelinising sensory-motor polyneuropathy; CSF at normal pressure, proteins elevated, without pleocytosis).

The first patient received four cycles of plasmapheresis and five doses of hyperimmune gamma-globulin (0.4g/kg/day) and the second, five doses of hyperimmune gamma-globulin (0.4g/kg/day). The first transplant patient underwent a full recovery and was extubated after seven days. After eight years of follow-up, she remains asymptomatic and has no neurological consequences. Six months

after the event, the second patient has experienced neurological improvements, but he continues to have lower limb paresis and is still in physical therapy. Tacrolimus levels were normal at all times in both cases. Tacrolimus was replaced with everolimus in the second patient, but there were no changes in his slow neurological progress.

GBS in solid organ transplants is a very rare condition, despite the fact that CMV infections are common, and it may put the patient's life at risk. Very few cases have been described in the literature, and most are in men.[5] In our patients, the CMV infection was the probable main trigger for the condition, considering its proximity in time. Early diagnosis is important so as to begin common neurological treatment for all forms of GBS, and treat the cause where applicable, as soon as possible.

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C) BRIEF CASE REPORTS

Effect of kidney transplant on ventricular dysfunction in a patient on haemodialysis

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Dear Editor,

We present the case of a 36 year old man with chronic renal failure and hypertension secondary to non-biopsied chronic glomerulonephritis. An arteriovenous fistula (AVF) was created, and six months later in August 2007, the patient had to begin replacement therapy by haemodialysis. The echocardiogram (ECG) taken on 22 August 2007 did not show findings with pathological significance (mild left ventricular hypertrophy, absence of valvular abnormalities, valvular competence and good systolic function with a stress test that was negative for ischaemia). Six months after starting dialysis he began to experience progressive dyspnoea, pressure on the chest and poor tolerance for dialysis, so he was changed to daily haemodialysis. He underwent additional testing including another ECG in February 2008, which showed a systolic dysfunction of the left ventricle (visual ejection fraction [EF] 40%, telediastolic volume [TDV] 157mm, telesystolic volume [TSV] 105mm, a slight dilation of the left atrium with mild to moderate mitral regurgitation and severe pulmonary hypertension. New ECGs were repeated in August and September 2008, and we observed a decrease of up to 25% in systolic function, loss of ventricular configuration with end-diastolic volume increasing to 185ml, severe functional mitral regurgitation with increased atrial dilation and increase in the degree of pulmonary hypertension (figure 1). In November 2008, the patient underwent a kidney transplant with a good recovery, and creatinine level at the time of discharge was 1.5mg/dl. It was decided to close the AVF during the postoperative period. In the months following the transplant the patient remained asymptomatic and the ECG parameters measured in July 2009 are better: mitral regurgitation has disappeared completely, EDV is 129ml and the EF is 45% (figure 1).

Congestive heart failure is 12 to 36 times more frequent in patients on dialysis than in the general population. Proper treatment is a topic for debate. It is not clear whether or not the general recommendations for treating heart failure in the general population are equally effective and safe in the population on dialysis. Furthermore, this patient group is at high surgical risk. The lack of evidence on the effect of transplants on perioperative morbidity and mortality means that these patients are often not evaluated for transplant, and when they are, it is often not clear whether or not they should be included on the waiting list. Wali et al.[1] evaluated the impact of transplant on 103 patients with chronic heart failure and an EF of less than 40%, and they saw a clear improvement 12 months after the transplant, even in patients whose heart function was affected the most. Kidney transplant