after 12 months. Glycaemia and HbA_{1C} do not seem to change in accordance with the glucose load. There is a good correlation between glucose and HbA_{1C}. High transporters have higher glucose values after one month on PD (*P*=.039), but not of $HbA_{1C.}$

During the first years in which PD has been reported, and on the basis of the glucose load that was contributed to obtain sufficient ultrafiltration, it was considered to be a dialysis technique with a potential diabetogenic effect. It is possible that in these first few years, due to a lack of knowledge about the deleterious effect that glucose contribution has on the peritoneum with the development of GDP[2], the relatively common use of very hypertonic solutions, which furthermore did not use bicarbonate as a buffer, may have caused some cases of diabetes.In the last decade since the introduction of solutions in dual chambers with a mixture of lactate and bicarbonate or bicarbonate alone, with which the formation of GDP is minimal and use of 3.86%-4.25% glucose PD dialysate is practically nil, the induction of diabetes and even the development of moderate hyperglycaemia, as our study shows, have become anecdotal. The increase in lipids reported in some articles[6] is not relevant in our study in terms of its maintenance over time and it has not been confirmed by other authors[7].

In conclusion, our non-diabetic PD patients treated with glucose solutions did not show changes in their glucose levels throughout the 36 months on dialysis. HbA_{1C} was unchanged after a year on the technique. The potential development of diabetes in PD was not confirmed by our results.

Conflicts of interest

The authors declare that they have no conflicts of interest related to the contents of this article.

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Results 5 years after living donor renal transplantation without calcineurin inhibitors

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To the Editor,

Calcineurin inhibitor-based (CNI) immunosuppression regimens have improved the outcomes of renal transplantation. Unfortunately, the use of CNI has been associated with interstitial fibrosis and tubular atrophy, affecting graft function and graft survival[1]. In order to avoid exposure to CNI, agents such as sirolimus (SRL) have emerged as new therapeutic options. Therapeutic strategies with SRL include the minimisation, suspension, elimination and total absence of CNI[2].

Experiences with CNI-free SRL/mycophenolate mofetil (MMF)/ST immunosuppression have not obtained sufficient acute rejection (AR) prophylaxis[3]. The introduction of induction therapy improved AR rates and short-term efficacy (1-3 years) with contradictory results[4-7]. We previously reported excellent and satisfactory results after 1 and 3 years without CNI[8,9] and we now present an observational and retrospective study of efficacy and safety after 5 years of the SRL/MMF/ST regimen compared with cyclosporine (CS)/MMT/ST and selective induction with basiliximab in 41 patients enrolled between May 2004 and January 2005.

The study design has previously been reported in detail[8]. In this report, the results were analysed in two populations: the intention-to-treat (ITT) population, which included all patients with a functioning graft, and the population on treatment (OT), which included patients who were maintained on the same original study immunosuppression regimen.

The demographic data of patients are displayed in Table 1. Five-year patient survival was 90% in the SRL group and 80.9% in the CS group (*p*=ns). The causes of death in the SRL group were cardiovascular (n=1) and infectious (n=1), which was similar to the CS group: cardiovascular (n=2), infectious (n=2) and gastrointestinal bleeding (n=1). Five-year graft survival was 80% for SRL and 76.1% for CS (*p*=ns). The causes of graft loss in the SRL group were: graft thrombosis (n=1), *de novo glomerulonephritis* (n=1), urological complications (n=1) and a lack of adherence to treatment (n=1). In the CS group they were: graft thrombosis (n=1), *de novo glomerulonephritis* (n=1), lupus (n=1), chronic kidney disease (n=1) and death with a functioning graft (n=1).

Eight patients (40%) from the SRL group and 3 (14%) from the CS group received basiliximab induction. After 5 years, there was a decrease in the dose of CS (133±29.9mg/day, range 120-200) and of SRL (1.75±0.66mg/day, range 1-3) compared to 12 months after transplantation (205.7±66mg/day and 3.2±1.7mg/day CS and SRL, respectively). The mean dose of MMF was higher in the CS group (1218.75±363g/day, range 500-2000), compared with the SRL group (1093.9±417g/day, range 500-2000) (*p*=.3). All patients in the study continued to take 5mg/day of oral prednisone. Four patients (25%) in the CS group (*p*=.039) with a functioning graft changed their regimen to SRL due to interstitial fibrosis and tubular atrophy confirmed by biopsy. We maintained all patients in the SRL group with a functioning graft on the SRL/MMF/ST regimen. After one year of follow-up, 2 patients in the SRL group (11.1%) and 3 in the CS group (17.7%) had episodes of AR (*p*=ns).

Graft function calculated by the glomerular filtration rate estimated using the MDRD (Modification of Diet in Renal Disease) formula[10] and serum creatinine is displayed in Table 2. We did not find a statistically significant difference between the two groups, independently of whether they were an ITT population or a population OT. Patients in the SRL group had a higher elimination of proteins in 24h urine (*p*=.039) than patients in the CS group in the ITT population. Serum haemoglobin was similar in both cases. Cholesterol and triglycerides were significantly higher in the SRL group (Table 2).

There were a total of 81 adverse effect events, which were mostly infectious (14 in the SRL group and 16 in the CS group). There was a similar incidence in new onset diabetes after transplantation (NODAT) (10% in the SRL group versus 9.5% in the CS group). No patient developed a malignancy during follow-up. Six patients (37.5%) in the SRL group and 31.3% (n=5) in the CS group were taking angiotensin-converting-enzyme inhibitors and/or angiotensin receptor blockers after 5 years (*p*=.7). Similarly, more patients in the SRL group were taking lipid-lowering drugs than in the CS group (n=7, 43.8%, versus n=6, 37.5%) (*p*=.2).

In summary, despite the fact that our results need to be carefully reviewed due to certain limitations, such as the sample size, retrospective recording and a population of low immunological risk, we concluded that living donor transplantation patients with selective induction on the SRL/MMF/ST regimen have similar graft survival and function 5 years after transplantation to those on the CSA/MMF/ST regimen.

Table 1. Clinical and demographic parameters

	Group A Sirolimus	Group B Cyclosporine	*P* value
Patients (n)	20	21	ns
Recipient's age (years), mean SD (range)	29,6 7,6 (18-40)	31,2 9,21 (18-52)	ns
Sex (male:female)	12:8	12:9	ns
BSA, mean SD (range)	1,73 0,24 (1,31-2,19)	1,63 0,1 (1,43-1,97)	ns
Dialysis time (months), mean (range)	24,25 13,7 (2-62)	26 12,6 (3-60)	ns
HLA match, mean SD (range)	2,7 1 (0-5)	2,9 1,1 (0-4)	ns
Donor's age (years), mean (range)	37,8 (21-56)	37,9 (27-59)	ns
CMV serology			
D+/R-	2	2	
D+/R+	14	16	
D-/R-	2	2	
D-/R+	2	1	

CMV: cytomegalovirus, ns: not significant, SD: standard deviation.

Table 2. Graft function based on the analysis of patients on treatment and those who we intended to treat

	Group A (SRL)	Group B (CSA)	*P* value
MDRD eGFR (ml/min/1,73 m^2)			
ITT population	n = 16	n = 16	
Mean±SD (range)	53.8±19 (20-90.9)	54.7±18.7 (29-83.7)	0.88 (ns)
Population OT	n = 16	n = 12	
Mean±SD (range)	53.8±19 (20-90.9)	54.1±19.1 (29-83.7)	0.91 (ns)
Serum creatinine (mg/dl)			
ITT population	n = 16	n = 16	
Mean±SD (range)	1.6±0.6 (1.0-3.7)	1.49±0.4 (1.0-2.2)	0.54 (ns)
Population OT	n = 16	n = 12	
Mean±SD (range)	1.6±0.6 (1.0-3.7)	1.47±0.5 (1.0-2.2)	0.67 (ns)
Protein in 24h urine (mg/day)			
ITT population	n = 16	n = 16	
Mean±SD (range)	293.6±280 (50-814)	110.6±192 (0-620)	**0.039 (s)**
Population OT	n = 16	n = 12	
Mean±SD (range)	293.6±280 (50-814)	136.7±205 (0-620)	0.09 (ns)
Haemoglobin (g/dl)			
ITT population	n = 16	n = 16	
Mean±SD	13.1±2.21	12.2±1.68	0.24 (ns)
Population OT	n = 16	n = 12	
Mean±SD	13.1±2.21	12.6±1.83	0.57 (ns)
Total cholesterol (mg/dl)			
ITT population	n = 16	n = 16	
Mean±SD	221.3±43.4	192.5±34.3	**0.046 (s)**
Population OT	n = 16	n = 12	
Mean±SD	221.3±43.4	190.4±41.6	0.063 (ns)
Triglycerides in blood (mg/dl)			
ITT population	n = 16	n = 16	
Mean±SD	208.4±101.8	149.2±36.1	**0.041 (s)**
Population OT	n = 16	n = 12	
Mean±SD	208.4±101.8	147±32.6	**0.036 (s)**

CS: cyclosporine, eGFR: estimated glomerular filtration rate, ITT: intention-to-treat population, MDRD: modification of diet in renal disease, ns: not significant, OT: on treatment, SD: standard deviation, SRL: sirolimus.

Conflict of interest

The authors declare the following conflicts of interest:

– Dr. Gustavo Martínez Mier receives lecture fees from Pfizer, Roche and Novartis and consultancy fees from Novartis and Sanofi.

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C) BRIEF CASE REPORT

Hepatitis C virus infection, interferon α and lupus; a curious combination

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To the Editor,
Drug-induced lupus is a syndrome that shares clinical and analytical characteristics with idiopathic systemic lupus erythematosus and which appears after exposure to certain drugs that induce autoantibody formation.

In 1945, Hoffman described the first case of drug-induced lupus, which involved the antibiotic sulfadiazine as the agent responsible for the condition. Eight years later, in 1953, Morrow et al. published a new case relating to the use of hydralazine[1]. Since then, the list of associated drugs has continued to increase and in recent years, biological therapies, such as tumour necrosis factors (TNF) and interferons (IFN), have joined with classic agents, such as procainamide, the aforementioned hydralazine, isoniazid or minocylcine[2,3,4].

The mechanism causing this condition has not been fully defined; immunogenetic (certain HLA alleles) and pharmacogenetic (slow acetylator phenotype) factors appear to play an important role in its aethiopathogeny[1,5].

In terms of clinical presentation, the most common symptoms are fever, general malaise, muscle pain, joints pain, arthritis, rash and serositis. Unlike idiopathic lupus, kidney, haematologic and nervous system disorders are uncommon[6]. Antihistone antibodies are typical laboratory findings. Hypocomplementaemia and anti-double-stranded DNA, characteristics of idiopathic lupus, tend to be absent, although the latter can test positive in cases of anti-TNF- or IFN-induced lupus (Table 2).

The interval of time between starting the drug and the condition appearing is highly variable, being between 2 weeks and 7 years in the case of IFN-α; a case developing two months after the drug's suspension has been described[7].

This condition's prognosis is favourable, such that discontinuation of the responsible drug is followed by recovery in the majority of cases, in a time frame that can stretch from weeks to months. Until then, non-steroidal anti-inflammatory drugs (NSAID), hydroxychloroquine and low-dose systemic corticosteroids can be used temporarily to control symptoms.

CASE

We present a 51-year-old male, with chronic kidney failure secondary to IgA glomerulonephritis, on a periodic haemodialysis programme, hypertensive, an ex-user of cocaine by inhalation and with chronic hepatitis C virus (HCV) disease, for which reason he was treated with ribavirin and pegylated IFN-α (180μg per week) for 49 weeks, obtaining a sustained viral response. Two weeks after finishing this treatment, he sought consultation due to asthenia and generalised joint pain of 10-15 days evolution, also experiencing in the last 48 hours 38 ºC fever and increased right hip pain. In the physical examination he presented pain on moving the aforementioned joint, with neither functional weak-