ual diuresis remained at 800ml/day throughout most of the pregnancy.

In puerperium, the patient developed another episode of heart failure and hypertensive crisis in the context of hydrosaline overload, which was resolved by decreasing dry weight (upon discharge it was 10kg less than at the start of the pregnancy). Coinciding with these findings, we also detected decreased values for haemoglobin, thrombocytopenia, elevated lactate dehydrogenase (LDH), and increased transaminase levels in laboratory test results. A peripheral blood smear was normal, ruling out haemolysis. A direct Coombs test was also negative.

Following birth, we observed a complete recovery in clinical and laboratory parameters for the mother, but she still required 6 hypotensive drugs in order to control blood pressure.

Monitoring pregnancy in patients on dialysis requires strict multi-disciplinary control, and we believe that individual experiences and those reported in reviews of case reports are very important for reaching a consensus or shared criterion for managing and treating these patients, so as to achieve a greater rate of success and maternal/foetal survival.[7,8]

Conflicts of interest

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

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Tacrolimus associated to posterior reversible atypical encephalopathy syndrome and brain haemorrhage in renal transplant recipient

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To the Editor:

Posterior reversible encephalopathy syndrome (PRES) can occur in recipients of solid organ transplants associated with calcineurin inhibitors. The incidence of this syndrome in kidney transplant recipients is low, approximately 0.34%,[1,2] and should be suspected when neurological symptoms arise in association with characteristic lesions found in cerebral magnetic resonance (CMR) images, which revert after reducing or suspending the dose of tacrolimus. In order to resolve this condition and avoid neurological sequelae, an early diagnosis and suspension of the causal calcineurin inhibitor is needed.[3]

Here we present the case of a kidney transplant recipient who developed atypical PRES associated with tacrolimus.

Ours was a 32-year old male patient with a personal history of arterial hypertension and stage 5 chronic kidney disease, who had received a kidney transplant 2 years earlier with the prescription of sirolimus, but who was switched to tacrolimus due to gastrointestinal intolerance to the first drug, and had been taking tacrolimus for the past 5 months.

The patient sought treatment at our hospital of IV level of complexity due to a first-onset generalised tonic-clonic seizure of 60 seconds in duration. A physical examination showed that the patient was without fever, had a blood pressure of 187/133mm Hg, and was sleepy in a postictal state, with no other relevant findings. A laboratory analysis revealed mild leukocytosis, hyperlactaemic metabolic acidosis, creatinine: 5.43mg/dl (baseline: 3.0mg/dl), and normal electrolyte levels. We performed a simple cerebral computed axial tomography (CAT), which revealed several hyperdense lesions, the largest of which was in the right frontal lobe; we then administered a CMR (Figure 1), which revealed several lesions: frontal lobe with mass effect and cortical/subcortical without restrictions in the ADC sequence. We performed several analyses to determine the extent of the lesions, including: cerebrospinal fluid analysis, haemocultures and urine cultures (negative), and a stereotactic biopsy of the right frontal lobe lesion that revealed reactive gliosis and small foci of interstitial bleeding. Given these findings, we were

able to rule out other possible diagnoses and settled on the probable diagnosis of neurotoxicity from tacrolimus. We decided to suspend the medication and observe the patient's evolution, which was very favourable from a neurological standpoint. The patient was discharged under a renal replacement therapy regimen, since basal GFR were not recovered, and no immunosuppressant medications were prescribed. Three months later, a follow-up simple cerebral tomography (Figure 2) showed that the supratentorial lesions had healed, with only a small encephalomalacic area in the right frontal lobe at the site of the haematoma.

DISCUSSION

Calcineurin inhibitors have been described as potent immunosuppressants that are considered to be quite effective in the prevention of acute transplant rejection. However, these drugs are associated with several adverse side effects; in the specific case of tacrolimus (also known as FK-506 or Fujimycin), reports have described increased incidence rates of diabetes and nephrotoxicity, among others, but nephrotoxicity in particular, with even greater incidence rates than those produced by cyclosporine.[4] The symptoms include headache, trembling, paraesthesia, visual impairments, convulsions, and coma.[5] One form of presentation of this neurotoxicity is PRES, a clinical/radiological entity that is uncommon and was first described only recently (1996).[6] A CMR has been considered as the gold standard[7] for diagnosing this condition; we typically see an increase in signal intensity for T2-weighted images in both posterior regions, with compromised cortical tissue due to the lack of regulation of posterior circulation.[6] However, these signals were then described in the frontal region due to vasogenic oedema of the subcortical white matter.[6,8] The FLAIR sequence (fluid-attenuated inversion recover) increases the capacity for detecting minute lesions. Diffusion weighted imaging sequences (DWI) and the apparent diffusion coefficient (ADC) are useful for differentiating vasogenic oedema with mild restriction from oedema with no restriction, which indicates reversibility.

Follow-up imaging controls are indispensable for supporting the diagnosis.[1,6] There is no specific timeline established for these controls; some authors recommend 4 weeks after resolution of clinical signs. These controls can take place in the form of CMR or cerebral CAT scans.[6]

In our patient, we observed characteristics that can be considered atypical for this syndrome, such as increased involvement of the right frontal lobe, with cortical lesions and intra-lesion bleeding that surpassed the PRES lesions, making these typically reversible lesions progress towards irreversible damage and encephalomalacia, as observed in the follow-up cerebral imaging tests from this patient (Figure 2).

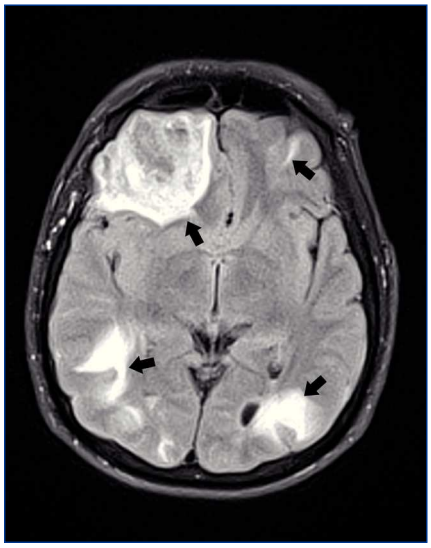

Figure 1. Cerebral magnetic resonance, FLAIR sequence. Heterogeneous hyper-intense right frontal lesion with a mass effect as evidenced by haemorrhage. Cortical/subcortical hyper-intense areas in the left frontal and bilateral occipital lobes.

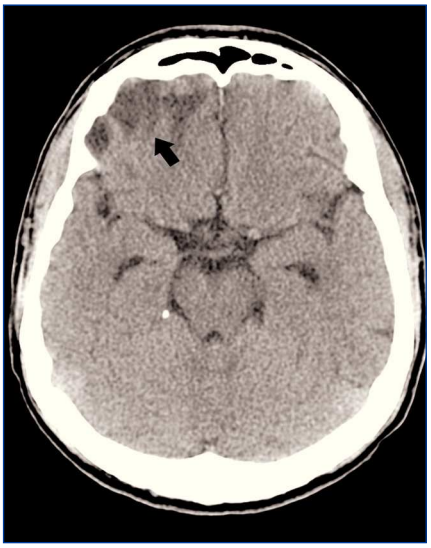

Figure 2. Simple cerebral computed axial tomography, control. Hypodense area can be observed in relation to a malacic area of the frontal lobe at the site of the prior haemorrhagic lesion. Resolution of all other supra and infratentorial lesions.

Conflicts of interest

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

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Peripheral nervous system involvement in a haemodialysis patient treated with pegylated interferon

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To the Editor:

The prevalence of hepatitis C virus infection among patients on haemodialysis is estimated at 3%-23% in developed countries. Some of the adverse effects described in treating patients on haemodialysis with interferon as a monotherapy include[1]: flu-like symptoms, anaemia, immunological graft intolerance, depression, leukopoenia, confusion, diarrhoea, bone pain, thyroid alterations, thrombocytopenia, and convulsions. To a lesser degree, there have also been descriptions in the medical literature of autoimmune diseases and neurological alterations in relation to this type of treatment in patients on haemodialysis. Pegylated interferon improves drug absorption and extends the half-life of this medication.

Table 1. Laboratory analysis results.

	Pre-treatment	Month 3. Suspension	Month 3-4. Evolution			Month 5
Hb (g/dl)	11.8	9.6	8.8	7.2	9.9	10.2
Leukocytes	5280	3380	12430	10910	7170	6750
Platelets	111000	51000	92000	151000	155000	121000
AST (UI/l)	16	21	33		31	22
ALT (UI/l)	8	11	15		9	6
GGT (UI/l)	18					29
CPK (UI/l)	61			27	68	
Albumin (g/dl)	3.5					2.5
CRP (mg/dl)	1.3		20.5		4.6	2.7
TSH (µUI/ml)				9.62		7.42

Results from laboratory analyses arranged by treatment with pegylated interferon. Before treatment, at the moment treatment was suspended (month 3), after suspension of treatment (months 3-4), and 5 months after start of treatment (month 5).
ALT: alanine-aminotransferase; AST: aspartate-transaminase; CPK: creatinine phosphokinase; GGT: gamma-glutamyl-transferase; CRP: C-reactive protein; TSH: thyrotropin-stimulating hormone.

Here we describe the case of a 47-year old male on haemodialysis with no known allergies, a history of smoking and social drinking, and a previous addiction to parenteral drugs. The patient suffered from stage 5 chronic kidney disease of an unknown origin. The patient had received a transplant but had to return to haemodialysis after 8 years. The patient also had arterial hypertension, positive serology for hepatitis C virus (HCV), positive CRP (genotype 3a), positive cryoglobulins, chronic gastritis, bronchiectasis, and had problems with his vascular access in the form of a permanent right jugular catheter.

The patient had no alterations in transaminase levels or clotting factors, and in the absence of ultrasound indications of portal hypertension, we started him on treatment with alpha-2-a pegylated interferon as a monotherapy, considering the patient to be a candidate for a second kidney transplant. During the first three months of treatment, the patient suffered only mild adverse side ef-

Table 2. Autoimmune state following suspension of interferon.

	Results
Thyroid peroxidase antibodies	Negative
Thyroglobulin antibodies	Negative
Anti-TSH receptor antibodies	Negative
ANA	Negative
Anti dsDNA	Negative
Anti-Sm	Negative
Anti-Ro	Negative
Anti-La	Negative
Anti-RNP-70	Negative
Rheumatoid factor	49IU/ml (0-14)
Protein electrophoresis	Polyclonal increase in gamma fraction
C3 and C4	Normal

ANA: anti-nuclear antibodies; TSH: thyrotropin-stimulating hormone.