

vs post-HD 23,500 ng/ml). After the second session, the patient resumed diuresis at a polyuria rate, recovering the kidney function he had prior to rhabdomyolysis.

We report this case to draw attention to the early start of replacement therapy, with the use of dialysers with an intermediate cut-off (40 kD) probably being useful in that, by increasing myoglobin clearance and decreasing serum levels, they can contribute to an earlier recovery from AKF.

#### REFERENCES

1. Huerta-Alardín AL, Varon J, Marik PE. Bench-to-bedside review: rhabdomyolysis-an overview for clinicians. *Crit Care*. 2005;9:158–69.
2. Vanholder R, Sever MS, Ereğ E, Lameire N. Rhabdomyolysis. *J Am Soc Nephrol*. 2000;11:1553–61.
3. Khan FY. Rhabdomyolysis: a review of the literature. *Neth J Med*. 2009;67:272–83.
4. Ronco C, Bellomo R, Kellum JA. Critical care nephrology. In: *Myoglobin as a toxin*. 2nd ed. Philadelphia, PA: Saunders Elsevier; 2009. p. 1103–9.
5. Zager RA, Johnson AC, Becker K. Plasma and urinary heme oxygenase-1 in AKI. *J Am Soc Nephrol*. 2012;23:1048–57.
6. Torres PA, Helmstetter JA, Kaye AM, Kaye AD. Rhabdomyolysis: Pathogenesis, diagnosis, and treatment. *Ochsner J*. 2015;15:58–69.

7. Bellomo R, Kellum JA, Ronco C. Acute kidney injury. *Lancet*. 2012;380:756–66.
8. Petejova N, Martinek A. Acute kidney injury due to rhabdomyolysis and renal replacement therapy: a critical review. *Crit Care*. 2014;18:224.
9. Fabbri P, Rona R, Migliari M, Viganò M, Pesenti A. Myoglobin removal of small-protein leakage membrane (EMIC2) in patients in the ICU: a case series. *Crit Care*. 2014;18 Suppl. 1:P398, <http://dx.doi.org/10.1186/cc13588>.

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## Guillain-Barre syndrome secondary to tacrolimus in a patient with corticoreistant nephrotic syndrome secondary to focal and segmental glomerulonephritis and a coexisting IgA nephropathy<sup>☆</sup>

### Síndrome Guillain-Barré secundario a tacrolimus en paciente con síndrome nefrótico corticorresistente secundario a glomerulonefritis focal y segmentaria y nefropatía IgA

Dear Editor,

Guillain-Barre syndrome (GBS) manifests itself as a progressive and symmetrical muscular weakness in lower limbs and arreflexia. Occasionally, patients require invasive ventilation and with a 15–30% mortality rate.<sup>1</sup> The association between respiratory and gastrointestinal infections is well known and occurs in two-thirds of cases.<sup>2,3</sup> In a solid organ transplant it is a rare complication, however it is slightly more frequent in bone marrow transplantation. However, Guillain-Barre syndrome is an exceptionally rare complication in a nephrotic syndrome patient, treated with tacrolimus and whose renal

biopsies reveal the separate entities, a focal and segmental glomerulonephritis and a IgA nephropathy. We present the case of a 65-year-old patient with multiple comorbidity, nephrotic syndrome secondary to focal and segmental glomerulonephritis and an IgA nephropathy treated with tacrolimus and drifting into in a Guillain-Barre syndrome.

This is a 65-year-old patient who was referred to a nephrology department from primary care in 2014 because of proteinuria and uncontrolled blood pressure. Among the personal history, it can be highlighted in [Table 1](#). He was firstly seen in October 2014 at nephrology consultation. He was in a good general condition. Analysis revealed GFR > 60 (MDRD) Cr

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**Table 1 – Personal history.**

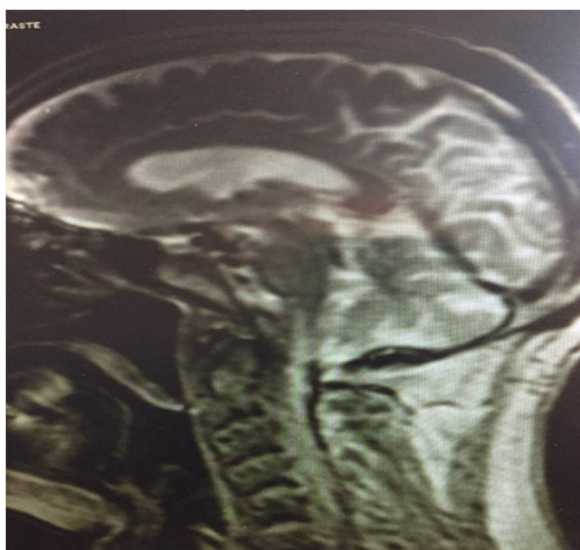
No detected allergy Ex-smoker, with 1 package per day until 1987.	Right leg cellulitis in 2009 Recurrence of cellulitis in 2010, assessed by vascular surgery. During the assessment a presence of haematuria and proteinuria was detected Known hypertension since 2002 and treated with ACEi by GP, and presenting urticaria-angioedema in 2009.
Alcoholic habits until 1987	Asymptomatic cholelithiasis in 2009.
Miner from 1977 until retirement in 2003. Herniated disc surgery in 1987.	Acute renal failure in 2010 in relation to right extremity cellulitis.
Left cryptorchidia studied by urology with a negative extension study.	
Eczema 1999 in probable relation with non-affiliated psoriasis. Right varicose veins; Saphenectomy with ligation in 2000.	

1.1 mg/dl Hb 11 mg/dl, Na 136 mmol/l K 4.3 mmol/l, proteinogram with inflammatory profile, good diuresis, unchanged immunological profile, ACRO 3600 mg/g, proteinuria of 4 g/24 h, no hypoproteinaemia, Alb 3 mg/dl and TP 6 mg/dl. There was no anemia. Immunoglobulins were not altered.

With a normal abdominal ultrasound, a renal biopsy was performed with a diagnosis of focal and segmental glomerulonephritis. Treatment with corticosteroids was started with a 1 mg/kg/day on September 2015. The following month the patient had to be hospitalized in internal medicine presenting an Addison crisis in relation to the suppression of the steroids by the patient. The patient remained hospitalized for a few days, the corticotherapy was reintroduced and he was discharged. At January 2016 corticoresistance was verified, a proteinuria of 5.5 g in 24 h was observed. Treatment with cyclosporine was initiated with a poor response and multiple side effects. Therefore, it was suspended after three months of use and a change to mycophenolate mofetil 1000 mg/12 h was made. From October to November the patient was hospitalized due to anasarca and nephrotic syndrome. A second renal biopsy was performed and tacrolimus was initiated adjusted to the levels of (range 8–12 ng/ml). The subsequent biopsy reveals IgA M1 E1 S1 T1 nephropathy in the Oxford classification. The patient appears once again at the emergency room in January 2017, presenting general deterioration with important loss of

tone and mobility at the lower limbs, that has been progressive. Subsequently the patient manifested that it started to occur mildly several weeks after the onset of tacrolimus. Given the suspicion, of a possible Demyelinating neuropathy by tacrolimus or a GBS, the consultation to neurology was made and the drug was progressively withdrawn. An MRI of the skull was performed ([Image 1](#)), a lumbar puncture reflected proteins 76 mg/dl and glucose of 68 mg/dl. Secondary causes of GBS were also discarded. The neurophysiological study was done with the following result: signs of symmetrical generalized peripheral neuropathy (sensory polyneuropathy) of probable axonal origin and moderate-severe intensity, with greater involvement of the lower extremities. However, the symmetrical progression of the affection makes more suspicion a GBS. The patient was seen by psychiatry and rehabilitation, with current monitoring of the two specialties. He continued his treatment with a lower dose prednisone. At discharge his Glomerular Filtration was over 60 ml/min, a Cr of 1.2 mg/dl and a proteinuria of 6 g/24 h. Overall condition improved considerably from admission.

GBS is an uncommon disease with a worldwide incidence of 1–2 cases per 100,000/year,<sup>4</sup> while the incidence of GBS in patients receiving tacrolimus secondary to the nephrotic syndrome is not known. This syndrome, often described as an immunological disorder, has been thought to be rare among immunosuppressed patients, since immunosuppression acts as a protection. The pathophysiology of tacrolimus toxicity is not well studied, although most cases are described in association with high systemic levels of the drug. Although nerve conduction studies in patients with tacrolimus polyneuropathy may also show evidence of demyelinating changes,<sup>5</sup> the symmetrical ascending presentation and the absence of a history of elevated tacrolimus levels goes toward the diagnosis of GBS over tacrolimus polyneuropathy.<sup>6</sup> The treatment of GBS secondary to tacrolimus in patients with nephrotic syndrome is not known, except the withdrawal of the medication. Even this case is exceptionally rare, it is necessary to take into account the presence of this pathology when the medication is used, an early and timely diagnosis, would avoid greater sequelae in all the patients that might be affected.



**Image 1 – A brain RMN of a patient affected with GBS secondary to tacrolimus.**

#### REFERENCES

- Hardy TA, Blum S, McCombe PA, Reddel SW. Guillain-Barre syndrome: modern theories of etiology. *Curr Allergy Asthma Rep.* 2011;11:197-204.

2. Drachman DA, Paterson PY, Berlin B, Roguska J. Immunosuppression and the Guillain-Barré syndrome. *Neurology*. 1970;200:390.
3. Wen PY, Alyea EP, Simon D, Herbst RS, Soiffer RJ, Antin JH. Guillain-Barre syndrome following allogeneic bone marrow transplantation. *Neurology*. 1997;49:1711–4.
4. Ponticelli C, Campise MR. Neurological complications in kidney transplant recipients. *J Nephrol*. 2005;18:521–8.
5. Dionne A, Nicolle MW, Hahn AF. Clinical and electrophysiological parameters distinguishing acute-onset chronic inflammatory demyelinating polyneuropathy from acute inflammatory demyelinating polyneuropathy. *Muscle Nerve*. 2010;41:202–7.
6. Labate A, Morelli M, Palamara G, Pirritano D, Quattrone A. Tacrolimus-induced polyneuropathy after heart transplantation. *Clin Neuropharmacol*. 2010;33:161–2.

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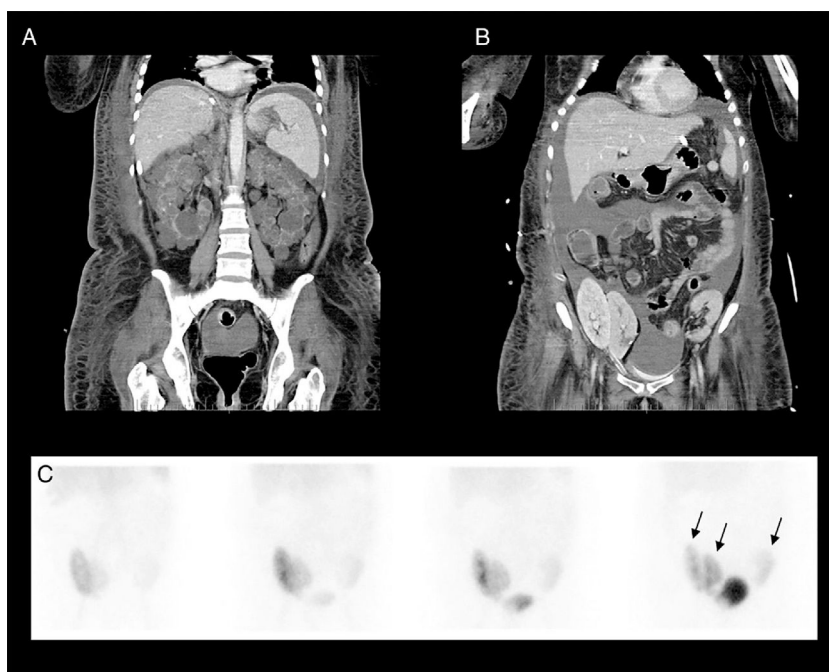
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## Triple functioning renal allograft after repeated liver–kidney transplantation due to liver failure

### Funcionamiento de un triple trasplante renal alogénico tras repetir un trasplante de hígado-riñón por fallo hepático

A 56-year-old woman who underwent 10 years earlier simultaneous liver–kidney transplantation due to polycystic

disease (Fig. 1A) showed progressive transaminase elevation secondary to hepatotoxicity due to venlafaxine. The



**Fig. 1** – Baseline CT coronal views show polycystic kidneys before the first liver–kidney transplantation (A). Follow-up CT locates the three transplanted kidneys in both sides of the pelvis (B). A renogram study using <sup>99m</sup>Tc-MAG<sub>3</sub> was performed identifying different degrees of radiotracer uptake in all three transplanted kidneys (C).