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Acute renal failure due to rhabdomyolysis. Renal replacement therapy with intermediate cut-off membranes (EMIC2)☆

Fracaso renal agudo por rabdomiólisis. Tratamiento con hemodiálisis y membranas de cut-off intermedio (EMIC2)

Dear Editor,

Rhabdomyolysis is a clinical syndrome caused by damaged skeletal muscle tissue and the release of its intracellular components, including myoglobin, lactate dehydrogenase, creatine kinase (CK), and electrolytes into the blood stream and the interstitial space. Its symptoms vary from a nearly asymptomatic condition, with myalgia and elevated CK levels, to an extremely serious condition with marked CK elevations, severe electrolyte disorders d, acute kidney failure (AKF), and disseminated intravascular coagulation (DIC).[1] The aetiology of the syndrome can be highly varied, with both hereditary (hereditary myopathies), and acquired factors (extreme physical activity, exposure to extreme temperatures, vascular ischaemia, trauma, drug use, toxins, sepsis, electrocution, etc.) having been reported.[2]

The most significant complication of rhabdomyolysis is AKF that occurs in up to 33% of patients present.[3] The mechanism responsible for AKF lies in the release of myoglobin. Three myoglobin-mediated nephrotoxic mechanisms have been described. Renal vasoconstriction, the formation of intratubular casts, and direct damage to tubular cells.[4,5] The best treatment for rhabdomyolysis-associated AKF is prevention. Increasing volume with crystalloid infusions to maintain good renal perfusion and high urinary flow, along with initial alkalinisation, are the bases for prevention.[6] In the event that the aforementioned measures fail, it will be necessary to start renal replacement therapy, which is not indicated based on the levels of myoglobin or CK, but based on the presence of life-threatening conditions such as hyperkalaemia, hypercalcaemia, anuria or volume overload.[7] Once decided to conduct renal replacement therapy, whether through intermittent haemodialysis or continuous techniques, we must consider that the toxin responsible for AKF, myoglobin, has a Pm of 17 kD, and is poorly removed by high-flux dialysers.[8,9] We present a case of rhabdomyolysis with AKF in a kidney transplant patient who was treated with intermittent haemodialysis with an EMIC2 dialyser (cut-off 40 kD).

This is a 45-year-old patient with chronic kidney disease of unknown aetiology on peritoneal dialysis since 2009. He received the first kidney transplant from a deceased donor in 2010, with early vein thrombosis. A thrombophilia test showed a state of hypercoagulability with hyperhomocysteinaemia and elevated factor VIII. The second kidney transplant from a deceased donor was performed in 2013, with an indefinite systemic anticoagulation prescription with sintrom®. He had an episode of late acute rejection in January 2016 which was treated with steroids. Later on, he developed nephropathy due to BK virus with stage 4 CKD (Cr 4.1 mg/dl). In October 2016, he had an episode of deep vein thrombosis in the left lower limb (LLL). Previously, anticoagulant therapy was suspended due to lower gastrointestinal bleeding secondary to a colon polyp. Treatment with sintrom was resumed and in December 2016 he was readmitted for acute LLL pain and sudden-onset oedema up to the root end of the limb, again observing deep vein thrombosis in the femoral popliteal area. At that time, the patient was not adequately anticoagulated (INR 1.4), and it was decided to treat him with sodium heparin. Poor evolution with significant oedema of the LLL, frailty, and signs of poor distal infusion, with the patient developing AKF in addition to CKD (Cr 6.6 mg/dl) with dark urine and oliguria. Ultrasound ruled out vascular involvement of the kidney graft and confirmed the existence of rhabdomyolysis (CK 44,915 mU/ml, lactate dehydrogenase 3100 U/l, GOT 392, GPT 113) and severe dyselectrolytaemia (K 6.6 mEq/l, bicarbonate 16 mEq/l). Despite the vigorous crystalloid infusion, the patient's anuria continued, with it becoming necessary to replace kidney function with emergency haemodialysis. Two 6-h dialysis sessions were completed with a 1.8 m^2 EMIC2 dialyser (Fresenius Polysulfone®) and a cut-off of 40 kD with the goal of clearing myoglobin. Pre- and post-dialysis myoglobin was measured at the first session, showing a 50% decrease (pre-HD 47,110 ng/ml

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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.

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Guillian-Barre syndrome secondary to tacrolimus in a patient with corticoresistant nephrotic syndrome secondary to focal and segmental glomerulonephritis and a coexisting IgA nephropathy☆

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.[1] The association between respiratory and gastrointestinal infections is well known and occurs in two-thirds of cases.[2,3] 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 glomerulofritis and a IgA nephropathy. We present the case of a 65-year-old patient with multiple comorbidity, nephrotic syndrome secondary to focal and segmental glomerulofritis 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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