than 80% of glomeruli, determines a dismal prognosis, indicating a worse response to immunosuppressive treatment[5].

Our case report illustrates that an uncommon extra-renal involvement like an episcleritis, can be the form of presentation of a systemic disease with lethal potential. Although the prognosis in terms of progression for ESRD is mainly determined by the renal histology, the type of ANCA involved and serum creatinine value when treatment is begun, the patient survival is still dependent on the level of clinical suspicion leading to an early diagnosis and treatment.

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Severe ethanol poisoning treated by haemodialysis

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To the Editor,

Standard treatment for severe ethanol poisoning consists of an aggressive treatment with cardiovascular and respiratory support, and close monitoring of the electrolytes, preventing hypothermia and hypoglycaemia. There are many cases of severe ethanol poisoning that have been successfully treated with conservative treatment. Haemodialysis is recommended for patients with signs of severe poisoning, and serum ethanol levels above 600mg/dl, (this figure is less for adolescents).[1]

We present the case of a patient with severe ethanol poisoning treated with haemodialysis, whose serum ethanol levels decreased rapidly.

The patient was a 57 year-old man, with epilepsy from 30 years of age, former alcoholic. He had experienced traumatic brain injury in 2008 as the result of an epileptic seizure. He was found unconscious in his home, with two empty whisky bottles and was suspected to have taken 4g of carbamazepine. In the examination we observed that he was in a state of coma, with slightly anisocoric pupils, pulse: 67 beats/min, blood pressure 80/50mm Hg, temperature 36.2ºC; and pulmonary auscultation revealed reduced vesicular murmur in the left haemothorax. There were no other significant findings.

He was haemodynamically unstable, and upon admission to the intensive care unit, vasopressor treatment and respiratory support were indicated. Toxic levels of ethanol (650mg/dl) were found. The biochemistry analysis showed: Na: 137mmol/l; K: 4mmol/l; Cl: 106mmol/l; glucose 173mg/dl; urea 42mg/dl; creatinine: 1.39mg/dl; anion gap: 18mOs/kg; osmolar gap: 126mOs/kg. Blood gasometry: pH: 7.05; HCO_3: 16.6mmol/l; pCO_2: 60mm Hg; pO_2: 47mm Hg. Due to instability and poor response to conservative treatment, haemodialysis was performed for 3 hours with a polysulfone dialyser at a blood flow of 250ml/min. After this session, serum ethanol levels reduced to 373mg/dl and metabolic acidosis was corrected.

The patient then had symptoms of bronchial aspiration and acute pancreatitis, which was resolved with antibiotic and conservative treatment.

Dialysis was recommended to treat severe ethanol poisoning for the first time in 1960, given that it is four times quicker than physiological elimination of ethanol.[2] However, deciding which patients with severe poisoning are eligible to undergo haemodialysis is a controversial matter. Some authors suggest that it is sufficient with a conservative treatment,[3] while others believe that haemodialysis should be considered for those patients with a serum level above 600mg/dl,[4] given that it could reduce the length of the coma and the risk of bronchial aspiration, correct hypothermia and hypoglycaemia, improve metabolic acidosis, and reduce the risk of arrhythmia. Furthermore, that alcohol is easily eliminated by haemodialysis as it is a small, water-soluble molecule which does not bind to proteins. Its volume of distribution is also limited.

We recommend indicating haemodialysis as a therapeutic option for patients with signs of severe ethanol poisoning, whose clinical profile does not improve following conservative treatment, provided that the acute complications of haemodialysis are assessed.

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Delayed diagnosis of primary hyperoxaluria in a young patient with advanced chronic renal failure

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To the Editor,

Primary hyperoxaluria (PHO) is a fairly rare metabolic alteration. Its annual incidence is 0.11-0.26/100 000 births and its prevalence is 1-2.9/1000 000 inhabitants, approximately.[1] In Spain, there is a relatively high number of cases, given its increased incidence on the Canary Islands (especially La Gomera).[2] Diagnosis is usually delayed: with an average interval of 3.4 years between the onset of the symptoms and its diagnosis. Only 30% of cases are diagnosed early.[3] As such, in most cases, patients present with oxalate deposits in the definitive diagnosis, even in the cardiovascular system, which inevitably leads to death. PHO is caused by an enzymatic defect located in the hepatocyte peroxisome, which enhances glyoxylate conversion to poorly soluble oxalate.[4] The *AGXT* gene is affected in primary hyperoxaluria type I, which is found in the chromosome 2q36-37. It corresponds with the alanine-glyoxylate aminotransferase enzyme (43kDa protein)[2], which needs vitamin B_6 (pyridoxine) to function correctly. Various mutations in the *AGXT* gene have been observed, involving multiple defects, among which the most frequent is the lack of immunoreactive and catalytic activity (42%).[5] The only treatment performed successfully in these patients is liver or liver and kidney transplant, which may supplement enzymatic activity and kidney function, respectively.

We present the case of a 24-year-old Maghreb man, with a history of chronic renal failure (CRF) secondary to nephrocalcinosis. He presented with several infections, two abscesses in the right psoas muscle and the left sternoclavicular joint, although the aetiological origin could not be identified in either of the two cases. In the same year he had a thalamic stroke and right perimesencephalic haemorrhage. He was admitted to his area hospital with severely deteriorated condition, with recurrent fever and episodes of arthritis in his right shoulder due to *S. epidermis* and in his left sternoclavicular joint due to enterococcus. Both were secondary to permanent catheter-related bacteraemia in the right jugular vein (he previously presented with thrombosis in several vascular accesses). During hospital stay (which lasted a year) numerous complications occurred: chronic severe anaemia, upper digestive haemorrhage, uraemic pericarditis, and pneumonia with pleural effusion. In the last few weeks of the hospital stay, he had left-side neck pains, significantly limited functions, fasciculations, fever, and negative blood culture tests. Magnetic resonance imaging (Figure 1) was performed. Secondary spondylodiscitis was suspected and the patient was referred to our health centre for a decompressive laminectomy.

During the physical examination upon arrival, we found deterioration of his general condition, cachexia with muscular atrophy (which prevented him from walking), hypotension, tachycardia and fever. He had a normal heart rate and a pansystolic murmur with hypoventilation of the lung bases. He had painless hepatosplenomegaly. Both knees were swollen and the right shoulder was visibly swollen.

Anaemia was discovered in the laboratory tests (Hb: 8.3g/dl), leukocytosis: 18 900, parathyroid hormone (PTH): 83pg/ml and changes to inflammatory and nutritional parameters (GGT: 544IU/l; albumin: 0.7g/dl and ferritin: 5.93ng/ml PCR). A bone scan was performed, which showed signs of advanced secondary hyperthyroidism. The main alterations can be seen in Figure 1. The cervical CT revealed a significant kyphosis at the C5-C6 vertebral body height, alteration in their morphology and a severe reduction in the antero-posterior diameter, which significantly compressed the medulla at that point and caused foraminal stenosis.

A biopsy and a bone marrow aspiration were performed which found multinucleated giant cells derived from monocyte macrophages containing oxalate crystals birefringent to polarised light. In the bone marrow the medullar space had been substituted by concentrically disposed crystals, grouped together in a star or rosette shape, which were refringent to polarised light, with peripheral reaction of giant cells and trabecular bone destruction (Figure 2). With these findings the patient was diagnosed with primary hyperoxaluria. Palliative treatment with vitamin B and intensive dialysis was indicated, but the patient died 30 days after hospitalisation.

This case illustrates the harmful consequences associated with the delayed diagnosis of this rare disease, which is fatal if aggressive treatment is not indicated early. Current therapeutic alternatives for patients with CRF are