She had stage V CKD and had been on periodic haemodialysis for four years. She had a single chamber pacemaker due to chronic atrial fibrillation with symptomatic bradycardia (repeated syncope). Other history findings included type 2 diabetes mellitus treated with oral anti-diabetic medication, and high blood pressure. Her regular medication included omeprazole, amiodarone and aspirin (none of which affected on potassium homeostasis). Her adherence to medical treatment was poor and she often consumed unsuitable foods. Physical examination was unremarkable except for the aforementioned muscle weakness. The constants were correct and the patient was normoglycaemic. The electrocardiogram (ECG) showed a pacemaker rate, without any other abnormalities. The urgent analysis revealed potassium levels of 8.2mEq/l. While waiting for the haemodialysis session, she began conventional treatment for hyperkalaemia, with calcium gluconate, intravenous salbutamol and an infusion of insulin and glucose being administered.

Minutes after beginning this treatment, the patient experienced a sudden decrease in her level of consciousness, with undetectable blood pressure and signs of peripheral hypoperfusion. At that moment, the ECG showed failure of ventricular capture (Figure 1). Urgent haemodialysis was performed, with both symptoms and electrocardiographic abnormalities being quickly and completely reversed.

DISCUSSION

Severe hyperkalaemia is a common problem in patients with advanced CKD.[1] It may affect 5%-10% of patients on chronic haemodialysis monthly. It may also require urgent haemodialysis in up to 24% of patients with end-stage CKD and it has a significant mortality rate.[1]

Potential causes of hyperkalaemia in patients such as ours are multifactorial and may include, in addition to CKD, metabolic acidosis, hyporeninemic hypoaldosteronism (common in diabetics), defects of cell Na^+/K^+-ATPase and poor diet[1-3] (recognised by our patient).

In patients with cardiac pacemakers, hyperkalaemia can cause various types of dysfunction of the device, with very serious consequences.[4,5] In our case, there was a failure of ventricular capture. In this situation, excess of extracellular potassium increases the resting potential of the myocardial cell membrane.[4] If this potential exceeds the energy emitted by the pacemaker according to its programming, cardiac muscle cell depolarisation is inhibited. This situation is recognised in the ECG by the absence of depolarisation after the spicules. The clinical manifestation, which is clearly serious, is asystole.[4,5]

In conclusion, the presence of a cardiac pacemaker does not always protect the myocardium from the deleterious effects of hyperkalaemia. Moreover, the device requires a normal electrolyte balance for proper operation. Therefore, heart rate monitoring, which is essential for all symptomatic hyperkalaemia, should not be overlooked in patients with pacemakers. Likewise, the presence of a pacemaker must not be a decisive factor when urgent haemodialysis is indicated for severe hyperkalaemia.

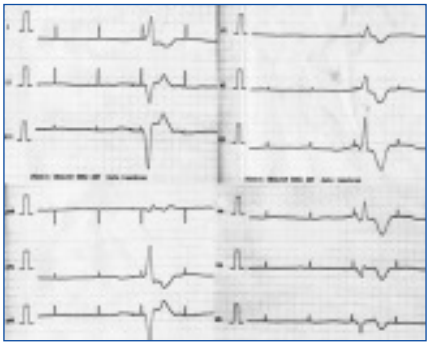

Figure 1. 12-lead electrocardiogram showing the failure of capture (absence of QRS complexes after the first, second and fourth spicules).

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

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Pregnancy in patient with cirrhosis and cryoglobulinemic vasculitis

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To the Editor:

Pregnancy is a rare event in patients with cirrhosis[1] and women with hepatitis have an increased risk of complications during pregnancy.[2,3] There are few reports on this topic. The cryoglobulinemic vasculitis associated with HCV infection is a severe systemic

disease involving the kidneys; it develops due to deposits of cryoglobulins. The course is often fatal or leads to end-stage renal disease. The disease tends to relapse. Women with this disorder rarely become pregnant and conclude successfully the pregnancy.

Our experience: a 30-year-old woman suffering from HCV-cirrhosis was admitted for severe peripheral edema with proteinuria 7g/day and albumin 2.3g/dL. The glomerular filtration rate was in the normal range. Renal biopsy showed a membranous-proliferative glomerulonephritis. The patient received 1 cycle of 12 plasmapheresis associated with Mycophenolate Mofetil (MM) 2g/day and prednisone 12.5mg/day therapy. After 1 month, we observed a partial remission of proteinuria (3g/day). The MM therapy was continued. After 2 years, the patient reported amenorrhea lasting for 3 months. The beta-HCG test and U.S. scan confirmed the pregnancy. The MM was interrupted whereas prednisone therapy was maintained at 5mg/day. During pregnancy, proteinuria was always less than 2g/day and renal function was regular. The pregnancy continued without major problems. A cesarean section was performed at the thirty-sixth week of pregnancy. Laboratory tests showed: white blood cells: 3.170/mmc, Hb: 8.2g/dL. PLT: 46.000/mmc, AST: 60IU/L, ALT 33IU/L, gammaGT: 14IU/L, total bilirubine: 1.84mg/dL, total protein: 4.1g/dl, albumin: 2.16g/dL. The fetus was healthy and growth corresponded to the twenty-eighth week of pregnancy. Anti-HCV antibodies detection was negative and the child follow-up did not show any significant diseases after 3 years from birth. The patient resumed MM and prednisone therapy. One year after the child's birth, the patient showed good health with proteinuria 1 g/day; she continued MM 2g/day and prednisone 5mg/day. After 18 months postpartum, proteinuria increased to 4g/day. We carried out again a cycle of six plasmapheresis, achieving a reduction of proteinuria (<1g/day). Therefore, we carried out the maintenance of MM and prednisone therapy at the same dose. Although pregnancy in patient with cirrhosis remains rare, recent improvements in the treatment of cirrhosis led to an increase in life expectancy and quality of life, making pregnancy a most frequent event. Outcomes of pregnancy in patients with cirrhosis are poorly described. Regarding neonatal well-being, there is no association between vertical transmission of HCV and gestational age at delivery or the presence of chorioamnionitis. There is no evidence demonstrating an increased risk of HCV transmission in HIV-negative women who breast feed.[4] There are some reports regarding a worsening of HCV-liver disease after pregnancy.[5] Regarding immunological pathology, the prognosis associated with many forms of systemic vasculitides was quite grim. Advances in this field have allowed us to focus on issues related to quality of life such as fertility, conception, and pregnancy among women with vasculitis.[6] This case report shows the possibility of a favourable outcome, if the pregnancy occurs during a clinical stabilization phase of cirrhosis and vasculitis.

Conflict of interest

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

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Severe hypocalcaemia post-denosumab

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To the Editor:

Denosumab is a human IgG2 monoclonal antibody that binds to RANKL (receptor activator of nuclear factor KB ligand gene), thus causing a reduction in osteoclast activity. It has been approved since 2010 for use in osteoporosis and does not require adjustment in accordance with renal function, although several studies indicate an increase of hypocalcaemia in patients with renal failure (RF). It is administered subcutaneously biannually.[1]

We report the case of a 46-year-old woman with high blood pressure, dyslipidaemia, stage 5 chronic kidney disease (CKD), focal segmental glomerulonephritis and severe seronegative rheumatoid arthritis resistant to different drugs. Due to the presence of pathologic fractures to the pelvis, the Department of Rheumatology decided to start treatment with 60mg denosumab. She received treatment with 2.4g/12 hours sevelamer, 0.50mg Rocaltrol®: 5 tablets/week, 1 tablet/8 hours Mastical®, 266µg/15 days calcifediol, antihypertensive drugs and painkillers. The analytical data were: urea 205mg/dl, creatinine 5.95mg/dl, calcium 8.8mg/dl, albumin 3.8g/dl, intact