For that reason, information on the antihypertensive drugs BRS independently of arterial function and structure [5]. significantly influence autonomic control and therefore alter the use of different antihypertensive drugs, such as which have been shown to impair BRS, enhance vascular inflammation. These variables include diabetes mellitus and smoking, reduced BRS, elevated arterial stiffness and vascular calcification obviously present in their study population are involved in the correlation between TTS and BRS (\( r = 0.41 \)), they failed to show a significant association between vascular calcification and increased arterial stiffness. This raises the question whether other variables not included in their analysis but obviously present in their study population are involved in reduced BRS, elevated arterial stiffness and vascular calcification. These variables include diabetes mellitus and smoking, which have been shown to impair BRS, enhance vascular calcification and lead to arterial stiffening [3,4]. Furthermore, the use of different antihypertensive drugs, such as \( \alpha \)- and \( \beta \)-blockers or angiotensin-converting enzyme inhibitors, can significantly influence autonomic control and therefore alter BRS independently of arterial function and structure [5]. For that reason, information on the antihypertensive drugs used in the study population should be given by the authors. The link between alterations in arterial structure, arterial function and autonomic regulation might offer new pathophysiological insights, but larger studies controlling for the above mentioned factors have to be done to prove this relationship.

Conflict of interest statement. None declared.

Vascular calcification and increased mortality in dialysis patients: is the baroreflex sensitivity the answer?

Sir,

In their interesting paper, Chesterton et al. [1] gave evidence for a possible link between vascular calcification, increased arterial stiffness (determined by time to shoulder, TTS) and impaired autonomic function (reduction in baroreflex sensitivity, BRS). This study adds new insights in the currently ongoing discussion dealing with vascular calcification and increased mortality in dialysis patients [2]. Impaired autonomic control of blood pressure due to vascular calcification could not only be a possible explanation for dialysis-induced hypotension but also a significant risk factor for the excessive cardiovascular mortality found in the dialysis population. Although Chesterton et al. [1] showed a significant association between vascular calcification and BRS (5.67 ± 0.76 vs 3.43 ± 0.38 ms/mmHg in the group with and without calcification) as well as a significant but low correlation between TTS and BRS (\( r = 0.41 \)), they failed to show a significant association between vascular calcification and increased arterial stiffness. This raises the question whether other variables not included in their analysis but obviously present in their study population are involved in reduced BRS, elevated arterial stiffness and vascular calcification. These variables include diabetes mellitus and smoking, which have been shown to impair BRS, enhance vascular calcification and lead to arterial stiffening [3,4]. Furthermore, the use of different antihypertensive drugs, such as \( \alpha \)- and \( \beta \)-blockers or angiotensin-converting enzyme inhibitors, can significantly influence autonomic control and therefore alter BRS independently of arterial function and structure [5]. For that reason, information on the antihypertensive drugs

Can rhabdomyolysis be the only cause of acute renal failure in leptospirosis?

Sir,

Acute renal failure (ARF) is a well-known complication of leptospirosis in its severe form (Weil’s syndrome). Generally, ARF is accompanied by jaundice and thrombocytopenia, with only sporadic case descriptions of milder forms of ARF in anicteric patients (rarely requiring dialysis). Thrombocytopenia is also closely correlated with ARF occurrence [1] and is described in all anicteric cases with ARF. The pathophysiology of ARF in leptospirosis evolves hypovolaemia, direct tubular toxicity and rhabdomyolysis. This case describes a patient with leptospirosis, severe rhabdomyolysis and ARF, with no jaundice or thrombocytopenia. Clinical and laboratory findings point to rhabdomyolysis as the major factor responsible for kidney injury.

A 38-year-old man, with no previous disease, had a history of myalgia 2 days previously and noticed reddish urine.

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painless and tense calf muscles. Serum laboratory evaluation showed serum creatinine (sCr) 7.5 mg/dl, urea 280 mg/dl, sodium 139 mEq/l, potassium 6.8 mEq/l, calcium 0.98 mmol/l, phosphorus 8.6 mg/dl and creatine kinase 602 2341U/l. Haematological examination showed the presence of leucocytosis (16.7×10^9/l) only, with no anemia (haemoglobin 13.7 mg/dl) or thrombocytopenia (456×10^9/l). Fractional excretion of sodium was 0.8% and the urinary excretion of potassium was 630 mmol/day. The patient needed dialysis support for 2 weeks (eight sessions) and evolved with a decrease in creatine-kinase levels and complete recovery of renal function (sCr, 1.2 mg/dl). Urine output was maintained during the entire hospital stay, with a mean output of 2.050 ml/day. Leptospirosis diagnosis was confirmed by positive serologic tests (ELISA IgM and microscopic agglutination test). Investigation for other infectious diseases (HIV, cytomegalovirus, toxoplasmosis and Coxsackie) was negative.

The pathophysiology of renal failure in leptospirosis involves proximal tubular dysfunction, augmenting distal sodium delivery and, consequently, potassium excretion by the intact distal tubule [2]. In the presented case, the presence of hyperkalaemia is explained by the rhabdomyolysis. However, the low fractional excretion of sodium and urinary potassium of the patient described is dissimilar to the findings described by Covic et al. [3]. These authors demonstrated, in a large series of ARF due to leptospirosis, a high fractional excretion of sodium (>1%) in all patients, even in those with volume depletion. Moreover, in the same series, 20/22 patients with hypokalaemia had a urinary excretion >1000 mmol/day.

On the other hand, the low urinary excretion of sodium and potassium observed in this case is in agreement with ARF due to rhabdomyolysis [4]. In conclusion, the absence of jaundice, normal platelet value and low renal excretion of sodium and potassium allowed us to conclude that the major renal lesion in this case was due to rhabdomyolysis, with no or minimal involvement of leptospirosis.

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Dialysis encephalopathy secondary to aluminum toxicity, diagnosed by bone biopsy

Sir,

Dialysis encephalopathy is a syndrome observed in chronic renal insufficiency patients on dialysis, characterized by dementia, speech alterations, myoclonias, asterixis and convulsions, associated with typical electroencephalogram alterations. These clinical findings show a poor prognosis, resulting in death in the majority of cases. The association with aluminum toxicity is indicated frequently as the underlying cause and described in the majority of the reports in the literature, although other etiologies cannot be discarded [1–5].

Case. A 40-year-old black male patient had chronic renal failure of undetermined aetiology, and was on haemodialysis for 5 years and without personal antecedents of aluminum use or exposure to heavy metals. After 3 years of dialysis therapy, he began to present clinical findings of slight mental confusion and shivering in his extremities after haemodialysis sessions, showing spontaneous improvement, but with a repetitive and progressive character. Hospitalized 30 days after the onset of symptoms, he presented an intense state of mental confusion, speech apraxia and myoclonias, evolving into a diminished level of consciousness (Glasgow ≤8) and the need for mechanical ventilation. On this occasion he presented the following serum biochemical results: serum creatinine, 10.3 mg/dl; blood urea nitrogen, 57 mg/dl; serum potassium, 4.0 mEq/l; serum calcium, 4.4 mEq/l and anaemia, with no signs of an infectious process (haematocrit, 27%; haemoglobin, 8.5 g/dl). A cerebral computed tomography scan was performed, which was normal. He had a normal liquor and negative toxicological exams. The electroencephalogram showed wide θ and α-δ waves, often in a triphasic fashion, and diffuse slow spikes. Following support, clinical improvement was observed on the second day of treatment and the patient was discharged from hospital in good clinical condition. However, from this period onward, he began to present similar symptoms with variable intensity, always after haemodialysis sessions. An investigation for aluminum toxicity was begun. The dialysis unit presented reverse osmosis-treated water with normal levels of aluminum (<10 µg/l); the same occurred with the patient’s serum aluminum (47 µg/l, an exam confirmed twice on other occasions, normal up to 30 µg/l), as well as ferritin at 150 ng/ml (15–200 ng/ml) and parathormone at 100 pg/ml (7–53 pg/ml). The test for desferoxamine was not suggestive of aluminum toxicity. In spite of the lack of evidence, a bone biopsy was performed which showed numerous deposits of aluminum. Desferoxamine treatment was then initiated, with a progressive improvement in the symptoms until disappearance and normalization of the electroencephalogram.

Comment. Many studies have shown good results with desferoxamine following dialysis encephalopathy by aluminum toxicity, as well as treatment of the dialysis water by reverse osmosis, with electroencephalogram normalization [6,7]. However, in all the cases described, diagnosis was based on the verification of a serum aluminum level increase or a positive desferoxamine test. In our case, it was necessary to perform a bone biopsy for confirmation of the diagnosis.