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Letters to the Editor

Acute kidney injury secondary to rhabdomyolysis in a nonagenarian patient taking statins. Lessons to be considered☆

Fracaso renal agudo secundario a rabdomiólisis por estatinas en una paciente nonagenaria. Lecciones a tener en cuenta

Dear Editor,

Elderly patients with chronic kidney disease (CKD) are more prone to develop episodes of acute kidney injury (AKI), frequently resulting from drug dosage non adjusted for glomerular filtration rate (GFR) or by drug-to-drug interactions due to CKD. We present a case of AKI secondary to rhabdomyolysis in a nonagenarian patient taking statins as well as other potentially nephrotoxic drugs. A comment on practical lessons to be learned has also been included.

A 91-year-old patient with a history of hypertension (HTN) and hypercholesterolaemia diagnosed at the age of 85 on treatment with atorvastatin 10 mg. Two years later the patient was switched to rosuvastatin 20 mg because of poor control of cholesterol levels. Treatment with this drug was continued with normalization of cholesterol levels. The rest of treatment included telmisartan 80 mg, hydrochlorothiazide 25 mg, esomeprazole 40 mg and ibuprofen 600 mg/24 h for the management of arthralgia. The serum creatinine levels was 1.55 mg/dl.

The patient presented at the emergency room with a history of 72 h of sudden right flank pain without irradiation, accompanied by oliguria, and no fever. She also complained of muscle weakness in lower extremities.

Physical examination showed no remarkable findings except for decreased osteotendinous reflexes in lower extremities.

Lab results upon admission were: haemoglobin 13.3 g/dl, glucose 99 mg/dl, creatinine 4.47 mg/dl, urea 1.24 g/l, albumin 3.28 g/dl, AST 360 U/l, ALT 165 U/l, GGT 11 U/l, alkaline phosphatase 93 U/l, amylase 58 U/l, total cholesterol 117 mg/dl, uric acid 6.3 mg/dl, sodium 138 mEq/l, potassium 2.93 mEq/l, chloride 90.7 mEq/l, pH 7.45, pCO_2 38 mmHg, CO_3H 26.1 mmol/l; proteinuria: 0.6 g/24 h, and CPK 9.612 μ/l.

Abdominal ultrasound showed kidneys normal in size and structure, without evidence of pelvicalyceal ectasia.

Fluid therapy was initiated, followed by progressive recovery of renal function together with complete normalization of CPK and transaminases levels. Creatinine was 1.9 mg/dl at discharge and 1.44 mg/dl 2 months later according to outpatient readings.

Our patient had an episode of rhabdomyolysis-related AKI, which was likely associated to the use of rosuvastatin since no other cause of rabdomyilysis was detected.[1]

Of note, the patient, who had CKD stage 4 according to KDIGO[2] with a baseline CKD-EPI GFR of 29.06 ml/min/1.73 m^2. was receiving excessive dose of rosuvastatin.

The present case illustrate the importance of outpatient measurements of GFR in elderly patients.[3] According to its core data sheet, the use of rosuvastatin is not recommended in patients with a GFR <30 ml/min, with a starting dose of 5 mg/day indicated for elderly patients. The stage of CKD may have been missed in primary care if the GFR was not measured using equations.

The undeniable usefulness of statins for primary prevention among patients aged 85 and over can also be discussed.[4] The special feature of this report lies in the fact that our patient developed rhabdomyolysis following long-term treatment with statins (5 years) an almost unparalleled case in the literature, suggesting that the complication may arise at any

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time. Muscle necrosis has been described following treatment with both drugs.[5,6] Between 15 and 33% of patients develop AKI.[7] Up to 37% of them require haemodialysis. Mortality is 5%, although it rises to 25% when complicated by AKI.

The mechanism of muscle toxicity is not fully understood. Statins may interfere in the synthesis of the Q10 coenzyme (CoQ10 or ubiquinone) involved in energy production of muscle cells.[8]

Muscular symptoms usually start weeks or months after the initiation of treatment, but just as in our patient, it may occur at any time. In a series including 44 patients, mean treatment duration before symptoms was 6.3 months (range: 0.25–48). Symptoms resolved following drug withdrawal after a mean of 2.3 months (range: 0.25–14).[9]

Also important is the fact that the patient was taking the following drugs: a non-steroid anti-inflammatory drug, an angiotensin receptor antagonist, and a diuretic. This combination is sometimes referred to as a "Triple whammy" and has been associated with a higher incidence of AKI.[10] In our patient, other drugs may have led to decreased GFR and the development of muscle necrosis.

In summary, our case highlights the need for a thorough assessment of the risk-benefit profile when prescribing drugs to elderly patients with CKD, which should be staged based on the measurement of GFR. Statin-associated rhabdomyolysis may occur at any time of the disease course, while concomitant use with certain drugs may increase toxicity.

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Acute renal colic: Beyond kidney stones

Cólico renal agudo: más allá de los cálculos renales

Dear Editor,

A 59-year-old woman without relevant past medical history presented with hematuria and renal colic. After a negative diagnostic work-up, sickling vaso-occlusive crisis in the setting of sickle cell trait (SCT) was diagnosed. This report aims to raise awareness that SCT should be included in the differential diagnosis of unexplained hematuria and/or renal colic.

Renal abnormalities are frequently described in patients with sickle hemoglobinopathies, but SCT patients (heterozygous carriers, with one sickle cell gene and one normal gene) are mostly asymptomatic.[1] However sickle cell crisis can occur if the patient is exposed to hypoxic conditions, high altitude and intense physical exercise.[1] Acute events include vaso-occlusive crises such as papillary necrosis of the kidney, ischemic stroke and infections.[2]