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.

REFERENCES

1. Torres PA, Helmstetter JA, Kaye AM, Kaye AD. Rhabdomyolysis: pathogenesis, diagnosis, and treatment. Ochsner J. 2015;15:58–69.
2. Stevens PE, Levin A, Kidney Disease: Improving Global Outcomes Chronic Kidney Disease Guideline Development Work Group Members. Evaluation and management of chronic kidney disease: synopsis of the kidney disease improving global outcomes 2012 clinical practice guideline. Ann Intern Med. 2013;158:825–30.
3. Martínez-Castelao A, Górriz JL, Bover J, Segura-de la Morena J, Cebollada J, Escalada J, et al. Documento de consenso para la detección y manejo de la enfermedad renal crónica. Nefrologia. 2014;34:243–62.
4. Stone NJ, Robinson JG, Lichtenstein AH, Bairey Merz CN, Blum CB, Eckel RH, et al. 2013 ACC/AHA guideline on the treatment of blood cholesterol to reduce atherosclerotic cardiovascular risk in adults: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. J Am Coll Cardiol. 2014;63:2889–934.
5. Chang CH, Kusama M, Ono S, Sugiyama Y, Orii T, Akazawa M. Assessment of statin-associated muscle toxicity in Japan: a cohort study conducted using claims database and laboratory information. BMJ Open. 2013;3, pii: e002040.
6. Martínez-López D, Enríquez R, Sirvent AE, Redondo-Pachón MD, Millán I, Amorós F. Rabdomiólisis y fracaso renal agudo tras trabajo intenso en un paciente en tratamiento con rosuvastatina. Nefrologia. 2012;32:127–8.
7. Oshima Y. Characteristics of drug associated rhabdomyolysis: analysis of 8,610 cases reported to the U.S. Food and Drug Administration. Intern Med. 2011;50:845–53.
8. Rundek T, Naini A, Sacco R, Coates K, DiMauro S. Atorvastatin decreases the coenzyme Q10 level in the blood of patients at risk for cardiovascular disease and stroke. Arch Neurol. 2004;61:889–92.
9. Hansen KE, Hildebrand JP, Ferguson EE, Stein JH. Outcomes in 45 patients with statin-associated myopathy. Arch Intern Med. 2005;165:2671–6.
10. García RM, Cols M, Chevarría JL, Osuna RG, Carreras M, Lisbona JM, et al. Fracaso renal agudo secundario a combinación de inhibidores del sistema renina-angiotensina, diuréticos y AINES. La Triple Whammy. Nefrologia. 2015;35:197–206.

Jose Maria Peña-Porta*, Alba Martinez-Burillo, Oscar Urbano-Gonzalo, Rafael Alvarez-Lipe

Servicio de Nefrología, Hospital Clínico Universitario Lozano Blesa, Zaragoza, Spain

* *Corresponding author.*
E-mail address: jpenaporta@gmail.com (J.M. Peña-Porta).

http://dx.doi.org/10.1016/j.nefroe.2015.08.006

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]

A 59-year-old Caucasian woman presented with dark-red urine and colicky pain in the left flank. She had no fever, dysuria, urinary urgency and no history of trauma, nephrolithiasis or additional episodes of hematuria. She was of Asian ascendancy and had non-consanguineous parents. Her medical history was unremarkable, except for hypertension. Her chronic medication consisted of candesartan/hydrochlorothiazide and she denied non-steroidal anti-inflammatory drugs use.

Physical examination revealed modest overweight, blood pressure of 110/60 mmHg, no evidence of hepatosplenomegaly, although left costovertebral tenderness was noted.

Laboratory tests showed normal blood cell counts, coagulation parameters, electrolytes, and renal and hepatic tests. No rhabdomyolysis nor hemolysis were present. Urine dipstick revealed a specific gravity of 1.010, 3+ hemoglobin and no protein; the microscopic exam showed 168 red blood cells per high power field, but no casts or white blood cells. Urine cultures, including for mycobacteria, were negative. She had no anomalous cells in cytology and a normal cystoscopy. A computed tomography scan (Fig. 1A–C) suggested renal papillary necrosis and excluded other abnormalities. There were no criteria for diabetes mellitus. Hemoglobin's electrophoresis showed the presence of an abnormal hemoglobin (Fig. 2) which was subsequently documented to be HbS. A molecular study revealed an heterozygosity for the hemoglobin sickle mutation of the β-globin gene (HBB:c.20A>T) establishing a diagnosis of SCT.

Clinical findings resolved after conservative therapy with rest, hydration and analgesia. The patient was recommended to avoid circumstances that may precipitate vaso-occlusive crises. Her daughter was referred for genetic testing.

SCT is generally asymptomatic, although occasionally associated with significant morbidity, including papillary necrosis.[1] It is a highly prevalent recessive illness, mainly found in African but also in Asian populations.[3] In SCT patients, HbS is abnormally found with plasma concentrations typically between 35% and 45% of total hemoglobin.[4]

Hematuria is the most common complication[1] of SCT, but the usual etiologies, such as stones, urinary tract infections or neoplasms should be considered first. Hence, initial testing must include urine analysis, culture, cytology and imaging studies. In SCT patients, occlusion of small vessels by sickled blood cells, causes microthrombi and ischaemia.[2] In the kidneys, this obstruction is predominantly apparent in the renal papilla due to their marginal blood supply. Papillary necrosis can be difficult to diagnose. The presence of sloughed papillae in the urine examination is diagnostic, but has low sensitivity.[5] In our patient an ultrasound and a tomography scan allowed the diagnosis. Treatment is usually conservative, but adjuvant therapies (desmopressin or epsilon-amino caproic acid) can be of benefit.[1] Most severe cases may require arterial embolization or surgical exploration. The outcome is generally good but the patient must be informed about avoidable precipitating conditions and genetic counseling.[2]

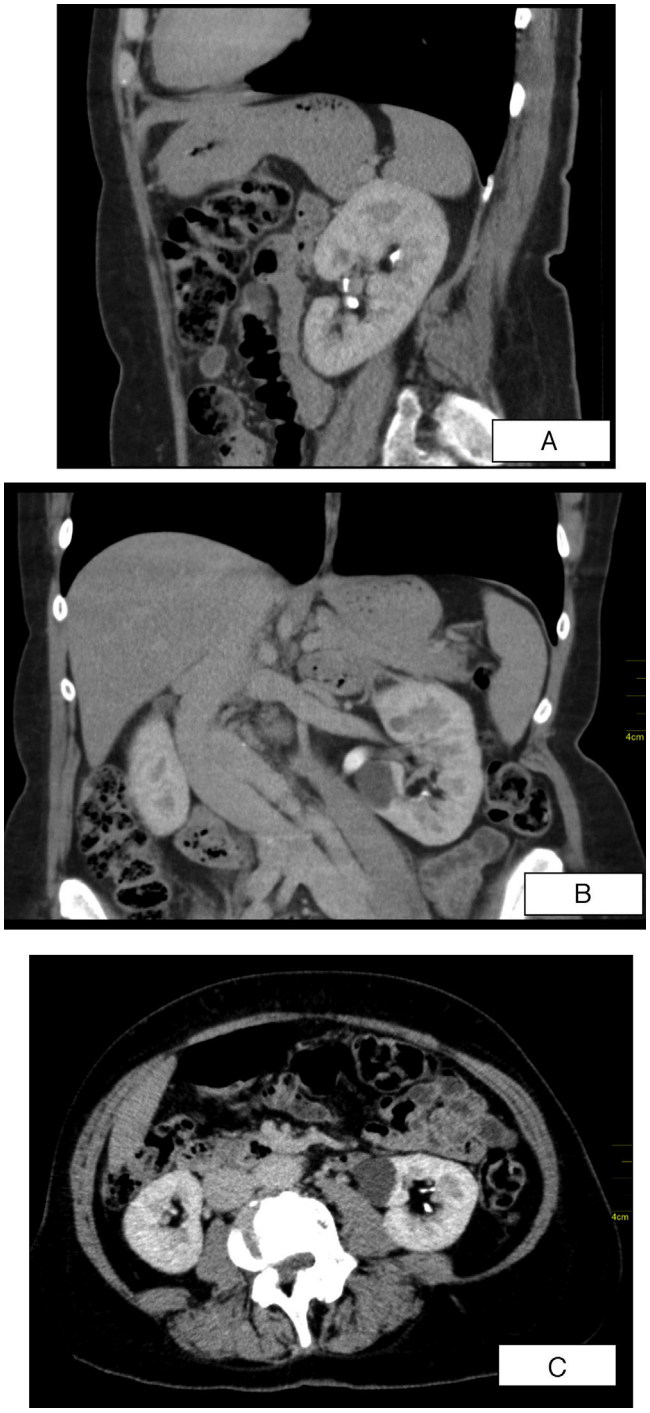

Fig. 1 – A, B, C: Sagital, coronal and axial sections of abdominal computed tomography revealing structural heterogeneity of the left renal parenquima with diminished enhancement at the tip of the medullary pyramid and the presence of small cavities filled by contrast on the borders of the small calices – aspects highly suggestive of papillary necrosis ischemia.

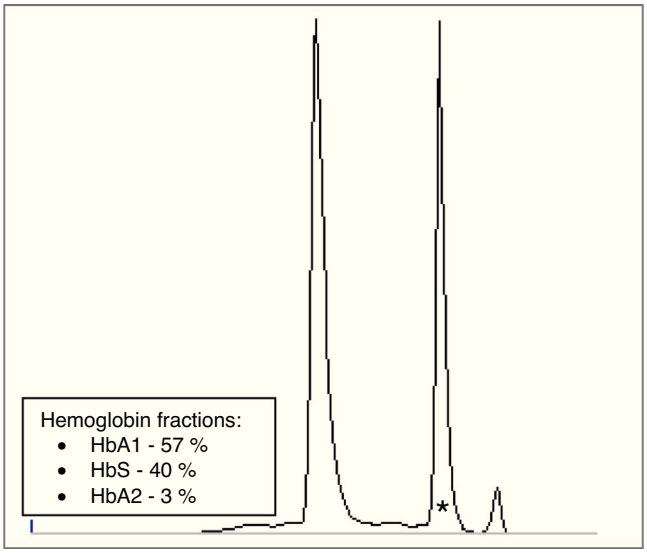

Fig. 2 – Hemoglobin electrophoresis (cellulose acetate): amino acids substitution in hemoglobin variants alter charge and subsequently hemoglobin's mobility pattern. Presence of an abnormal hemoglobin variant in Z5 region, compatible with HbS (*).

Hemoglobin's electrophoresis should be part of the diagnostic work-up of renal colic and/or hematuria, namely in patients whose etiology has never been established.

Conflict of interest

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

REFERENCES

1. Kiryluk K, Jadoon A, Gupta M, Radhakrishnan J. Sickle cell trait and gross hematuria. Kidney Int. 2007;71:706–10.
2. Phuong-Thu TP, Phuong-Chi TP, Alan HW, Susie QL. Renal abnormalities in sickle cell disease. Kidney Int. 2000;57:1–8.
3. World Health Organisation. Sickle cell anaemia. Agenda item 11.4, in 59th World Health Assembly, 27 May 2006. Geneva, Switzerland: World Health Organisation; 2006.
4. Gabriel A, Przybylski J. Sickle-cell anemia: a look at global haplotype distribution. Nat Educ. 2010;3(3):2.
5. Jung Dae Chul, Kim Seung Hyup, Sung Jung II, Sung Hwang II, Kim Sun Ho. Renal papillary necrosis: review and comparison of findings at multi-detector row CT and intravenous urography. Radiographics. 2006;26:1827–36.

Sandra Silva[a,*], Martinho Almeida[b], Lurdes Rodrigues[c], Maximino Costa[a]

[a] *Unidade de Nefrologia, Departamento de Medicina, Hospital Pedro Hispano, Matosinhos, Portugal*
[b] *Unidade de Hematologia, Departamento de Medicina, Hospital Pedro Hispano, Matosinhos, Portugal*
[c] *Serviço de Urologia, Departamento de Cirurgia, Hospital Pedro Hispano, Matosinhos, Portugal*

* *Corresponding author.*
E-mail address: oliveirasilva.sandra@gmail.com (S. Silva).

http://dx.doi.org/10.1016/j.nefroe.2016.02.001

Emphysematous pyelonephritis in a renal transplant recipient. A case study☆

Pielonefritis enfisematosa en trasplantado renal. Reporte de un caso

Dear Editor:

Emphysematous pyelonephritis (EPN) is a rare necrotizing condition of the kidney, and is particularly rare in renal transplant recipients. It is characterized by the presence of gas in the renal parenchyma, the perirenal space, or the urinary tract as a result of an infection. Infection is generally caused by gas-producing microorganisms, including *Escherichia coli* (over 80% of cases) or *Klebsiella pneumoniae*, due to mixed fermentation of glucose.[1] Over 80% of cases have been described in diabetic patients. Urinary tract obstruction is a major risk factor of EPN, and women are affected more often in a 4:1 ratio.[2] Mortality from EPN is primarily attributable to septic complications, with almost 78% of cases reported by the late 1970s. However, the improvement in management techniques has reduced this figure to 21% over the last two decades.[3]

The clinical course of EPN is typically severe and rapidly progresses to sepsis with multiple organ failures. CAT scans are considered the gold standard for diagnosis and staging.[1]

☆ Please cite this article as: Hernández-Vargas H, Sierra-Carpio M, Gil-Catalinas F, Bello-Ovalle A, Beired-Val I, Pimentel-Guzmán GI, et al. Pielonefritis enfisematosa en trasplantado renal. Reporte de un caso. Nefrologia. 2016;36:184–186.