Patent foramen ovale and anticardiolipin antibodies, a strange cause of bilateral kidney infarcion☆

Foramen oval permeable con anticardiolipina+, una causa rara de infartos renales múltiples

Dear Editor,

Renal infarctions are infrequent, usually they are a consequence of atheroembolism from the heart (atrial fibrillation, infective endocarditis, atrial myxoma, among others), renal vasculopathy or hypercoagulability states (resistance to activated protein C, hyperhomocysteinemia, antiphospholipid syndrome, etc.).[1] Paradoxical renal infarctions associated with patent foramen ovale have also been described. We present the first case of bilateral renal infarction associated with patent foramen ovale and anticardiolipin +.

The patient was 38-year-old male with persistent pain in right renal flank with a diagnosis of renoureteral colic/acute pyelonephritis. Pain worsening and new consultations, prompted patient admission. Physical examination revealed blood pressure 147/87 mmHg and body temperature of 36 °C, heart with regular rhythm, no murmurs and with preserved pulmonary sounds and pain on the right flank with palpation but not induced with percussion.

The blood chemistry showed increased LDH and APTT, with normal renal function and without proteinuria or hematuria. The electrocardiogram showed no signs of acute myocardial ischemia, and an abdominal-pelvic ultrasound revealed areas of hyperechogenicity in the right kidney. CT angiography revealed bilateral renal perfusion defects (Fig. 1), which confirmed the diagnosis of bilateral renal infarction by arteriography. The anticardiolipin antibodies were positive and a transesophageal echocardiogram revealed a patent foramen ovale without aneurysm of the interatrial septum, with the passage of bubbles while performing Valsalva maneuver (Fig. 2, Video 1). The patient developed ischemic colitis 12 days

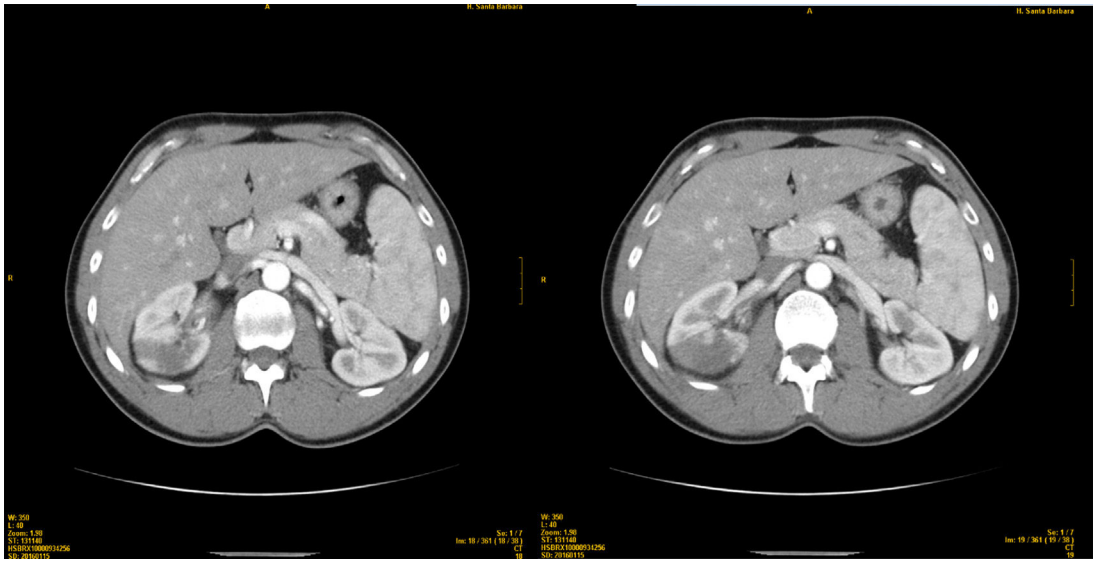

Fig. 1 – Bilateral renal perfusion defects.

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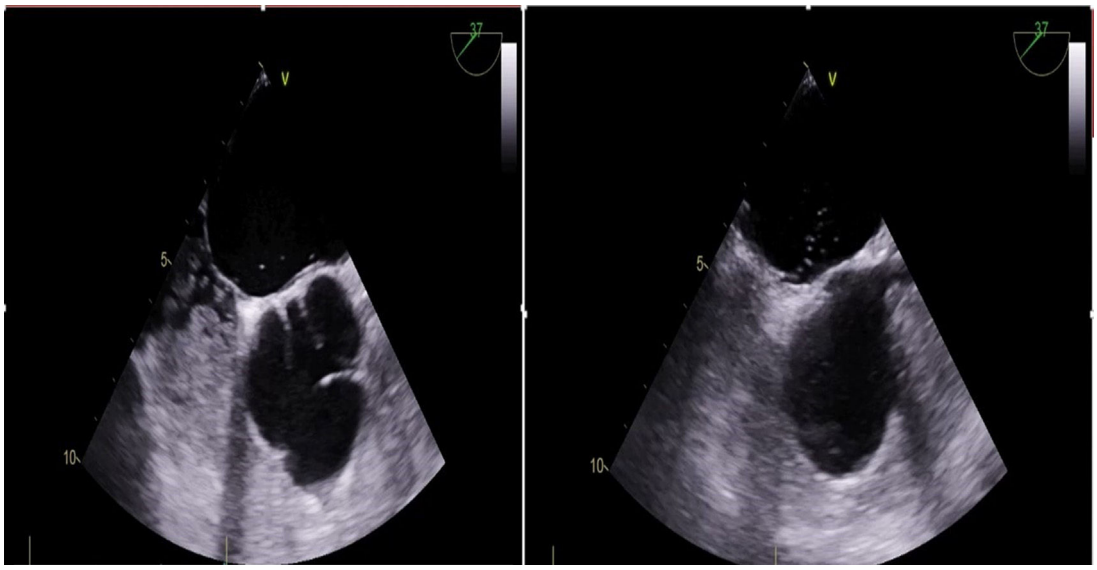

Fig. 2 – Permeable ovale foramen.

later: oral anticoagulation was started. The evolution was satisfactory and 6 months later the patient was asymptomatic and without recurrences.

Renal infarction is characterized clinically by low back pain and refractory arterial hypertension. Leukocytosis, hematuria and high CRP and LDH may be present. Renal perfusion defects in a CT angiography are very suggestive and renal arteriography is the gold standard for diagnosis

Iwasaki et al.[2] described a case of isolated patent foramen ovale manifested as renal infarction with impaired renal function. Nara et al.[3] diagnosed bilateral renal infarction in a patient with poorly controlled arterial hypertension, and discovered a patent foramen ovale. Finally, Ronco et al.[4] attended a hypertensive urgency in a patient that was found to have renal infarction and patent foramen ovale. Transesophageal echocardiography establishes the diagnosis of permeable foramen ovale. The antiaggregation begins with the first embolism; percutaneous closure can be considered in case of recurrence or aneurysm of the interatrial septum. The antiphospholipid syndrome presents with arterial and venous thrombosis,[5–7] the diagnosis is based on clinical and biochemistry findings[7] and is candidate to early anticoagulation.[8]

Here we have described the first case with patent foramen ovale, antiphospholipid syndrome and renal infarctions. We believe that it is indicated to perform an analytical and echocardiographic studies in patients with renal infarctions to diagnose structural abnormalities or thrombophilias so that antiplatelet or anticoagulant treatment is initiated early.

Conflict of interests

The authors do not declare conflicts of interest.

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Appendix A. Supplementary data

Supplementary material related to this article can be found, in the online version, at doi:10.1016/j.nefroe.2017.10.010.

REFERENCES

1. Bourgault M, Grimbert P, Verret C, Pourrat J, Herody M, Halimi JM, et al. Acute renal infarction: a case series. Clin J Am Soc Nephrol. 2013;8:392.
2. Iwasaki M, Joki N, Tanaka Yuri, Hara H, Suzuki M, Hase H, et al. A suspected case of paradoxical renal embolism through the patent foramen ovale. Clin Exp Nephrol. 2011;15:147–50.
3. Nara M, Komatsuda A, Fujishima M, Fujishima N, Nara M, Lino T, et al. Renal paradoxical embolism in a hypertensive young adult without acute ischemic symptoms. Clin Exp Nephrol. 2011;1584:582–5.
4. Ronco F, Rigatelli G, Dell' Avvocata F, Giordan M, Ronco C, Cardaioli P, et al. Embolic renal infarct, patent foramen ovale and coronary artery dissection: a strange case of cardio-renal connection. Cardiovasc Revasc Med. 2011;12:67.e5-7.
5. Jebeniani O, Bouomrani S, Hammami S, Ben Taher C, Moussa A, Mahjoub S, et al. Ischemic colitis as a first sign of primary antiphospholipid antibody syndrome. A case report. Tunis Med. 2009;87:884–6.
6. Jürgensen JS, Kettritz R, Scheider W, Koop H, Hildebrand TS, Frei U, et al. Catastrophic antiphospholipid syndrome masquerading as ischaemic colitis. Rheumatol Int. 2003;23:204–6.
7. Miyakis S, Lockshin MD, Atsumi T, Branch DW, Brey RL, Cervera R, et al. International consensus statement on an update of the classification criteria for definite antiphospholipid syndrome (APS). J Thromb Haemost. 2006;4:295–306.
8. Holbrook A, Schulman S, Witt DM, Vandvik PO, Fish J, Kovacs MJ, et al. Evidence-based management of anticoagulant therapy: antithrombotic therapy and prevention of thrombosis, 9th ed: American College of Chest Physicians evidence-based clinical practice guidelines. Chest. 2012;141:E152s.

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Obesity and kidney function; epidemiological study data: Prevalence of chronic kidney disease in Spain. EPIRCE study☆

Obesidad y funcion renal .datos del estudio epidemiologico: Prevalencia de la enfermedad renal cronica en España. Estudio EPIRCE

Dear Editor,

We would like to take advantage of World Kidney Day 2017 with the slogan "Obesity and Kidney Disease", to share epidemiological data from the study into the epidemiology of chronic kidney disease in Spain (EPIRCE).[1]

It is well known that obesity is a public health problem, and for several years different epidemiological studies have shown a clear relationship between obesity and the risk of developing chronic kidney disease (CKD).[2] The associated nephropathy is a result of hyperfiltration, glomerular hypertrophy and increased synthesis of vasoactive and fibrogenic substances and dyslipidaemia.[3]

The EPIRCE study is an observational study of a randomly-selected multistage sample in 42 sampling points (towns) stratified by habitat, age and gender, providing a representative cohort of the Spanish population (*n*: 2746). The prevalence of obesity (BMI >30 kg/m^2) was 26.1% and the *odds ratio* (OR) of CKD development was 3.5 (95% confidence interval [CI]: 2.0–6.0) while the prevalence of another cardiovascular risk factor, such as arterial hypertension (HTN), was 42% and the OR for CKD development was 6.2 (95% CI: 4.0–9.6).

Table 1 shows that the obese population is significantly more hypertensive and dyslipidaemic, with a higher rate of insulin resistance, and the higher BMI is associated with conventional risk factors (HTN, dyslipidaemia, HOMA) and with "worse" kidney function and higher proteinuria rate (Alb/creatinine). However, these changes are also seen in the "global" population, whether they are hypertensive or not.

The pathogenic mechanisms of nephropathy seem to be linked to: glomerular hyperfiltration and haemodynamic changes, the dyslipidaemia itself and a greater activation of the renin-angiotensin system, hyperinsulinaemia and a greater synthesis of leptin, oestrogen and TGF-β1.[4] Regarding the therapeutic approach it is essentially to lose weight, and the progression of nephropathy is reduced through blood pressure control, improvement of insulin resistance and lipid profile, as well as reduction of leptin and RAAS.[4] It should be noted that this association of HTN, obesity, dyslipidaemia or proteinuria is not a metabolic syndrome, a syndrome questioned not only by Reaven[5] himself but by the American Diabetes Association (ADA) and the European Association for the Study of Diabetes (EASD)[6] because the presence of a unique pathogenic substrate has not been proven. Many authors agree that the usefulness of the concept of metabolic syndrome is to highlight the association of multiple CVRFs when making clinical decisions.[5] We also consider that CKD is the principal CVRF[7] and the existence of a common pathogenic substrate which might explain the coexistence of obesity and CKD.[4]

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