4. Martínez Ruiz J, Pastor Navarro H, Carrión López P, Giménez Bachs JM, Donate Moreno MJ, Virseda Rodríguez JA. Fractura de cuerpos cavernosos. Serie de casos. Actas Urol Esp. 2008;32(6):599-602.
5. Maruschke M, Lehr C, Hakenberg OW. Traumatic penile injuries - mechanisms and treatment. Urol Int. 2008;81(3):367-9.
6. Kamdar C, Mooppan UM, Kim H, Gulmi FA. Penile fracture: preoperative evaluation and surgical technique for optimal patient outcome. BJU Int. 2008;102(11):1640-4.
7. Juaneda Castell B, Montlleó González M, Ponce de León Roca X, Gausa Gascón Ll, Caparrós Sariol J, Villavicencio Mavrich H. Fístula uretrocavernosa por fractura penena. Actas Urol Esp. 2008;32(10):1043-5.

L. M. Moratalla Charcos, T. Pastor Navarro, M. Beamud Cortés and J. M. Osca García*
Department of Urology, Hospital Universitario La Fe, Valencia, Spain

*Author for correspondence.
E-mail: luzmoratalla@comv.es
(L. M. Moratalla Charcos).

Congenital renal arteriovenous malformation: the value of magnetic resonance imaging for dignosis and intravascular management

Malformación arteriovenosa renal congénita: utilidad de la resonancia magnética para el diagnóstico y abordaje endovascular

To the Editor,

We report the case of a 40-year-old male, with a history of renoureterales attacks, who came to the emergency department for right lumbar pain, intermittent hematuria and poor response to analgesic treatment.

Abdominal X-ray showed no images suggestive of lithiasis. However, urinary tract ultrasound showed an enlargement of the right kidney (RK) related to grade III/IV ureterohydronephrosis, with no images of lithiasis. Given the persistent pain, an urgent nephrostomy was placed. This maneuver caused the immediate onset of severe bleeding through the nephrostomy tube, requiring its withdrawal and transfusion of packed red blood cells.

During the patient's hospital stay, an abdominopelvic computed tomography scan was requested for a more comprehensive evaluation. The scan detected the presence of 4 indeterminate nodular images, with a similar density to the cortex, three in the upper half of the kidney in relation to medulla and a fourth in lower half, in contact with the renal sinus, whose measurements were: 1, 3.4, 3 and 2.8 cm, confirming enlargement of the RK, but not dilatation of the excretory system.

On subsequent magnetic resonance imaging (MRI), the varicose structures of the renal hilus showed the typical "signal void" of blood in movement in T2 sequences, and after contrast administration, they showed intense enhancement in the arterial phase with rapid filling of the right renal vein, all of which was consistent with congenital arteriovenous malformation (AVM) of the RK with associated arteriovenous fistula (Fig. 1). Renal Doppler ultrasound served as a dynamic study to confirm the diagnosis.

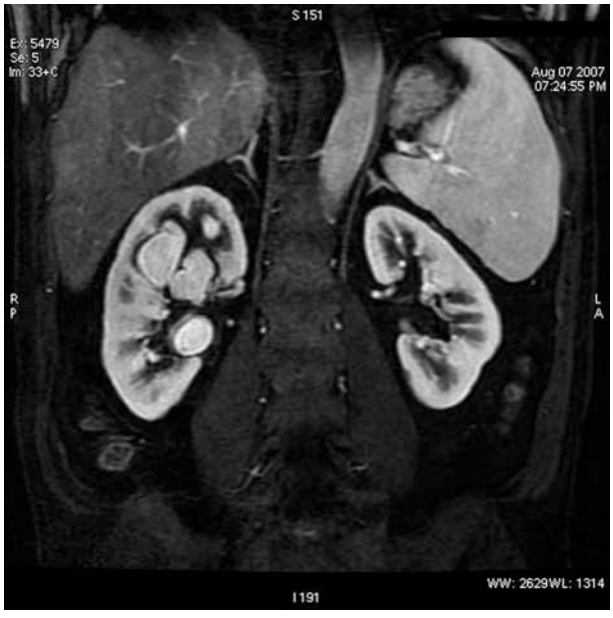

Figure 1 – MRI showing filling cavities with paramagnetic contrast in T1 sequences.

An arteriography was performed prior to embolization in which 4 vascular tangles were visualized in the right renal sinus, an opacified nephrographic phase in the RK and early

Figure 2 – Aortography. Three vascular tangles, a poorly opacified nephrographic phase, and early filling of the inferior vena cava are visible in the right kidney, and two renal arteries in the left kidney.

filling of the inferior vena cava, all findings characteristic of renal AVM (RAVM). A microcatheter were placed with two Nitinol coils in the upper segment and a third microcoil in the lower segment, thus obtaining the total disappearance of the arteriovenous shunt (Fig. 2).

On the days following percutaneous embolization the patient experienced hypertension (HT) and postembolization syndrome with low grade fever, severe flank pain, and leukocytosis, which was treated with intravenous antibiotics, analgesia, and rest. At discharge, the patient was asymptomatic, with normal creatinine, but with persistence of HT.

RAVMs are a rare clinical entity[2-4]. Their existence was first described in 1923 by Varela[1].

Their estimated prevalence is less than 0,04%[5], but is increasing as a consequence of the rise in the use of diagnostic imaging techniques[6].

Congenital RAVMs (20-25% of total) may be diagnosed at any age, although they usually occur between age 30 and 50 years[3,4]. Infections are more frequent in the RK and in women (2:1)[3]. There are located in order of frequency in the upper pole (45%), middle third (30%), and lower pole (25%)[4]. They may occur as part of various hereditary syndromes, such as the Sturge-Weber and Klippel-Trenaunay syndromes[2]. They typically have a cirsoid morphology, with tortuous nests and multiple arteriovenous communications at the segmental and interlobar level. They are composed of dysplastic subepithelial vessels (absence of elastic lamina) located at the pyelocalycial level[7]. Our case belongs to this group of renal AVMs.

Clinical suspicion and a comprehensive differential diagnosis of hematuria is essential to make the diagnosis. Plain radiological evaluation of the abdomen does not provide any characteristic sign, except in cases where the fistula is associated with a calcified aneurysm[4].

Conventional ultrasound is of little use. The sac-like and anechoic images located at the level of the renal sinus result in confusion with renal sinus hiliocystosis or vascular ectasis[2] , as occurred in our case. Doppler ultrasound is required to quantify the flow and confirm the existence of a *shunt* at the level of these sac-like structures[2,7].

MRI can identify "flow voids" in T2, sequences at the level of the fistulous cavities, thus indirectly determining the presence of blood in movement. In T1 sequencing with gadolinium, these cavities are filled early with contrast, thus confirming the arteriovenous shunt. This phenomenon appeared with total clarity in our case and was key to diagnostic imaging of RAVM.

According to the literature, arteriography shows a series of characteristic signs that allow definitive diagnosis of RAVM, such as the cirsoid tangle, immediate filling of the renal, gonadal and cava veins, and an opacified nephrographic phase[4] , all findings which were present in our case.

Arteriography and subsequent embolization seem at present to be the first choice of treatment. However, the treatment choice varies with clinical symptoms, fistula output and the available facilities at each site. In those asymptomatic cases with low output arteriovenous fistula, observation is the initial approach. Therefore, we will treat cases where there is hemodynamic impact, poorly controlled HT, gradual increase in the size of the fistula and recurrent or persistent hematuria. Surgery is reserved for the cases where the fistula output is high, affecting large vessels (extrarenal fistulas) and in those cases where percutaneous occlusion has failedi[8].

The intravascular approach is a minimally invasive and highly selective option, convenient for the patient and with infrequent complications, thus reducing hospitalization duration and costs. The complications reported in the literature include HT, transient in most cases, and postembolization syndrome[4,7,9], both present in our patient. In high-flow fistulas involving the main vessels, recanalization of the fistula and migration of the embolization coil may occur, causing deep venous thrombosis, pulmonary thromboembolism, skin necrosis and colonic infarction[2,8,10].

In conclusion, in our case MRI was essential for diagnosing RAVM and we consider that the intravascular approach in selected cases constitutes an excellent therapeutic option for the treatment of RAVM.

REFERENCES

1. Varela MC. Aneurisma arteriovenoso de los vasos renales y asistolia consecutiva. Rev Med Lat Am. 1923;14:32-44.
2. Pereira Arias JG, Ullate Jaime V, Pereda Martínez E, Gutiérrez Díez JM, Ateca Díaz-Obregón R, Ramírez Rodríguez MM, et al. Aneurisma cirsoide renal: una rara causa de hematuria severa. Arch Esp Urol. 2007;60(5):589-93.
3. Cabello Padial J, Soler Fernández JM, Murillo Mirat J, Herrera Puerto J, Zapata Blanco J. Fístula arteriovenosa renal congénita tratada mediante embolización. Arch Esp Urol. 1994;47(5):523-5.
4. Domínguez Bravo C, Soler Fernández JM, Poveda Pierola A, Asuar Aydillo S, Laguna Álvarez E, Caballero Gómez M, et al.

Herrera Puerto. Fístulas arteriovenosas renales. Actualización y presentación de un nuevo caso. Actas Urol Esp. 1991;15:46-54.
5. Cho KJ, Stanley JC. Non-neoplasic congenital and acquired renal arteriovenous malformations and fistulas. Radiology. 1978;129:333.
6. Mora M, Machuca J, Cisneros J, Martínez Piñeiro JA. Fístulas arteriovenosas intrarrenales. Arch Esp Urol. 1987;40:12.
7. Crotty KL, Orihuela E, Warren MM. Recent advances in the diagnosis and treatment of renal arteriovenous malformation and fistulas. J Urol. 1993;150:1355-9.
8. Osawa T, Watarai Y, Morita K, Kakizaki H, Nonomura K. Surgery for gigant high-flow renal arteriovenous fistula: experience in one institution. BJU Int. 2006;97:794-8.
9. Sala Barange J, Maroto Genover JM, Rovira Rius JM, Rives Ferriol A, Carretero González P. Fístula arteriovenosa renal como causa de hematuria. Arch Esp Urol. 1987;40(3):143-7.
10. Ramírez Zambrana A, Lavalle Martín F, Zapata Blanco J, Mateos Blanco J, Toledo Serrano M, Molina Suárez JL. Fístula arteriovenosa renal congénita. Actas Urol Esp. 2009;33(6): 696-9.

N. Miranda Utrera*, M. Pamplona Casamayor, F. Guerrero Ramos and O. Leiva Galvis
Department of Urology, Hospital Universitario 12 de Octubre, Madrid, Spain

**Author for correspondence.*
E-mail: natipuchi@hotmail.com
(N. Miranda Utrera).