Clinical case

Female patient, aged 40 years, with a personal history of recurrent cystitis and two vaginal births, a habitual smoker of 10 cigarettes/day came to the Emergency Room due to pain in the right ileal fossa, fever and urination syndrome. An analysis was performed, which revealed leukocytosis with a left deviation (23,436 leukocytes/ul, 93% segmented), 456,000 platelets/ul, renal function within normal range (urea 34.1mg/dl and creatinine 0.9mg/dl), globular sedimentation velocity (GSV) of 42mm/hour within the first hour, C-reactive protein at 34.1 and pyuria in the sediment (50 leukocytes per field), positive nitrite test and bacteria. At that point, a urine culture was extracted that came back positive for Escherichia coli 96 hours later. After extraction of the urine culture, an empirical antibiotic treatment was started with amoxicillin/clavulanic and gentamycine; treatment with both antibiotics was finished, since the antibiogram confirmed that the microorganism was sensitive to them. In the abdominal ultrasound, we see a structure compatible with crossed renal ectopia in the right ileum fossa, which is fused at its upper point with the right kidney, and no kidney in the left renal fossa. With the clinical, analytical and imaging data, the diagnosis of acute pyelonephritis with a crossed ectopic kidney was carried out. An anterograde pyelography was performed, in which both kidneys can be observed fused together and located in the right hemiabdomen (figure 1).

Discussion

Crossed ectopia without or with fusion (90% of all cases) occurs when the ectopic kidney is located on the side opposite to its ureter's insertion point in the bladder. This is a rare congenital defect.[1,2] At times, blood vessels responsible for irrigating the ectopic kidney cross the midline and can be responsible for stenosis of the pyelourethral union of the ectopic kidney or its normal counterpart.[3] Most of these cases evolve asymptomatically and the diagnosis is made when a disease affects the ectopic kidney, such as an infection, lithiasis, tumours or other more infrequent possibilities.[1,2,4] The diagnosis is performed by ultrasound, intravenous urography or anterograde pyelography,[5] isotopic studies, computed tomography or nuclear magnetic resonance imaging. The treatment for this congenital defect merely concerns the pathology affecting the kidney; no other treatment is necessary if the patient is asymptomatic.

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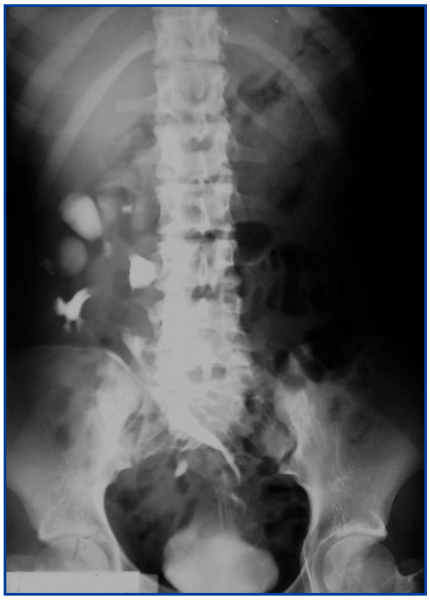

Figure 1. Anterograde pyelography in which we can observe the absence of the kidney from the left hemiabdomen and see the crossed-fused ectopic kidney; note the junction of the ureter that leads to the corresponding left side.

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Jaw claudication: could it be Wegener's granulomatosis?

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Dear Editor:

Only ten cases of jaw claudication (JC) have been described in conjunction with Wegener's Granulomatosis (WG).[1-4] We present a case of WG with a clinical presentation similar to that of temporal (giant cell) arteritis (TA).

A male patient aged 63 years was examined for fever, pain and hardening of the right temporal artery and JC that had been developing over four months, with no other findings from the physical exam. The laboratory showed a GSV of 120mm/hour (normal < 20) with no microhaematuria or kidney failure. Thoracic radiograph was normal. Biopsy of the temporal artery was negative. Having ruled out other pathologies, the profile was interpreted as TA and 60mg methylprednisone/day was administered; the symptoms improved, and the corticosteroids were then gradually reduced. Six months later, the patient was taking 20mg methylprednisone daily and presented constitutional symptoms, as well as epistaxis, bilateral pulmonary nodules with cavitation and microhaematuria, with a nasal biopsy that showed necrotic granulomatous inflammation, which resulted in the diagnosis of WG. Treatment was begun with cyclophosphamide (150mg/day) and methylprednisone (60mg/day) and the symptoms improved. Antineutrophil cytoplasmic antibodies (ANCA) were positive with high titres (240AU, normal < 10) and the ELISA test revealed

specificity for the proteinase 3 antigen. One year after the diagnosis, the patient was asymptomatic.

JC is an ischaemic symptom of fatigue or pain with mastication caused by the narrowing or obstruction of the facial branches of the external carotid (which irrigate muscles used in mastication), and which is present in 45% of patients with TA. Other, less common causes of JC are primary amyloidosis, polyarteritis nodosa (PAN), Churg-Strauss syndrome (CSS), Takayasu's arteritis, GW, hairy cell leukaemia, McArdle's disease, crioglobulinaemia associated with vasculitis, and carotid atherosclerosis.

Compromise of the temporal artery associated with JC has been shown with a very low frequency with PAN, CSS, Takayasu's arteritis, crioglobulinaemia associates with vasculitis, primary amyloidosis and WG.[2] To increase the complexity of the issue, TA may affect the kidneys and lungs just as WG can.[2] In turn, TA can be associated with other types of vasculitis (such as CSS, PAN and WG),[4] with rheumatoid arthritis, primary biliary cirrhosis and neoplasias. The association of WG with other types of vasculitis, such as CSS and AT, has also been described.

It is interesting to recall that among the causes of giant cells in a temporal artery biopsy, we find systemic lupus eritematous, isolated angeitis of the central nervous system, Takayasu's arteritis, and TA. A compromised temporal artery without giant cells has also been described in some cases of systemic vasculitis such as hypersensitivity angeitis, crioglobulinaemia, CSS, WG and PAN.

There are ten patients described in the literature who had WG and an initial clinical profile compatible with TA.[1-4] All of these patients were older than 60 and had JC with or without sudden loss of sight, severe headache with or without double vision, or polymyalgia rheumatica upon diagnosis. The GSV was high at the onset of symptoms in all patients. Biopsy of the temporal artery showed TA in two patients, arteritis without giant cells in four patients, and for the rest it was negative to normal, as with our patient. Within six months, the ten patients developed renal and/or pulmonary lesions characteristic of WG, with typical histologies in the biopsy or positive ANCA.

In summary, we can state that there are five different categories to describe a vasculitis-induced compromised temporal artery, which are: 1) temporal arteritis without giant cells due to multiple entities; 2) temporal arteritis with giant cells, whether caused by TA or not; 3) TA concurrent with WG or other forms of vasculitis; 4) WG with a clinical profile resembling temporal vasculitis but with a negative biopsy (our patient); and 5) TA with clinical characteristics of WG (very uncommon).

Documenting the different histological types of vasculitis that produce similar clinical manifestations emphasises the importance of obtaining a biopsy, whether diagnostic or prognostic, given that treatments may be very different.

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Massive hepatic haematoma in patient on haemodialysis

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Dear Editor:

Patients on periodic HD have an elevated risk of spontaneous haemorrhages or haemorrhages caused by minimal trauma to the retroperitoneal, renal, pericardial, mediastinic and subdural areas. Multiple factors affect predisposition to bleeding, including platelet dysfunction, anti-platelet agents and anticoagulants.[1,2] The development of a subcapsular hepatic haematoma is an exceptional complication that can occur spontaneously or with minimal closed trauma.[3,4] We present a patient aged 77 years who in 2003 was diagnosed with chronic kidney disease second to myeloma kidney (IgG Kappa). Treatment with melfalan/prednisone decreased the circulating paraprotein and improved the medullar affectation. In February 2004, the patient began HD. During the last two years, the patient received two cycles of melfalan/prednisone, and later, bortezomib (Velcade®). He remained stable, with the disease under control and without anaemia with EPO. From April to July 2006, he received conjugated oestrogen (Equin®) (20mg/day) for uraemic thrombopathy. Lately, the patient has been taking aspirin as an anti-platelet agent. On 7 August 2006, eight hours after finishing the HD session, he experienced dizziness and fell, striking his right hypochondrium, and lost consciousness for several minutes. When he was admitted to the hospital he was conscious and aware of his surroundings; he complained of pain in his right scapula. Blood pressure was 55/35, cardiac frequency 72l/min and he showed sinus rhythm. Analytical tests showed: haematocrit 19%, haemoglobin 6.9g/dl, 300,000 platelets/mm^3, 10,000