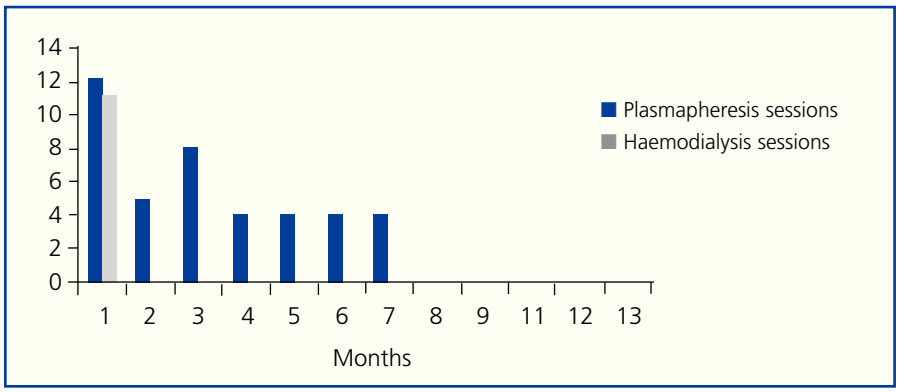

Figure 1. Number of haemodialysis and plasmapheresis sessions required during seven months (after three months, patient is re-admitted due to worsening profile).

Evolution: thrombotic microangiopathy was detected from the renal biopsy. Eight plasmapheresis sessions were administered in 17 days and the diuresis recovered progressively, although haemodialysis was still necessary during the first month. The patient was discharged and the plasmapheresis sessions became less frequent (figure 1).

Three months later, the patient was admitted again due to decreased renal function associated with abdominalgia, choroidal ischaemia and positive antiphospholipid antibody titres. The profile was interpreted as a relapse of the disease and plasmapheresis sessions were started again and administered during four months, which led to improvement in renal function and the ocular condition.

One year later, the platelets stabilised, the creatinine descended slowly (figure 2), and the antiphospholipid antibody titres were normal; there was no new thrombotic events.

The treatment of choice for primary APS is not well-defined[2] and varies according to the clinical presentation. Some experts recommend high doses of anticoagulants,[3] while others support the use of antiplatelet drugs or prophylactic anticoagulants in low doses. Other less successful treatments have used immunosuppressors and corticosteroids.[3] For patients on anticoagulants who suffer thrombotic events that severely affect the kidneys and/or other organs, plasmapheresis is an option,[4] as it was in our case.

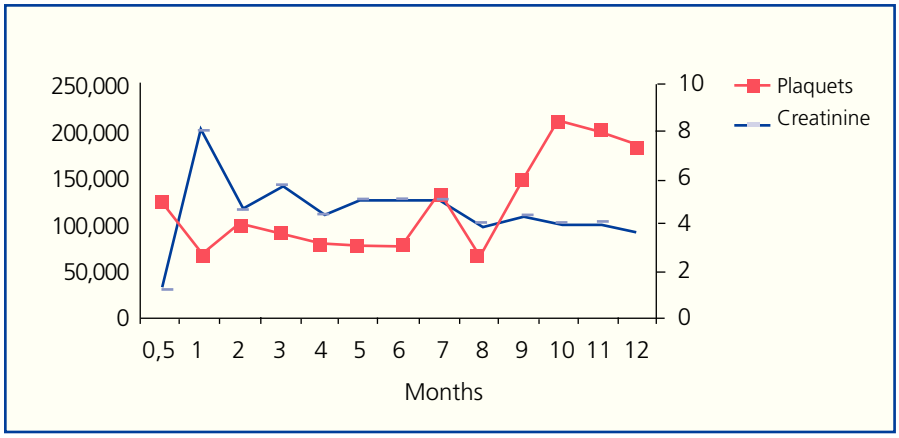

Figure 2. Development of the platelets and serum creatinine during the 12 months. A slow and progressive decrease in the creatinine coinciding with a platelet increase was observed.

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Pyelonephritis in crossed-fused renal ectopia

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Dear Editor:

Crossed-fused renal ectopia is the second most common variety of renal fusion, with an incidence of 0.01% in the general population. There are at least six varieties of crossed-fused renal ectopia, and it is thought to be produced by a change in the migration of the kidney due to a vascular obstacle, or due to genetic or teratogenic factors. It is generally associated with other alterations of the gastrointestinal and locomotor systems.

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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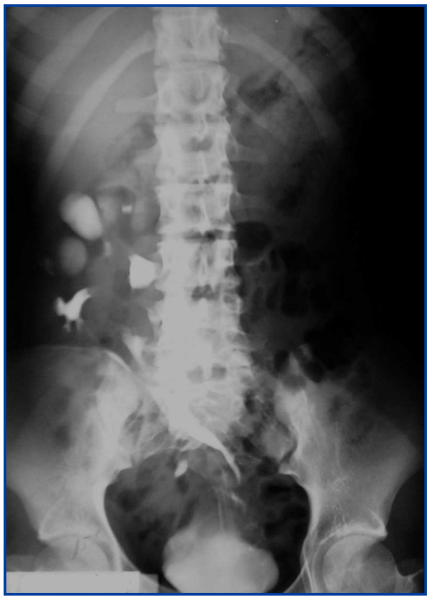

Figure 1. Anterograde pyelography in which we can observe the absence of the kidney from the left hemiabdomen and see the crossed-fused ectopic kidney; note the junction of the ureter that leads to the corresponding left side.

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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