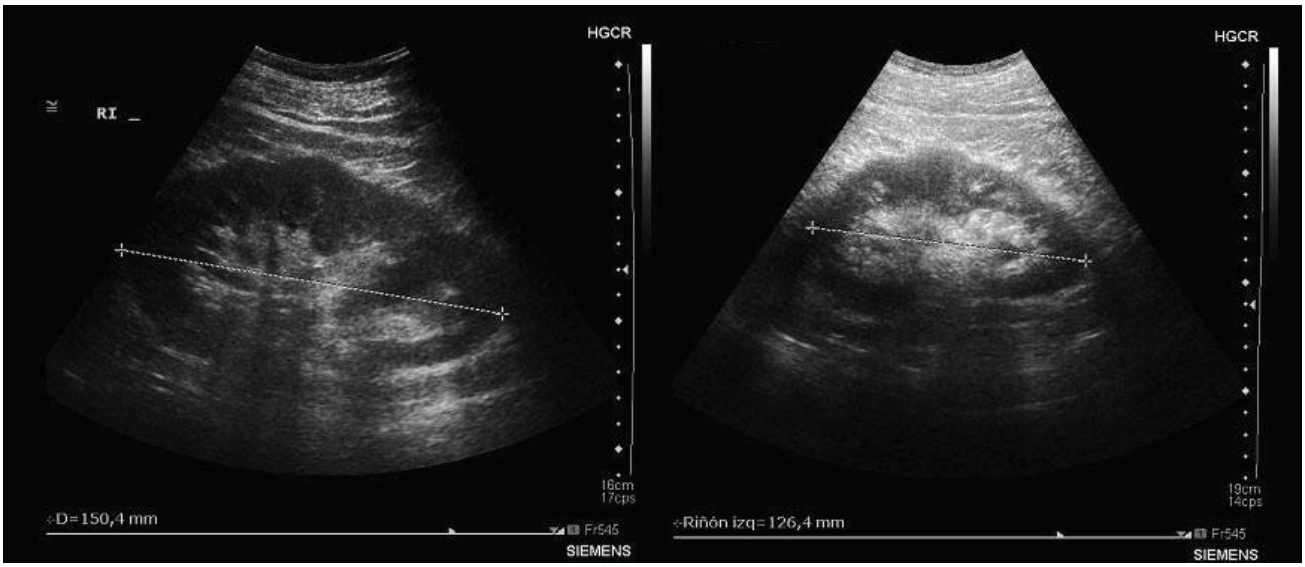

Figure 1. The image at right shows left nephromegaly, while the image at right shows the normal size of the kidney following treatment.

DISCUSSION

The quinolones are broad spectrum antibiotics that are easy to use and which possess absorption and bioavailability characteristics that make them one of the most widely used antibiotic groups. In this group it has been shown that ciprofloxacin[3] is able to cause acute tubulointerstitial nephritis, and there are also isolated reports implicating the rest of the drugs belonging to this same group. Levofloxacin is a third-generation quinolone with a broad spectrum of action and with the same side effects as the rest of the quinolones. Its association to acute tubulointerstitial nephritis is very infrequent; a Medline search spanning the period between 1998 and March 2008 revealed only four cases of renal failure induced by levofloxacin[4-7], and none of them were accompanied by nephromegaly.

Our case represented a diagnostic challenge due to the suspicion of acute pyelonephritis during the entire clinical course. Only in the light of the torpid evolution of events did we attempt to rule out other possible etiologies characterized by large kidney, renal failure and fever. Thus, having discarded hereditary causes of nephromegaly, we evaluated non-hereditary disorders such as amyloidosis, Gaucher's disease, mycoses, tuberculosis, AIDS, renal oncocytomatosis, angiofollicular ganglionic hyperplasia, myeloma, primary renal lymphoma, secondary renal lymphoma and acute leukemia.[8] After ruling out some of these etiologies from the start, we examined the more plausible possibilities such as lymphoma, leukemia, myeloma, tuberculosis and amyloidosis. Since positive results were not obtained, a renal biopsy was decided, which revealed the above mentioned alterations. After corticoid therapy with initial doses of 60 mg/kg of prednisone followed by slow withdrawal, kidney function was seen to normalize, with a reduction in kidney size (right 12.6 cm and left 13 cm), on occasion of the last ultrasound control (fig. 1).

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Emphysematous pyelonephritis in peritoneal dialysis

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To the editor: Emphysematous pyelonephritis is a serious disorder that mainly affects diabetic patients.

We present a case of torpid emphysematous pyelonephritis.

A 72-year-old patient presented with a history of chronic liver disease (of probable alcoholic origin) with occasional edematous decompensation and several digestive bleeding episodes, colon diverticuli, pericardiectomy due to constrictive pericarditis, and chronic renal failure (of indeterminate origin) with the start of peritoneal dialysis in 2003.

The patient was admitted in October 2007 due to sepsis of urinary origin – the causal microorganism being *E. coli*.

During admission, abdominal ultrasound showed small kidneys, with no other anomalies. Treatment was provided in the form of amoxicillin-clavulanate for three weeks, the patient remaining at home without clinical symptoms.

In December 2007 he was admitted to the Service of Nephrology due to pain in the left inguinal region and leukocytosis (35,000). The patient was without fever, hemodynamically stable and presented no other symptoms. Exploration of the affected zone revealed no alterations other than pain in response to pressure. Empirical antibiotic treatment was started with ciprofloxacin, and emergency ultrasound of the inguinal region revealed the presence of an abscess measuring 3 cm in size. After 24 hours, the patient presented abdominal pain and turbid dialysis fluid. An emergency abdominal CAT scan was thus decided, revealing (fig. 1) a water-fluid level accumulation in the retroperitoneum and left renal fossa, and abundant gas bubbles occupying the region of the psoas muscle, which appeared atrophic. The accumulation was seen to descend between the muscles of the root of the left thigh, with an abundant presence of gas.

In view of the CAT findings, emergency surgery was decided under broad-spectrum antibiotic coverage (piperacillin-tazobactam plus metronidazole), performing a left lumbotomy and drainage of the abscess. The peritoneal dialysis catheter was removed, and the patient was kept under continuous venous-venous hemodiafiltration.

The urine and peritoneal dialysis fluid cultures proved positive for *E. coli* sensitive to the previously administered antibiotic treatment.

Six days later, and in view of clinical worsening of the patient, repeat surgery was carried out to remove the left kidney and perform retroperitoneal debridement.

However, deterioration was progressive, and the patient died after 24 hours.

Emphysematous pyelonephritis is an infrequent acute disease characterized by the presence of gas within the renal parenchyma, and presents a high percentage mortality.

The condition is generally unilateral and affects patients with known risk factors, such as diabetes mellitus or urinary tract obstruction – though it has also been reported that the use of dextrose in the peritoneal dialysis fluid may be a risk factor for infections of this kind.[1]

In most cases the isolated germ corresponds to *E. coli*.[2]

Treatment is the subject of controversy. Percutaneous drainage should be considered initially, and particularly in critically ill patients. With such treatment a lesser percentage mortality has been reported compared with emergency nephrectomy. However, subsequent kidney removal may prove necessary in some cases.[3,4]

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Usefulness of prolonged haemodialysis in acute methanol poisoning

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To the editor: The case of a patient with severe methanol and toluene poisoning has been recently published in Nefrología.[1] We have treated a patient with severe methanol poisoning and we want to emphasize the importance of prolonged haemodialysis in its treatment.

CASE REPORT

This was a 40-year-old male patient who was brought into the Emergency Department after suffering frfom severe headache, dizziness, irritability and incoherent language. According to the family, the patient had chronic alcoholism although he had not had any alcohol in the last 3 days. In the emergency room, there was deterioration in level of consciousness with progressive coma, so the patient was admitted to the ICU where orotracheal intubation, and mechanical ventilation was used.

Laboratory analysis on admission showed an arterial blood gas with a pH of 6.98, PO_2 96, PCO_2 31, and bicarbonate 4.9 mEq/l. Plasma creatinine was 1/53 mg/dl, BUN 33 mg/dl, sodium 135 mEq/l, potassium 6.2 mEq/l, chloride 102 mEq/l, haemoglobin 16 g/dl, glucose 198 mg/dl, serum osmolarity 421 mOsm/kg and lactic acid 10.5 mmol/l. A head CT did not reveal any visible parenchymal or signs of cerebral haemorrhage.

After questioning the family again, the possibility that the patient had consumed approximately half a litre of methanol was mentioned.

Treatment was initiated with pyridoxine, thiamine, nalaxone, tiapride, flumazenil, sodium bicarbonate, inotropics and intravenous ethanol.

Haemodialysis began approximately 3 hours after admission to ICU. A high-