

Letters to the Editor

Diarrhea and malnutrition in kidney transplant recipient: A case of infection by cryptosporidiosis[☆]

Diarrea y desnutrición en paciente trasplantado renal: un caso de infección por criptosporidiosis

Dear Editor,

Cryptosporidiosis is an opportunistic infection, which causes diarrhea in renal transplant patients. Without an appropriate treatment, it may produce a serious electrolyte and water disorder.

Here we present a clinical case of a 57 y.o. male with CKD secondary to IgA nephropathy. He had been on regular hemodialysis for 17 months. He received a cadaveric renal transplant without postoperative complications and maintained serum creatinine levels in the 2.9–3.5 mg/dl range. The treatment for immunosuppression included: prednisone (5 mg/24 h), calcineurin inhibitors and mycophenolate mofetil.

Eleven months later, the patient is admitted to the hospital because watery diarrhea (8–10 times per day) of 12–15 day duration, diffuse abdominal discomfort and weight loss.

The physical exam shows abdomen without pain during palpation and increased peristalsis. Blood biochemistry revealed a worsening of renal function together with acidemia due to metabolic acidosis and a low value of total protein (3.7 mg/dl) indicating malnourishment.

Stool culture for bacteria was negative; *Clostridium difficile* toxin was also negative; CMV, rotavirus and adenovirus antigens were negative. Finally a stool parasites exam reveals the presence of *Cryptosporidium*. Paromomycin 700 mg trice daily was started and maintained for 2 weeks. The patient improved clinically. The diarrhea disappeared and renal function returned to baseline.

Cryptosporidiosis is an opportunistic infection produced by the parasite *Cryptosporidium*, an intracellular pathogen with more than 35 species. Most of the infections in humans¹ are produced by the subtypes *C. parvum* and *C. hominis*.

The prevalence of this disease in Europe is 1–2%, in North America is 0.6–5% and it is as high as 20% in Asia Africa and South America.²

The first case of this infection was published in 1972, and 7 years later Wiesburger³ presented the first transplanted patient with Cryptosporidiosis. Presently it is a rare case of diarrhea in renal transplant patients.

As observed in our patient, the main symptom is diarrhea that if became persistent may cause malabsorption with malnourishment.⁴

To make the diagnosis it is required to demonstrate the presence of the parasite in tissues or fluids, the most common method is the Ziehl-Neelsen staining.^{2,4,5} Other approaches such as colonoscopy have lower efficacy although sometimes help to reach the diagnosis.⁵

Regarding the treatment the first choice is Paromomycin or Spiramycin. In our case it demonstrated to be effective with disappearance of symptoms and normalization of biochemistry. Also Nitazoxanide for 5–21 days has been shown to eradicate the parasite.⁶

In conclusion, Cryptosporidiosis is rare in transplanted patients but given the state of immunosuppression these patients are at risk of these type of infections.⁵ Therefore Cryptosporidiosis should be included in the differential diagnosis of diarrhea mainly if it is associated to clinical and biochemical signs of malnourishment.

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Cortical necrosis: An uncommon cause of acute renal failure with a very poor outcome[☆]

La necrosis cortical: una causa infrecuente de fracaso renal agudo pero con un pésimo pronóstico

Renal cortical necrosis (RCN) was first described in 1883 by Friedlander. It is an extremely rare cause of acute kidney injury and usually is irreversible.¹ It is more prevalent in developing countries with deficient health systems. Usually there is extensive involvement of the cortex, but sometimes it is localized affecting focal areas. Renal cortical necrosis is observed in 2 peaks of age; first is in early childhood due to perinatal factors, and the second in women of reproductive age due to obstetric causes. Of the non-obstetric causes in the adult population, the most frequent etiology is septic shock.²

Here we present a case of a female, 21 y.o. from Venezuela with no previous relevant medical history who came to the emergency room for a 24 h history of fever, headache, blurred vision and photophobia. Physical examination showed neck stiffness and petechia in upper extremities and abdomen. Cranial CT scan without relevant findings. Spinal tap showed biochemical changes compatible with bacterial meningitis. The severity of the clinical symptoms prompted admission to ICU. She developed a Waterhouse–Friedrichsen syndrome

with multiorgan involvement. Blood cultures demonstrate the presence of *Neisseria meningitidis* group B.

The patient developed acute renal failure that required renal replacement therapy with hemofiltration. At that time a possible cause of the acute renal failure was immune allergic interstitial nephritis because eosinophilia was present that may have been related with the antibiotic therapy; therefore, steroid were given but without response. It was observed that the urine sediment had a large number of dysmorphic red blood cells, red cell casts, proteinuria of 0.9 g/day and low circulating levels of complement (C3 50.9 mg/dl and C4 11.6 mg/dl). Blood tests of autoimmunity and serology were negatives. During ICU admission the patient had low urine output and required RRT.

After clinical improvement the patient was transferred to the nephrology ward and a renal biopsy was obtained; there were 20 glomeruli all of them obsolete with multiple foci of cortical necrosis of ischemic origin that did not involve the subcapsular area and did not extend to the deeper areas of the kidney (Fig. 1A). Furthermore. There

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