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Case Report

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IG A NEPHROPATHY AND DIGESTIVE HYDATIDOSIS: ABOUT A CASE

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ABSTRACT

Introduction: Ig A nephropathy is a glomerulonephritis characterized by a nephrotic syndrome and hematuria with histological evidence of Ig A mesangial deposits. It can be primary or secondary to infections including parasites. We illustrate through this clinical case the renal impairment probably secondary to hydatid disease. **Clinical observation:** Mr C. A 29 year old is followed for hepatic, splenic and peritoneal hydatidosis. He is admitted to the nephrology department for lower limb oedema, ascites with weight gain of 30 kg. Blood pressure is 130/90mmHg. The rest of the somatic examination is normal. Biologically, there is a nephrotic syndrome with acute renal failure with preserved diuresis (serum creatinine 400 umol/l), microscopic hematuria and leukocyturia. Renal biopsy revealed IgA nephropathy. Treatment is symptomatic based on water depletion by loop diuretics and ultrafiltration in hemodialysis and etiologic based on treatment of hydatidosis with Albendazole. The evolution is marked by the regression of the nephrotic syndrome but persistence of moderate chronic renal insufficiency with a serum creatinine around 180 mmol/l. **Conclusion**: IgA nephropathy secondary to hydatid disease is a serious form that can progress to end-stage renal disease, hence the importance of rapid management of the infection.

KEYWORDS: IgA nephropathy, digestive hydatidosis; nephrotic syndrome.

INTRODUCTION

Ig A nephropathy (IgA N) is a glomerulonephritis characterized by mesangial deposits of Ig A. Its prevalence in the general population is 1.9 per 1,000 population.[1] It is the most common cause of glomerulonephritis.[2] and can be primary or secondary to other parasitic infections. We illustrate through this clinical case an IgA N secondary to hydatid disease.

OBSERVATION

Mr C.A., 29 years old, followed for hepatic, splenic and peritoneal hydatidosis and treated with Albendazole 400 mg twice a day.

He is admitted to nephrology for lower limb oedema, ascites with weight gain of 30 kg. Blood pressure is 130/90mmHg. The rest of the somatic examination is normal.

Biologically, there is a nephrotic syndrome with acute renal failure with preserved diuresis (serum creatinine 400 umol/l), microscopic hematuria and leukocyturia. The serum complement is normal, and the hemogram shows a deep anemia (hemoglobin at 3 g/dl) normochrom normocyte agenerative, without martial deficiency, without hemolysis.

Renal biopsy revealed an N.IgA.
In addition to Albendazol, the patient received symptomatic treatment with loop diuretic, converting enzyme inhibitor and hemodialysis with red blood cell transfusions.

The evolution is marked by regression of the nephrotic syndrome but persistence of moderate chronic renal failure with a blood creatinine of about 180mmol/l, corresponding to a creatinine clearance according to the MDRD formula at 40 ml/min.

DISCUSSION

Acute glomerulonephritis with mesangial IgA or IgA N deposits accounts for 17% of infection-related glomerulonephritis.[3] and may occur at a distance from the initial infection episode or at its resolution.[4]

It was first described in 2003, in diabetic patients, in the context of staphylococcal skin infection.[5] But other forms have been described in certain parasitoses, such as malaria, schistosomiasis and filariasis.[6-8] hydatid disease (echinococcosis) is a cyclo-zoonotic infection with the dog tapeworm. The liver and lungs are the two organs most frequently involved. The kidney is rarely affected: 2% of cases according to the Australian Hydatid registry which includes 1802 cases.[9]

Echinococcus Granulosus-related nephropathies reported are: IgA nephropathy.[10] extra membranous glomerulonephritis,[11] membranoproliferative glomerulonephritis.[12] and amyloidosis.[13-15]

The role of hydatid antigen in the pathogenesis of glomerulonephritis has been demonstrated, with the host tending to eliminate the parasite by innate immune mechanisms, which mainly include phagocytosis, induction of NK lymphocytes and complement activation via the alternate pathway.[16,17] Monocyte activation also results in a cascade of acquired immune responses initiated primarily by T. helper cell activation. This pathway is controlled by interleukins released by monocytes, including IL-1, IL-6 and IL-12.[13,15]

During IgA N, the Ig A antibodies normally produced to destroy the parasite show an aberrant glycosylation which modifies their physicochemical properties. They tend to bind to the mesangial cells of the renal glomerulus.

Cell-mediated immune responses play a fundamental role in the pathophysiology of the disease. Indeed, hypercellularity in the glomerular compartment, as well as in the interstitial compartment, is a common feature of many glomerulonephritis cases. These are mainly macrophages and T cells and their number is believed to be directly related to the clinical severity of the disease.[21] Several studies have shown that T cells initiate and amplify the immune response with a significant impact on the anatomopathology of the glomerulus. The same is true for macrophages, which alone are capable of inducing significant damage to the kidney tissue, such as mesangial proliferation and proteinuria, via the classic mechanisms of inflammation.[20]

The treatment of IgA N secondary to hydatid disease is based on antiparasitic treatment while the place of corticosteroid therapy remains highly controversial in this context.[21] The evolution of renal function is unfavourable with 30 to 80% of end-stage renal failure at the end of follow-up.[22]

Our patient was treated with Albendazol in addition to symptomatic treatment with regression of the nephrotic syndrome and persistence of moderate renal failure.

CONCLUSION

Multiple hydatidosis is less frequent but remains serious due to its localization, hence the interest of primary prevention to avoid its occurrence.

IgA N secondary to hydatid disease is a serious form and can progress to end-stage renal disease, hence the importance of rapid management of the infection.

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