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Association of Membranous Nephropathy and Tubulointerstitial Nephritis with Hepatic Hydatidosis: Case Report

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Abstract **Case Report**

Hydatid disease is an anthropozoonosis due to development of Echinococcus larva in the human host. This parasitic infection causes rarely a renal damage, that's why only a few cases of glomerular or tubulointerstitial nephropathies linked to hepatic or pulmonary hydatic cysts, have been reported during the last three decades. We report a singular case of membranous nephropathy and tubulointerstitial nephritis associated with a hepatic hydatidosis.

Keywords: membranous nephropathy, hydatid disease, tubulointerstitial nephritis, glomerulonephritis.

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INTRODUCTION

Hydatid disease or Echinococcosis is a parasitic infestation caused by a tapeworm of the genus Echinococcus. The 2 most common types being E. granulosis and E. multilocularis.

It's a disease that has a worldwide distribution, but it's particularly endemic in the tropical and mediterranean basin's countries.

The liver and lungs are the most frequently involved organs. Rarely occurrences of primary cysts in kidneys have been reported, with the consequent finding of jellylike hydatid material in the urine [1]

Renal hydatid disease is well defined, and many studies are present in the literature [2], but renal injury without direct renal invasion is a less well-known subject.

There are only a few reported cases in the literature, and no investigational study revealing renal involvement as proven by biopsy.

Furthermore, only a few cases of glomerular lesions associated with hydatid disease have been described.

We report a rare case of membranous nephropathy diagnosed in a context of multiple hepatic hydatid cysts.

CASE REPORT

37 years old male patient, with no particular medical history, had been suffering from recurrent headaches and visual blur for nearly a year, these symptoms worsened in the last 2 weeks preceding his admission.

At presentation to hospital, his blood pressure was 200/130 mmHg at both arms, heart rate 105 bpm, afebrile.

Physical examination showed edema of extremities.

Fundoscopic examination revealed hypertensive retinopathy stage III.

On dipstick, urine was positive for Albumin 3+, red cells +, glucose 2+ and negative for aceton -, nit -, ph :6

The laboratory data showed: 3.5 g excretion of urinary protein in 24 hours, severe renal failure; blood urea nitrogen was 86 mg/dl (30.7 mmol/l), serum creatinine 4.2 mg/dl (371 µmol/l), the liver function was normal except for that serum albumin level that was

significantly low with a value of 1.8 g/dl, total protein was 4.3 g/dl. The serum cholesterol level was 165 mg/dl.

Blood count was: Hemoglobin 12.5 g/dl with normochromic, normocytic formula with no evidence of hemolysis, leukocyte count of 10100/mm3 C-reactive protein was 3.

Autoantibody screen (antinuclear and antineutrophil cytoplasmic) was negative. The complement levels C3 and C4 were normal.

Chest X-ray showed no sign of infection or pulmonary edema nor pulmonary vascular congestion.

Renal ultrasound displayed normal size and moderately differentiated kidneys, without calyx dilation.

Abdominal ultrasound uncovered 2 well-defined cystic lesions of the liver compatible with type 3 hydatid cyst of Gharbi and al classification system [3] measuring respectively 5 x 4,8 cm and 5x 4,2 cm, a moderate ascites and no organomegaly.

A better characterization was obtained by computed tomography (CT) scan of the abdomen that showed normal-sized liver, with regular contours presenting 2 multi- compartmental, partially calcified cystic formations at segment IV (43x38 mm) and segment VI (50x40 mm) of the liver *(*Figure 1).

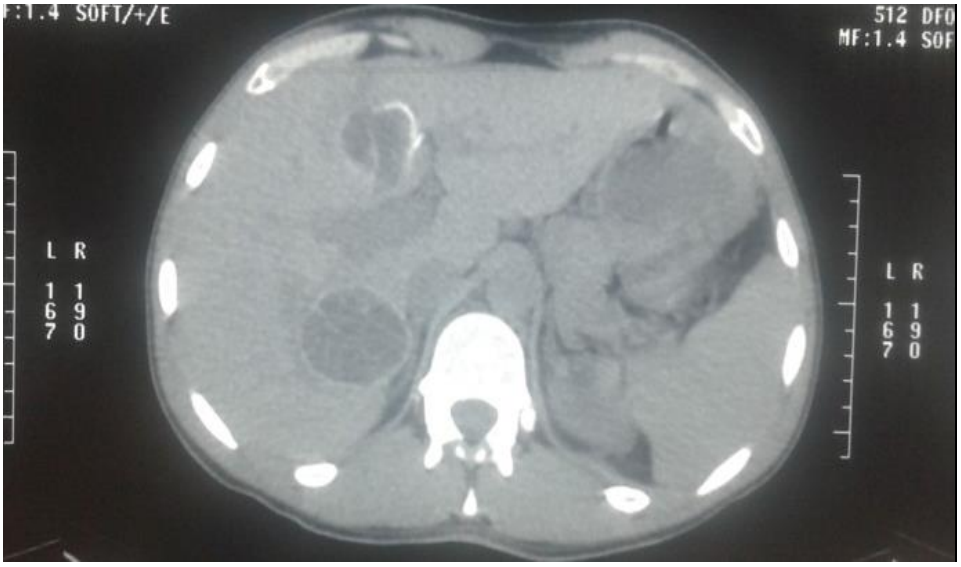

Figure 1: Abdominal tomography demonstrates 2 cystic lesions with areas of rim calcification measuring respectively (43x38 mm) and (50x40 mm)

Serology for Echinococcus IgM was negative, while IgG was positive testifying of an old parasitic infection.

A broad serodiagnostic was used, not permitting differentiation between E. granulosis and E. multilocularis, the parasitoses couldn't be differentiated unambiguously by the use of crude echinococcal antigen extract.

Thus on the basis of clinical, biological and radiological findings, the patient was diagnosed to have a hydatid disease of the liver.

The patient was subjected to renal biopsy as it was a nephrotic presentation. Renal biopsy revealed**,** thickened and prominent capillary loops with normal cellularity (figure 2).

Besides tubular atrophy and interstitial fibrosis was affecting nearly 50% of the biopsy.

IgG and C3 collected in the basement membrane and appeared in a diffuse granular pattern by immunofluorescence (figure 3) Which gives us a histopathologic aspect compatible with membranous glomerulonephritis stage II.

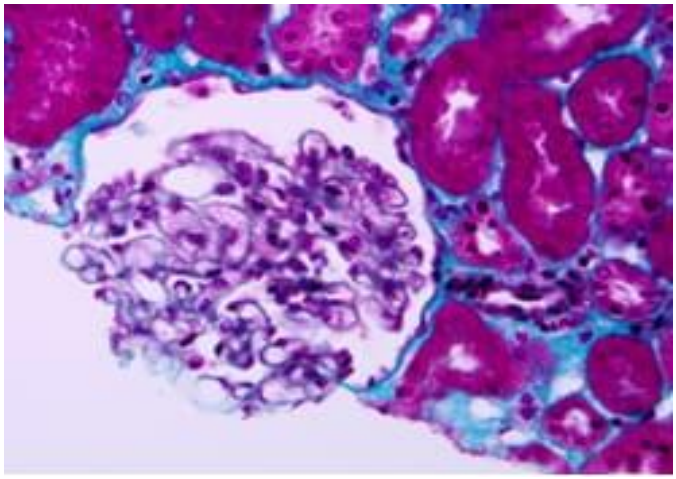

Figure 2: Renal biopsy: light microscopy

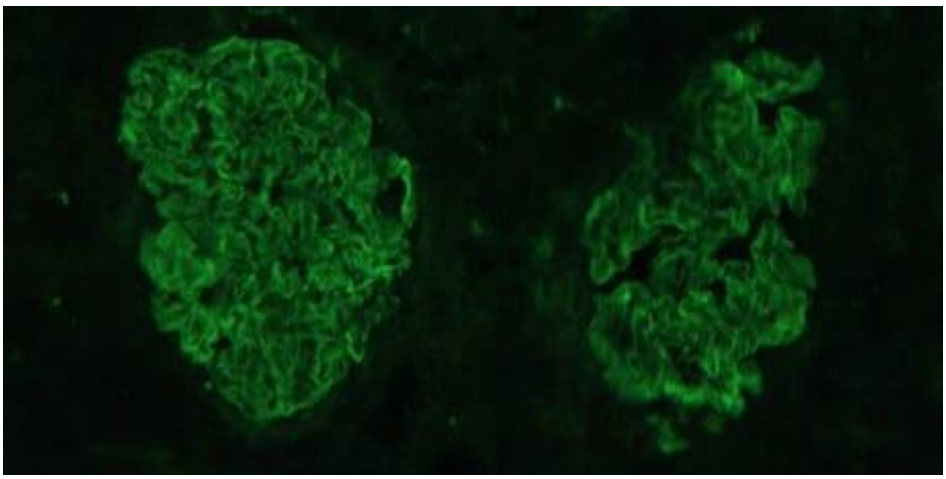

Figure 3: Renal biopsy: Immunofluorescence: IgG andC3 deposit

Anti PLA2R antibody was negative comforting the diagnosis of secondary membranous nephropathy.

Treatment was started with antihypertensive therapy to control hypertension and diuretics for depletion.

The renin-angiotensin system blockers were initiated after clinical stabilization.

The patient was also initiated with Albendazole 400 mg twice a day, for a total period of twelve weeks.

He was never treated with steroids or any other immunosuppressant.

After concerting with our surgeon, no hepatic cyst resection was done, due to the relatively small dimension of the cyst and the absence of clinical or biological impact.

Follow-up after one year: Serum creatinine showed no sign of improvement, consequently we started preparation for renal replacement therapy.

Urine proteinuria decreased significantly thanks to medication. Liver scanography lesions remained stable.

DISCUSSION

Echinococcosis, along with other parasitic infections lead to significant morbidity and mortality, especially in tropical regions.

Renal involvement in the course of such parasitosis occurs in three forms: acute renal injury caused by the systemic effects of severe infection, physical invasion by the parasite and renal injury caused by the host– parasite immune interaction [4].

Altough physical invasion by the parasite is the most common form, we can find few reports of immune mediated glomerular injury, in form of mesangioproliferative nephritis [5–7], minimal change disease [8], IgA nephropathy [9], and membranous glomerulopathy [10].

We presented a case of membranous nephropathy associated to hydatidosis as we found similar cases to be scarce in literature.

In 1974, Miatello and colleagues [11] were the first to describe a patient in whom nephrotic syndrome disappeared following the excision of a pulmonary hydatid cyst.

In 1981, each one of Vialtel and colleagues [12] and Sánchez Ibarrola and colleagues [10] reported a case of membranous nephropathy secondary to hydatid cyst. Both the cases had resolved by surgical resection of the cyst.

In 1992, Edelweiss and collegues reported a case of Hepatic hydatidosis with glomerular involvement which consisted of grade I membranous glomerulonephritis [13], prior to this the same team had been successful in showing echinococcal antigen and corresponding antibody in the glomeruli of sheep by immunoperoxidase studies [14].

In 2010, Altay and colleagues [15] conducted a study that included 80 patients diagnosed with liver ecchinococcus that consented to a renal biopsy; only one case has demonstrated membranous nephropathy, confirming again the scarcity of the association.

Most cases of glomerular lesions associated with hydatid disease are reported to be reversible by treating the infection [7,12].

However, in our case, the relatively small size of the two liver cysts, the presenceof calcifications testifying to the chronicity of those cysts, the absence of any form of clinical impact and especially the predominance of tubulo-interstitial fibrosis in the renal biopsy, encouraged us to have a non invasive approach by not opting for the surgery and privileging medical treatement.

Since our patient had no history of systemic or immunoallergic diseases nor acute or chronic infection, we thought that TIN (tubulointerstitial nephropathy) was probably consequence to the hydatid disease.

In 2009, Feroz A. and al [16] described a case of hydatid cyst disease with predominant tubulointerstital nephropathy which we found it to be the only report that showcases such association.

Besides, parasite-associated interstitial nephritis had been described for a few parasites, Leishmania donovani and Schistosoma mekongi [17,18].

Monocytes and lymphocytes infiltrate the renal interstitium due to acute inflammation [17]. Acute tubular necrosis is seen with some parasitic infections, and it is associated with acute interstitial nephritis [4].

The main pathologic mechanism of tubular necrosis described for malaria is massive monocyte activation [19]. The effect of parasitic agent on the monocytes leads to a cascade of mediator release such as septic shock. This leads to peripheral blood pooling, reduction of the effective blood volume and haemoconcentration.

Diminished renal perfusion becomes exaggerated.

We suggest that a similar mechanism may be responsible for tubule interstitial lesions in echinococcosis.

CONCLUSION

We recommend that urinalysis and, if indicated, renal biopsy should be performed for patients diagnosed with hepatic hydatid disease to track eventual renal involvement.

In fact, membranous glomerulopathy or other glomerulonephritis may develop during the course of echinococcus infection as it had been proved before [10] although it is a rare association.

More interestingly, ecchinococcusis is likely also responsible for tubulo-interstitial nephropathy, but a broader study is needed to adress the causality.

Disclosure: The authors report no conflicts of interest in this work

REFERENCES

1. Musacchio, F., & Mitchell, N. (1966). Primary renal echinococcosis: a case report.
2. Gilsanz, V., Lozano, G., & Jimenez, J. (1980). Renal hydatid cysts: communicating with collecting system. *American Journal of Roentgenology*, *135*(2), 357-361.
3. Gharbi, H. A., Hassine, W., Brauner, M. W., & Dupuch, K. (1981). Ultrasound examination of the hydatic liver. *Radiology*, *139*(2), 459-463.
4. Barsoum, R. S. (1999). Tropical parasitic nephropathies. *Nephrology, dialysis, transplantation: official publication of the European Dialysis and Transplant Association-European Renal Association*, *14*(suppl_3), 79-91.
5. Okelo, G. B. A. (1988). Mesangial proliferative glomerulonephritis in a patient with hepatic hydatid cysts: a case report in an African male.
6. Rincón, B., Bernis, C., Garcia, A., & Traver, J. A. (1993). Mesangiocapillary glomerulonephritis associated with hydatid disease.
7. Covic, A., Mititiuc, I., Caruntu, L., & Goldsmith, D. J. A. (1996). Reversible nephrotic syndrome due to mesangiocapillary glomerulonephritis secondary to hepatic hydatid disease. *Nephrology Dialysis Transplantation*, *11*(10), 2074-2076.
8. Gelman, R., Brook, G., Green, J., Ben-Itzhak, O., & Nakhoul, F. (2000). Minimal change

glomerulonephritis associated with hydatid disease. *Clinical nephrology*, *53*(2), 152-155.

9. Des Grottes, J. M., Oana-Cahoolessur, M., Neve, P., & Vanhaeverbeek, M. (1993). Mediastinal and pulmonary hydatidosis, bronchocentric granulomatosis and IgA glomerulonephritis. *Acta Clinica Belgica*, *48*(5), 338-343.
10. Ibarrola, A. S., Sobrini, B., Guisantes, J., Pardo, J., Diez, J., Monfa, J. M., & Purroy, A. (1981). Membranous glomerulonephritis secondary to hydatid disease. *The American Journal of Medicine*, *70*(2), 311-315.
11. Miatello, V. R., & Zanetti, N. L. (1974). Disappearance of a nephrotic syndrome following the excision of a pulmonary hydatic cyst. *Medicina*, *34*(5), 532-538.
12. Vialtel, P., Chenais, F., Desgeorges, P., Couderc, P., Micouin, C., & Cordonnier, D. (1981). Membranous nephropathy associated with hydatid disease.
13. Edelweiss, M. I., Daudt, H. M., Goldstein, H. F., & Garcia, C. (1992). Hepatic hydatidosis with glomerular involvement: report of a case. *Revista da Associacao Medica Brasileira (1992)*, *38*(1), 31-32.
14. Edelweiss, M. I. A., & Lizardo-Daudt, H. M. (1991). Naturally existing model of glomerulonephritis mediated by immune complexes associated with hydatidosis in sheep. *Nephron*, *57*(2), 253-254.
15. Altay, M., Ünverdi, S., Altay, F. A., Çeri, M., Akay, H., Özer, H., ... & Duranay, M. (2010). Renal injury due to hepatic hydatid disease. *Nephrology Dialysis Transplantation*, *25*(8), 2611-2615.
16. Aziz, F., Pandya, T., Patel, H. V., Ramakrishna, P., Goplani, K. R., Gumber, M., ... & Trivedi, H. L. (2009). Nephrotic presentation in hydatid cyst disease with predominant tubulointerstital disease. *International Journal of Nephrology and Renovascular Disease*, 23-26.
17. Dutra, M., Martinelli, R., de Carvalho, E. M., Rodrigues, L. E., Brito, E., & Rocha, H. (1985). Renal involvement in visceral leishmaniasis. *American Journal of Kidney Diseases*, *6*(1), 22-27.
18. Cheever, A. W. (1985). A review: Schistosoma japonicum: the pathology of experimental infection. *Experimental parasitology*, *59*(1), 1-11.
19. Clark, I. A. (1982). Suggested importance of monokines in pathophysiology of endotoxin shock and malaria. *Klinische Wochenschrift*, *60*, 756-758.
20. Musacchio, F., & Mitchell, N. (1966). Primary renal echinococcosis: a case report.
21. Gilsanz, V., Lozano, G., & Jimenez, J. (1980). Renal hydatid cysts: communicating with collecting system. *American Journal of Roentgenology*, *135*(2), 357-361.
22. Gharbi, H. A., Hassine, W., Brauner, M. W., & Dupuch, K. (1981). Ultrasound examination of the hydatic liver. *Radiology*, *139*(2), 459-463.
23. Barsoum, R. S. (1999). Tropical parasitic nephropathies. *Nephrology, dialysis, transplantation: official publication of the European Dialysis and Transplant Association-European Renal Association*, *14*(suppl_3), 79-91.
24. Okelo, G. B. A. (1988). Mesangial proliferative glomerulonephritis in a patient with hepatic hydatid cysts: a case report in an African male.
25. Rincón, B., Bernis, C., Garcia, A., & Traver, J. A. (1993). Mesangiocapillary glomerulonephritis associated with hydatid disease.
26. Covic, A., Mititiuc, I., Caruntu, L., & Goldsmith, D. J. A. (1996). Reversible nephrotic syndrome due to mesangiocapillary glomerulonephritis secondary to hepatic hydatid disease. *Nephrology Dialysis Transplantation*, *11*(10), 2074-2076.
27. Gelman, R., Brook, G., Green, J., Ben-Itzhak, O., & Nakhoul, F. (2000). Minimal change glomerulonephritis associated with hydatid disease. *Clinical nephrology*, *53*(2), 152-155.