



(CASE REPORT)



Segmental and focal hyalinosis, schistosomiasis: Association or coincidence?

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Abstract

Schistosomiasis is the second most devastating tropical parasitic disease worldwide and is responsible for many urological complications. However, glomerular injury is a rare complication mainly described with *Schistosoma haematobium*. We describe the first case of *Schistosoma* infection, associated to segmental and focal hyalinosis in a young Senegalese patient with nephrotic syndrome responding perfectly well to antiparasitic treatment with complete remission of the syndrome.

Keywords: Segmental and focal hyalinosis; *Schistosoma haematobium*; Nephrotic syndrome; Kidney disease; Complete remission

1. Introduction

Schistosomiasis is a parasitic disease caused by organisms from the genus *Schistosoma*. The species that causes human schistosomiasis are *Schistosoma haematobium*, *Schistosoma intercalatum*, *Schistosoma japonicum*, *Schistosoma mansoni* and *Schistosoma mekongi* [1,2]. The adult worms inhabit the mesenteric vessels of men, the definitive host, and the intermediate forms develop into snails from the genus *Biomphalaria* [3]. In Morocco, cases of *Schistosoma* are rare and diagnosed mainly among tourists. Schistosomiasis-associated kidney disease is not frequently described in literature.

We report the first case of *Schistosoma* infection associated with segmental and focal hyalinosis in a young Senegalese patient residing in Morocco.

2. Case report

A young Patient 23 years old from Senegal, his medical past was a urinary bilharzia treated 10 years ago, notion of terminal macroscopic hematuria 2 months ago? It is presented for an oedemato-ascitic evolution syndrome progressive. Objective clinical examination a bilateral white edema, soft, painless taking the bucket. Blood pressure is measured at 160/90 mmHg, at the urinary strip: 3 crosses of proteins, 4 crosses of hematuria, there is no lumbar contact or vesical globe. The rest of the somatic examination is without particularity with the absence of palpable peripheral lymphadenopathy, likewise, there was neither hepatomegaly or splenomegaly.

The biological assessment found a profile in favor of nephrotic syndrome: hypoprotidemia (47g/l), hypoalbuminemia (15g/L) and proteinuria (3 g/24 h) associated with renal failure a creatinine level of 18 mg/l. A urinary examination carried out in search of *Schistosoma haematobium* eggs which was unsuccessful.

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Abdominal ultrasound finds kidneys of normal size, well differentiated, without hepatomegaly or splenomegaly. A renal biopsy puncture reveals concluded a segmental and focal Hyalinosis.

A cystoscopy carried out confirmed the presence at the chorion of ovoid figures surrounded by a concentric fibrosis corresponding to a pathogenic agent of *Schistosoma* eggs, with no sign of malignancy. Blood count, CRP, lipid and liver counts are normal. Viral serologies of hepatitis B, C and HIV are negative. An immunological balance involving the determination of the complement, the search for circulating immune complexes, antinuclear antibodies, glomerular basal membrane anti-membrane, antiphospholipid and cryoglobulins is without particularity.

The management consisted of a monoprise of Praziquantel at the dose of 3g, and the corticosteroid dose of 1mg/kg/d or 80mg/J for 12 weeks. The evolution was marked after one month of treatment by complete remission.

3. Discussion

This is the first case report of *Schistosomae*-associated Segmental and hyalionsse focale. However, in relation to the high prevalence of schistosomiasis in endemic areas, it might be underdiagnosed due to the absence of systematic screening [4]. Glomerular disease secondary to schistosomiasis had been reported in 10–15% of patients mainly with *S. mansoni* infection and MPGN is the most common histological pattern [5,6,7], Glomerular injury is generally due to direct deposition of schistosomal antigens (GASP, gut-associated schistosomal proteoglycan). Chronic hepatosplenic infection plays an important role in the subsequent disease progression [4,6]. *S. hematobium* has a greater tropism for lower urinary tract (ureter, urethra, and bladder). There was not any glomerular deposition at immunofluorescence in contrast with MPGN due to *S. mansoni* where glomerular and peritubular IgA deposits are frequent [5,6,8].

The glomerular lesion in schistosomiasis has an immunological nature. Antigens from the parasite seem to be related to glomerulopathy and have been found in the sera of humans and animals infected by the *S. mansoni* [9]. Antibodies directed against the parasite have also been found in humans and animals with schistosomiasis, which seem to be related to the development of glomerular injury [10]. Among the isolated circulating antigens, those from the digestive tract of the adult parasite are the most involved in the pathogenesis of glomerulopathy [11]. The presence of schistosomal antigens in the kidney were found in 43.7% of patients with non-nephrotic proteinuria and in 63.4% of those with nephrotic syndrome and advanced renal insufficiency [12,13]. The presence of *Schistosoma* antigens in glomerular deposits and the detection of circulating immune complexes containing these antigens have been described [14,15]. These findings highlight the hypothesis of immune complex-mediated glomerulonephritis. Vesical involvement is common in the infection by *S. haematobium*, a parasitic disease predominant in African countries. In the *S. haematobium* infection, hematuria and dysuria can be observed due to inflammation and ulceration in the bladder mucosa, generally occurring 3 to 4 months after primary infection.

In endemic areas, many children present microscopic hematuria, which can become macroscopic with aging. Polyps, hypertrophic nodules and eggs deposition can be observed through cystoscopy. Granulomas, fibrosis and calcifications in the vesical wall can cause vesico-ureteral reflux and obstructive uropathy, leading to hydronephrosis, chronic bacteriuria, and vesical cancer and, less frequently, renal insufficiency [16].

Some studies show that renal lesions are irreversible because many cases have delayed diagnosis [17]. However, specific antiparasitic treatment can alter the renal disease development or progression when instituted in the initial phases [17]. Patients with proliferative forms do not respond to antiparasitic treatment nor to immunosuppression, suggesting that this type of glomerular involvement has a progressive pattern [17]. Specific antiparasitic treatment is indicated to all patients infected by *Schistosoma*, with specific drugs to achieve infection cure. There are 2 drugs available for treatment, praziquantel and oxamniquine. The praziquantel is available in the dose of 600 mg, being administered in dose of 50 mg/ kg for adults and 60 mg/kg for children. Side effects are mild and there is no evidence of severe toxic reactions. Oxamniquine is presented in pills (250 mg) and solution (50 mg/mL). The recommended dose is 15 mg/kg for adults and 20 mg/kg for children, in unique dose. Adverse effects include nausea, dizziness and urticariform reaction [17].

This case of Segmental and hyalionsse focale associated with *Schistosomae* infection is unusual but the mechanism of glomerular injury might be similar to *S. mansoni* or *haematobium* infection, praziquinil was an available and effective treatment in the infection.



Figure 1 Schistosoma haematobium eggs

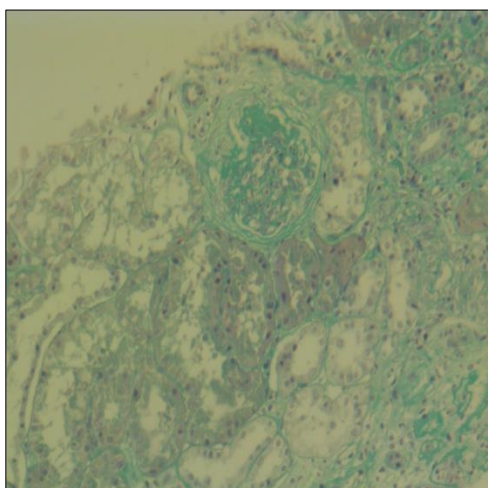


Figure 2 A renal biopsy showing the segmental and focal hyalinosis

4. Conclusion

Our case report is a valuable addition to the scientific literature, because Schistosoma infections can cause various glomerulonephritis with potentially poor prognosis, but the association with segmental and focal hyalinosis is rare and efforts might focus on the prevention, early detection and treatment of Schistosoma infections among.

Compliance with ethical standards

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Disclosure of conflict of interest

No conflict of interest.

Statement of informed consent:

Informed consent was obtained from all individual participants included in the study.

References

- [1] Ross AGP, Bartley PB, Sleigh AC, Olds GR, Li Y, Williams GM, McManus DP. Schistosomiasis. *N Engl J Med* 2002; 346: 1212- 1220.
- [2] Chitsulo L, Loverde P, Engels D. Schistosomiasis. *Nat Rev Microbiol* 2004; 2: 12-13.
- [3] Maguire JH. Trematodes (Schistosomes and Other Flukes). In: Mandell: Mandell, Douglas, and Bennett's Principles and Practice of Infectious Diseases. 7th ed. Philadelphia: Churchill Livingstone Elsevier, 2010, p. 3595-3605.
- [4] Abdou N, Boucar D, El Hadj Fary KA, Mouhamadou M, Abdoulaye L, et al. Histopathological profiles of nephropathies in Senegal. *Saudi J Kidney Dis Transpl.* 2003;14:212-4
- [5] Barsoum R. The changing face of schistosomal glomerulopathy. *Kidney Int.* 2004;66:2472-84;
- [6] Barsoum RS. Schistosomal glomerulopathies. *Kidney Int.* 1993;44:1-12
- [7] Nussenzweig I, De Brito T, Carneiro CR, Silva AM. Human *Schistosoma mansoni*-associated glomerulopathy in Brazil. *Nephrol Dial Transplant.* 2002;17:4-7.
- [8] Barsoum R, Nabil M, Saady G, Genin C, Saleh E, Francis M, et al. Immunoglobulin-A and the pathogenesis of schistosomal glomerulopathy. *Kidney Int.* 1996;50:920-8.
- [9] Madwar MA, Volier A. Circulating soluble antigens and antibody in schistosomiasis. *Br Med J* 1975; 1: 435-436.
- [10] Jassim A, Catty D, Hassan K. Antibody isotypes of immune complexes in schistosomiasis mansoni in Sudan. *Parasite Immunol* 1987; 9: 651-665.
- [11] Nash TE. Localization of the circulating antigen within the gut of *Schistosoma mansoni*. *Am J Trop Med Hyg* 1974; 23: 1085- 1087.
- [12] Sobh MA, Moustafa FE, el Housseini E, Basta MT, Deelder AM, Ghoniem MA. Schistosomal specific nephropathy leading to end-stage renal failure. *Kidney Int* 1987; 31: 1006-1011.
- [13] Sobh MA, Moustafa FE, Sally SM, Deelder AM, Ghoniem MA. Characterization of kidney lesions in early schistosomalspecific nephropathy. *Nephrol Dial Transplant* 1988; 3: 392- 398.
- [14] Houba V. Immunologic aspects of renal lesions associated with malaria. *Kidney Int* 1979; 16: 3-8.
- [15] van Marck EAE, Deelder AM, Gigase PLJ. Effect of portal vein ligation on immune glomerular deposits in *Schistosoma mansoni* infected mice. *Br J Exp Pathol* 1977; 58: 412-417.
- [16] Maguire JH. Trematodes (Schistosomes and Other Flukes). In: Mandell: Mandell, Douglas, and Bennett's Principles and Practice of Infectious Diseases. 7th ed. Philadelphia: Churchill Livingstone Elsevier, 2010.
- [17] Geraldo Bezerra da Silva Junior^{1,2}, Daniella Bezerra Duarte, Elvino José Guardão Barros³ , Elizabeth De Francesco Daher^{1*}, Schistosomiasis-associated kidney disease: A review , the Asian Pacific Journal of Tropical Disease ,2013 .