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Membranoproliferative Glomerulonephritis Associated With Renal Schistosomiasis in a Young Patient From Chad: A Report of a Rare Case

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Abstract

We report the case of a 17-year-old male patient from a rural area who was initially followed in a paediatric setting and subsequently referred to the Nephrology Department for corticosteroid-resistant nephrotic syndrome. The patient had a history of frequent freshwater exposure and tested positive for hepatitis C virus (HCV) antibodies. Clinical evaluation and paraclinical investigations revealed nephrotic syndrome characterised by severe proteinuria (+++), hypoalbuminemia (19.9 g/L), and generalised oedema, complicated by macroscopic haematuria (++) and impaired renal function. Renal biopsy demonstrated lobular membranoproliferative glomerulonephritis (MPGN) with extracapillary proliferation, moderate chronic tubulointerstitial nephropathy, and focal renal schistosomiasis. The clinical outcome was favourable following two courses of the antiparasitic drug praziquantel combined with corticosteroid therapy, resulting in parasite eradication, recovery of renal function, and return to normal daily activities.

Categories: Pediatrics, Nephrology
Keywords: chad, membranoproliferative glomerulonephritis, nephrotic syndrome, pediatric, renal schistosomiasis

Introduction

Schistosomiasis is an ancient parasitic disease, as demonstrated by the discovery of calcified *Schistosoma haematobium* eggs in Egyptian mummies dating from 1250-1000 BC [1]. It is the second most prevalent parasitic disease worldwide after malaria in terms of morbidity and mortality, with approximately 85% of cases occurring on the African continent, particularly in sub-Saharan Africa [2], making it a significant public health problem [3]. A recent survey from Chad conducted in two phases between November 2022 and April 2023 in 25 districts across seven provinces, all within Chad's tropical Sudanese-Sahelian zone, has found a prevalence of *S. haematobium* at 2.28%, while that of *Schistosoma mansoni* was 0.74%, with significant geographical heterogeneity [4]. Conversely, in the Torrock prefecture (Chad), the direct microscopic examination of urine from 875 children aged 1 to 14 years over a period of one year found 467 positive cases (24.9% of the total sample) [5]. During its course, schistosomiasis induces marked inflammatory, fibrotic, sclerosing, and calcifying lesions within the urinary tract, including the bladder, ureters, and kidneys [6].

Membranoproliferative glomerulonephritis (MPGN) is a rare glomerular disease. Indeed, MPGN is a disease characterised by immunofluorescence microscopy as either immune-complex mediated (IC-MPGN) or C3 glomerulopathy (C3G), the latter subdivided by electron microscopy to C3 glomerulonephritis (C3GN) and dense deposit disease (DDD). Both IC-MPGN and C3G typically have obvious C3 staining differentiating them from other causes of MPGN histology. Secondary causes of MPGN include infections, autoimmune disease, and neoplasia [7]. A recent systematic review on the prevalence of glomerular disease in Africa has found that MPGN was observed in 6.4% of cases, i.e., two to three times higher than its prevalence in Asia, North America, Latin America, and Europe [8]. In addition, in Africa the prevalence of MPGN is 9.8% and 3.3% in adults and children, respectively [8].

A study from Brazil has shown that the most frequent glomerular diseases associated with infection by *S. mansoni* or *Schistosoma japonicum* are MPGN and focal segmental glomerulosclerosis [9].

Neves et al. performed a retrospective analysis of 24 patients with a mean age of 38.58 ± 9.83 years diagnosed with schistosomiasis-associated glomerulopathy between 2002 and 2017; nephrotic-nephritic syndrome was observed in 13 (54.1%), hematuria in 20 (83.3%), and hypertension in 18 (75.0%). Renal

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histology showed a predominance of the membranoproliferative pattern (8%). On immunofluorescence, 19 patients (82.6%) showed immunoglobulin M (IgM) expression, 10 (43.4%) showed IgM+IgG expression, and one (4.3%) showed a "full house" pattern [10]. With a median follow-up of five years, 37.5% of patients had developed end-stage renal disease (ESRD).

Herein, we report on a very rare case of a 17-year-old male patient initially followed for atypical nephrotic syndrome unresponsive to corticosteroid therapy, in whom renal biopsy revealed MPGN associated with overt renal schistosomiasis.

Case Presentation

A 17-year-old male patient, living in a rural area in Guéra prefecture where schistosomiasis is endemic and working as a farmer, was referred to the Nephrology Department. Eight months before, he started presenting with progressive bilateral and symmetrical lower limb oedema, gradually extending to the abdomen and face, associated with macroscopic haematuria and intermittent vomiting. Initial consultations with traditional healers were ineffective. Worsening abdominal distension and pain eventually prompted medical evaluation by a paediatrician, i.e., two months before coming to our department. Facing oedema and proteinuria ++++, he was given prednisone 2 mg/kg /d (80 mg/day) for six weeks. Because there was no improvement, he was given IV methylprednisolone 1 g/m^2. Because two weeks later there was no improvement, he was referred to our centre. At this point, in addition to generalised oedema (unquantified weight gain), he had macroscopic haematuria associated with oliguria without dysuria, no skin lesions, occurring in an afebrile context.

The patient reported frequent freshwater exposure and had positive hepatitis C virus (HCV) serology; however, HCV RNA was negative. Indeed, he never received anti-HCV therapy. Clinical examination revealed nephrotic syndrome with proteinuria (+++), hypoalbuminemia, and generalised oedema, complicated by haematuria (++) and impaired renal function. Laboratory investigations demonstrated hypoproteinaemia (50 g/L), hypoalbuminemia (20 g/L), hypocalcaemia (56.6 mg/L), elevated serum creatinine (574 µmol/L), and 24-hour proteinuria of 3.9 g. Antinuclear antibody, antineutrophil cytoplasmic antibodies (ANCA) and serum complement levels were not evaluated due to their unavailability in our hospital (see Table *1*).

	At diagnosis	Three months after praziquantel	Normal values
Proteinuria	+++	0	0
Hematuria	++	0	0
Total proteins (g/L)	50	66	60-75
Albuminemia (g/L)	20	37	30-45
Serum creatinine (µmol/L)	574	102	90-110
Proteinuria (g/L)	3.9	<0.05	0

TABLE 1: Biological parameters before and after treatment of schistosomiasis

Renal and urinary tract ultrasonography showed kidneys of normal size with preserved corticomedullary differentiation.

Histopathological and immunohistochemical analysis was performed in the laboratory of anatomical and cytological pathology of Dakar (Senegal). This was an adequate renal biopsy that revealed lobular MPGN with 50% extracapillary proliferation at the fibrocellular stage, moderate chronic tubulointerstitial nephropathy, focal renal schistosomiasis, moderate necrotising tubulopathy, and evidence of arteriolar and glomerular nephrotoxicity (Figures *1*-*6*).

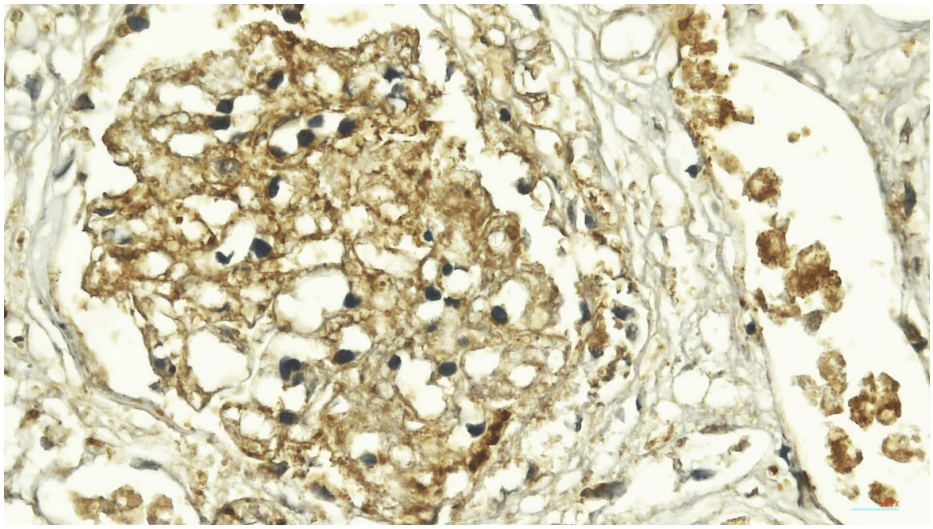


FIGURE 1: Immunohistochemistry anti-IgM antibody: diffuse glomerular deposits

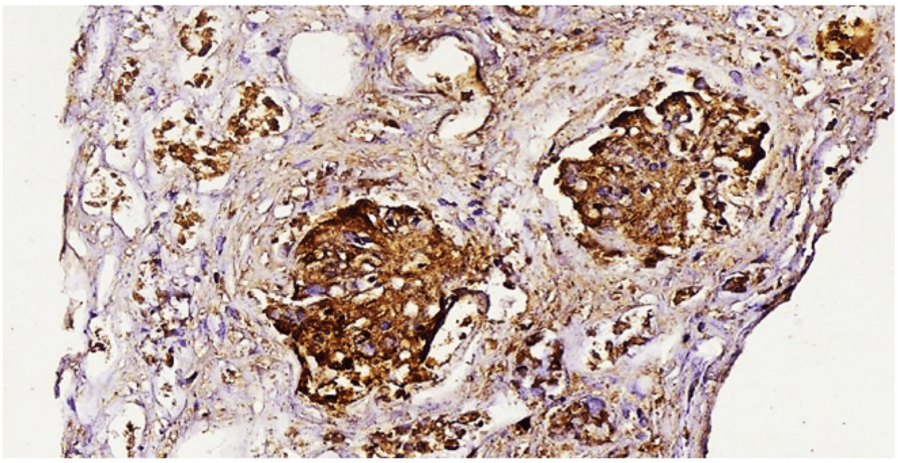


FIGURE 2: Immunohistochemistry staining with anti-C3 demonstrating granular deposits

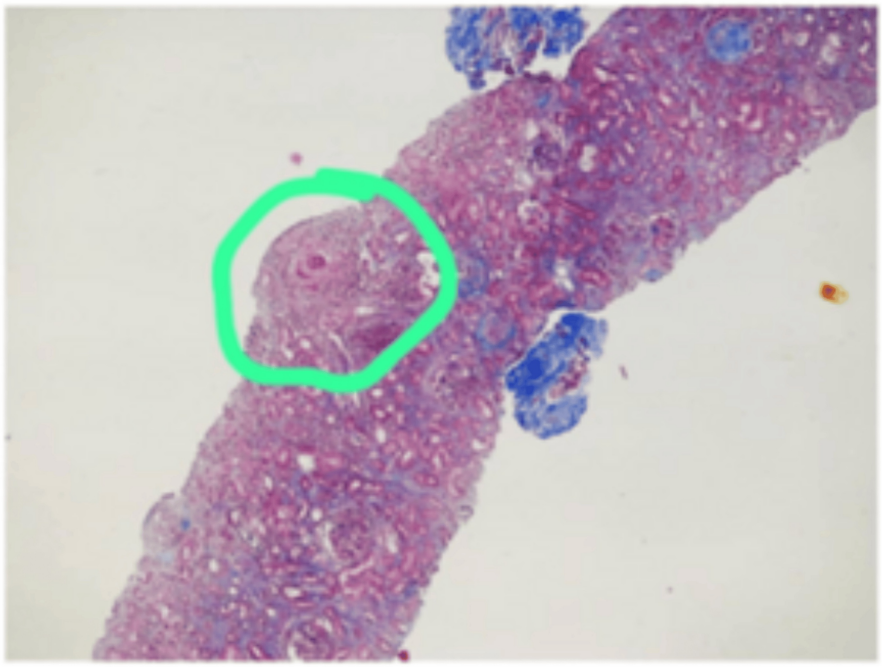


FIGURE 3: Renal schistosomiasis: moderate diffuse chronic tubulointerstitial nephropathy (Masson's trichrome stain Gx150; green circle indicates granuloma)

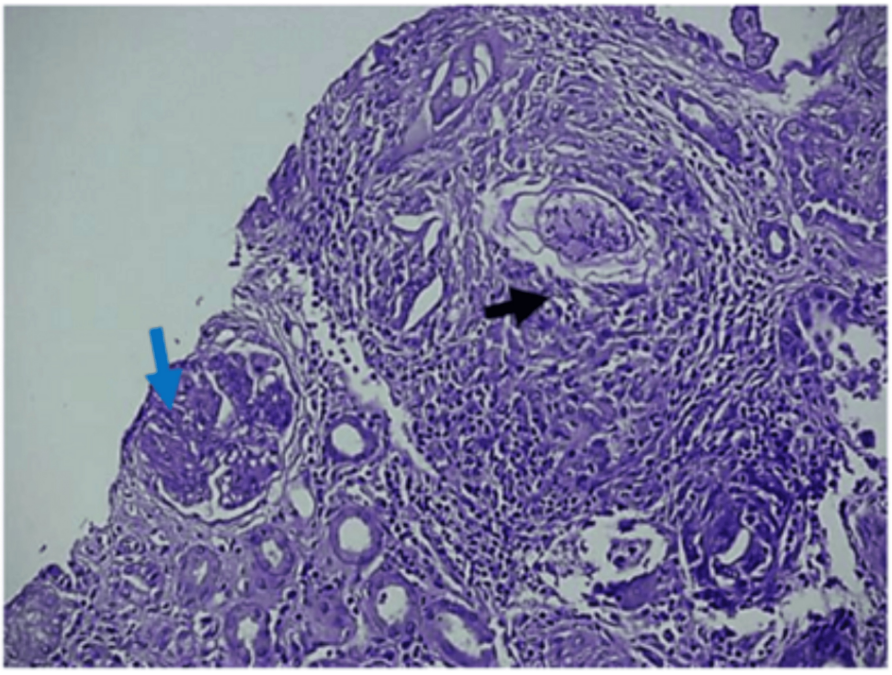


FIGURE 4: Renal schistosome granuloma with a viable schistosome egg (black arrow) in the centre and a glomerulus showing lobular MPGN (blue arrow), stained with PAS at x200 magnification

MPGN: membranoproliferative glomerulonephritis; PAS: Periodic acid-Schiff

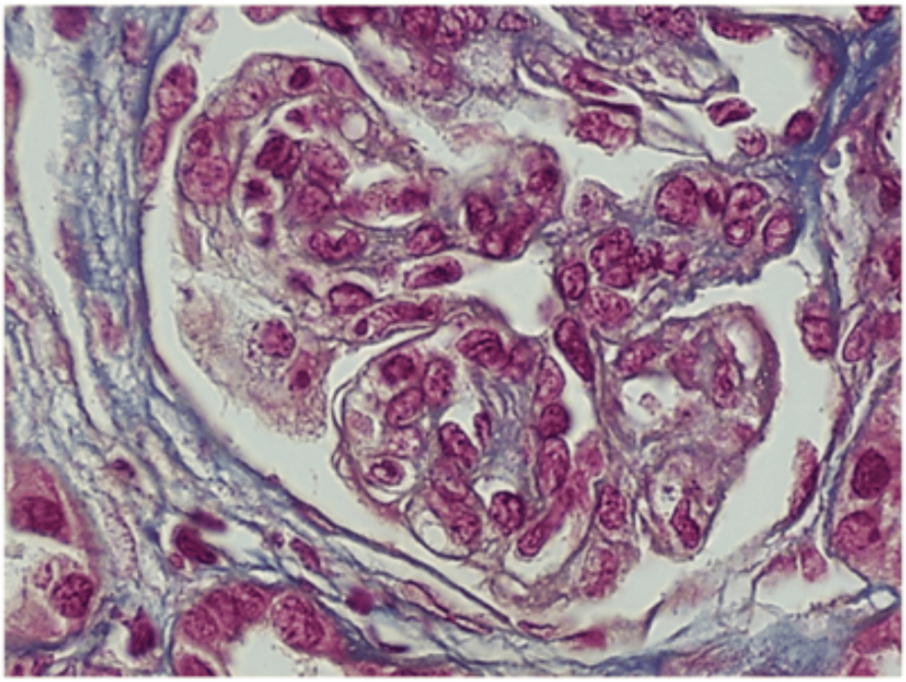


FIGURE 5: Lobular MPGN type 1, stained with Masson's trichrome at x300 magnification

MPGN: membranoproliferative glomerulonephritis

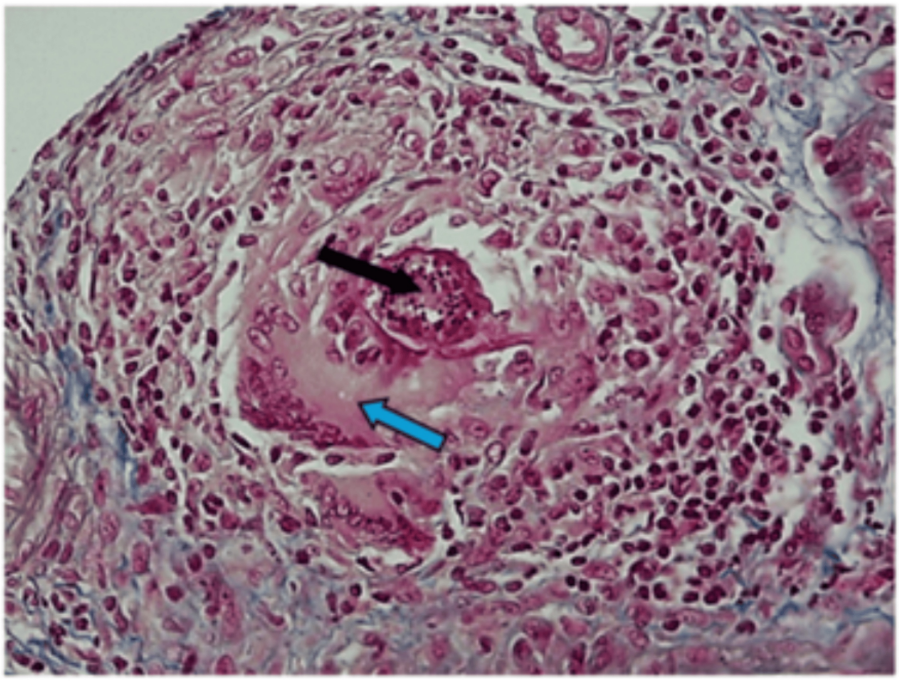


FIGURE 6: Renal schistosome granuloma with a viable schistosome egg (black arrow) and a giant cell (blue arrow), stained with Masson's trichrome at x400 magnification

Immunohistochemistry demonstrated abundant diffuse granular glomerular deposits of IgG and IgM, segmental C1q deposits, and absence of IgA deposits, supporting an immune-complex-mediated glomerular

disease.

Treatment consisted of two courses of praziquantel (40 mg/kg) combined with prednisolone (1 mg/kg for one month and then slowly tapered to 10 mg/day), indicated due to the presence of extracapillary proliferation. After three months of treatment, clinical and biological outcomes were favourable, with resolution of proteinuria (<0.05 g/d), normalisation of serum albumin (37 g/L), renal function (serum creatinine at 102 µmol/L), and resumption of normal social activities. Prednisolone has been withdrawn.

Table 2 summarises renal manifestations of schistosomiasis as well as its mechanisms.

Mechanism	Renal lesions
Obstruction/fibrosis	Hydronephrosis, renal atrophy
Infection	Chronic pyelonephritis, pyonephrosis
Immunologic	Mesangioproliferative GN, MPGN, FSGS
Chronic inflammation	AA amyloidosis
End-stage evolution	CKD

TABLE 2: Renal schistosomiasis

CKD: chronic kidney disease; GN: glomerulonephritis; MPGN: membranoproliferative glomerulonephritis; FSGS: focal and segmental glomerulosclerosis

Discussion

Renal involvement secondary to schistosomiasis remains rare and poorly documented in sub-Saharan Africa. In 2010, Dial and Noël reported a case of renal schistosomiasis in Senegal characterised by the presence of schistosome eggs in the renal parenchyma [11]. Urogenital schistosomiasis is most commonly associated with *S. haematobium*, and less frequently with *S. mansoni* or *S. japonicum*. In contrast, *Schistosoma intercalatum*, *Schistosoma mekongi*, and *Schistosoma guineensis* are primarily responsible for intestinal involvement [12].

Renal localisation is uncommon, accounting for less than 0.5% of cases compared to lower urinary tract involvement [13]. This rare localisation may be explained by portocaval anastomoses between portal and systemic venous circulations, including connections between splenic and renal veins, as well as mesocolic and renal venous systems [14].

This patient was coming from Guera prefecture, where a survey found schistosomiasis infecting nearly 50% of the population [15]. For this patient, renal biopsy demonstrated a typical pattern of MPGN [7]. Indeed, MPGN is a disease characterised by immunofluorescence microscopy as either IC-MPGN or C3G. Both IC-MPGN and C3G typically have obvious C3 staining differentiating them from other causes of MPGN histology. Secondary causes of MPGN include infections, autoimmune disease, and neoplasia.

The pathogenesis of MPGN in schistosomiasis is believed to be immunological [10,14], involving the formation and glomerular deposition of circulating immune complexes, as supported by the IgG, IgM, and C1q deposits observed in this case.

The coexistence of HCV infection, a leading infectious cause of MPGN in developing countries, may have contributed to disease severity and renal impairment at diagnosis [16]. However, we do not know for how long he had been infected with HCV; in addition, HCV infection spontaneously resolved. Immune complex deposition induces inflammatory injury, disruption of the glomerular basement membrane, and cellular proliferation, resulting in proteinuria and hematuria. Notably, the diagnosis of renal schistosomiasis was established despite negative urinary parasitological examinations, highlighting the essential role of renal biopsy. Combined therapy with praziquantel and corticosteroids led to complete clinical and biological remission, emphasising the importance of early diagnosis and appropriate management. Indeed, steroid therapy has been shown to be of benefit in chronic reanl/urological schistosomiasis [17]. However, currently the first-line treatment of schistosomiasis in its chronic phase relies on oral praziquantel 60 mg/kg/day (in two or three doses on days 0, 21, ±30) [18,19].

This case underscores the necessity of systematic parasitological evaluation in children presenting with nephrotic syndrome in endemic areas. Renal schistosomiasis should be considered even in the absence of typical urinary manifestations, particularly in young patients with freshwater exposure.

Conclusions

Secondary MPGN due to schistosomiasis is a rare but severe renal condition that may present as nephrotic syndrome in endemic regions. Renal schistosomiasis, i.e., with eggs in the parenchyma, is very rare. Accurate diagnosis requires parasitological investigations of urine and renal biopsy to guide appropriate therapy. Early recognition and combined antiparasitic and immunosuppressive treatment, if needed, can result in favourable outcomes.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Disclosures

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