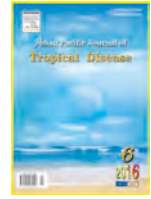




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### Nephritic syndrome and anasarca in a case of lymphatic filariasis: A rare association

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#### ABSTRACT

Lymphatic filariasis is caused by a nematode, namely, *Wuchereria bancrofti*, *Brugia malayi* and *Brugia timori*. Lymphatic filariasis is a spectrum of illness and can manifest as, asymptomatic microfilariasis, acute lymphatic filariasis, chronic lymphatic filariasis, tropical pulmonary eosinophilia and some systemic manifestations which involves joint, heart, kidney, nerve *etc.* Here, we present a case of anasarca with nephritic syndrome caused by lymphatic filariasis which is a rare presentation and only few cases have been reported in India so far.

## 1. Introduction

Filariasis is one of the oldest and neglected tropical diseases. Nearly 1.4 billion people worldwide are threatened by filariasis and over 120 million people are currently infected. After leprosy, filariasis is the second most common cause of long term disability[1]. India, China and Indonesia jointly bear the burden of two-thirds of all caseloads[2]. In India, filariasis is endemic in 17 states and six union territories affecting almost 31 million people[3]. It is endemic in Orissa, where its prevalence is very high in the coastal district of Cuttack. Although it is a common disease, there are no detailed studies indicating

possible involvement of the kidney in filariasis. However, there are isolated case reports of renal involvement[4-7]. Histological changes of renal involvement like acute mesangioproliferative glomerulonephritis, acute eosinophilic glomerulonephritis and membranous glomerulopathy have been documented[4-9]. Ultrastructural glomerular changes and immune complex deposits of immunoglobulin G, immunoglobulin M and C3 in the mesangium, capillary wall or glomerular basement membrane have been reported[7,8,10,11].

## 2. Case summary

A 35 years old nonalcoholic male patient, resident of Jamui District, Bihar was presented to our outpatient department with anasarca for 9 months (Figure 1). He initially developed shortness of breath on exertion along with cough with expectoration which was relieved by antibiotics. He then gradually developed swelling of both lower limbs simultaneously followed by facial puffiness and abdominal swelling later. He also complained of

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The study protocol was performed according to Helsinki declaration and approved by Clinical Research Ethics Committee, School of Tropical Medicine, Kolkata. Informed written consent was obtained from Clinical Research Ethics Committee, School of Tropical Medicine, Kolkata.

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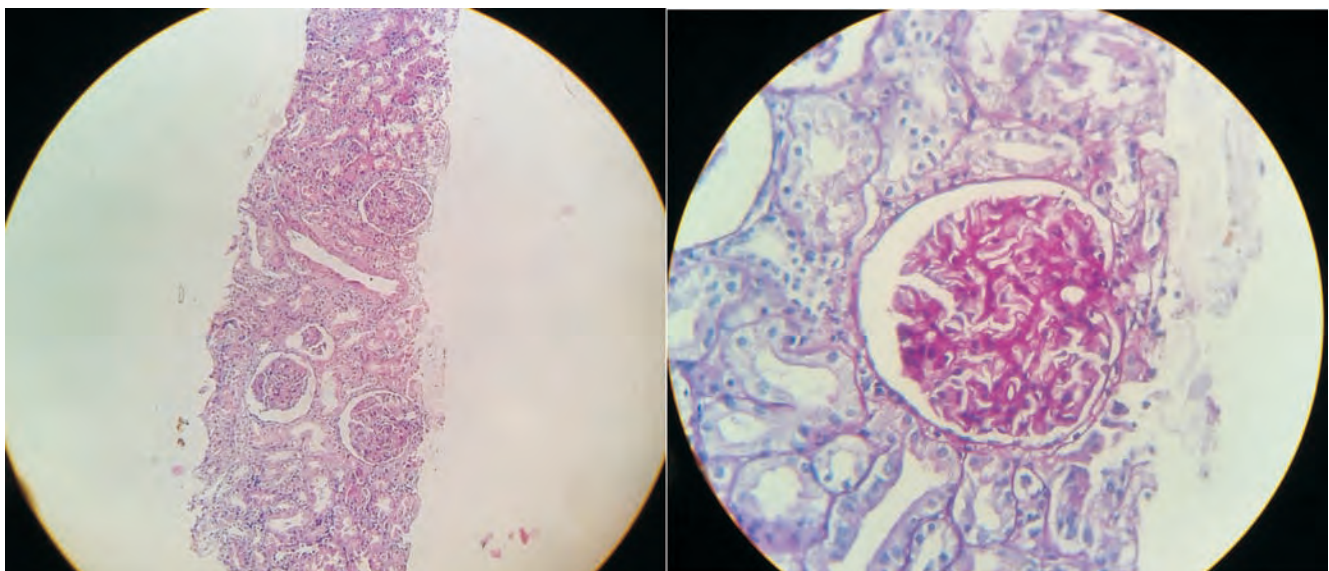
decreased volume of urine within 1 month. He had no past history of jaundice, haematemesis, melaena, palpitation and paroxysmal nocturnal dyspnoea. He had a past history of high total leucocyte count (11 500) and eosinophilia (58%), high urinary albumin and protein, and ascites and hepatomegaly on ultrasonography. Eosinophilia was corrected after intake of diethyl carbamazine citrate (DEC) of 100 mg thrice daily for 21 days given by local physician 6 months ago but other symptoms were not relieved.



**Figure 1.** Patient with features of anasarca.

On clinical examination, he had a puffy face with swollen eyelids, mild pallor and bilateral pitting pedal edema that extended up to both knees. His vitals were normal with blood pressure of 130/80 mm Hg. On abdominal examination, huge ascites with parietal pitting edema, scrotal and penile swellings were found. On chest auscultation, bilateral expiratory wheeze and coarse crepitation were found. Cerebral vascular spasm, central nervous system and musculoskeletal examinations remained unremarkable.

Laboratory investigations revealed haemoglobin level of 10.4%, red blood cells were normocytic normochromic [TC-9000 (N-50,L-13,E-34,M-3)], platelet was 195000, erythrocyte sedimentation rate was 13 mm/1st h, prothombin time was 14.2 and international normalized ratio was 1.18. Liver function test revealed normal bilirubin and enzymes, but total protein was 3.6 g/dL, albumin was 1.3 g/dL, urea was 16 mg/dL, creatinine was 1.03 mg/dL, albumin creatinine ratio was 7 084 mg/g and total cholesterol was 231 mg/dL. Routine urine exam revealed high proteinuria (24 h urinary protein was 4250 mg/day) with albumin +++, red blood cell of 15–20/hpf, pus cell of 4–5/hpf, granular cast of 3–4/hpf. Stool examination revealed no ova parasite cyst. His HIV status, hepatitis B surface antigen and anti hepatitis c virus status were nonreactive. Antistreptolysin titre was normal. Ascitic fluid showed 0.4 g/dL albumin and serum ascites albumin gradient was 0.9. He was put on tablets such as enalapril 2.5 mg *od*, frusemide 40 mg *od*, spironolactone 100 *bid* and metolazone 2.5 *od*. Keeping in mind about bilateral pedal edema, history of high eosinophil count and regression to normal level after giving tablet DEC, we evaluated him for filariasis. His peripheral blood smear showed no microfilaria but rapid filarial antigen test was positive. His kidney biopsy showed the features of membranous glomerulonephritis (irregular thickening of basement membrane, relative hypocellularity, mild excess of



**Figure 2.** Microscopic finding of kidney biopsy showing features of membranous glomerulonephritis.

mixed inflammatory cells in interstitium, tubules containing hyaline, granular cast, immunoglobulin G and granular, intraepithelial, global and diffuse C3 ++ (Figures 2A, B).

He was started on tablet DEC 100 mg *tid* for total 21 days and tablet doxycycline 100 mg *bid* for 14 days. Repeated 24 h total urinary protein after 15 days and one month of therapy decreased to 340 mg and 70 mg respectively. Routine examination of urine was repeated after two weeks and showed albumin +, RBC- 1–2/hpf, pus cell 0–2/hpf. His serum albumin was also increased to 1.4 gm/dL and eosinophil count became normal. He was discharged after 1 month of admission with clinical improvements like weight reduction (4.5 kg/30 days) and complete regression of ascites, facial puffiness, scrotal and penile swelling. He was asymptomatic during the two years follow up period.

### 3. Discussion

Several studies have shown a clear association of filariasis and glomerular disease[10,12-15]. Glomerular disease associated with filariasis is thought by most authors to be immune complex mediated[7,14,16]. Yap *et al.* reported an association of filariasis with glomerulonephritis that caused nephritic syndrome[13]. Pillay reported chronic glomerulonephritis in renal biopsy in patients with filariasis[4]. Waugh *et al* demonstrated mesangial deposits of immunoglobulin and complement by using immunofluorescent and electron microscopic studies suggesting an immune complex basis for glomerulonephritis in patients with filariasis[7]. Ngu *et al* and associates reported different renal histopathological abnormalities with filariasis[14]. Among 21 patients, they found four patients to have membranous glomerulonephritis and nine to have membranoproliferative and mesangial glomerulonephritis[14]. Though proteinuria and chyluria are much common in filariasis, glomerulonephritis due to filariasis is very rare[17].

Our case is unique because of the uncommon presentation of anasarca and membranous glomerulonephritis in filariasis which may easily be attributed to other forms of glomerulonephritis and filariasis may be overlooked if not thoroughly investigated. The main purpose of this study is to raise awareness among the students and physicians of the prevalence and importance of extra lymphatic disease in *Bancroftian filariasis* so that it is diagnosed early and treated properly and also to alert for the need of additional research in this field. To the best of our knowledge, this is the first reported case of nephritic syndrome with anasarca in West Bengal.

### Conflict of interest statement

We declare that we have no conflict of interest.

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