

troscopic detection of protruding suture material at the bleeding site is not widely appreciated.

In the patient reported, who had recurrent bleeding and abdominal pain for 11 months following subtotal gastrectomy, an intense hemorrhagic gastritis was found where silk suture material protruded into the lumen at the anastomotic site. Surgical resection of the anastomotic suture line was followed by clinical improvement.

Knowledge of this complication will result in prevention, earlier diagnosis, and surgical correction if the offending suture material cannot be removed gastroscopically.

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Tetanus Antitoxin Reactions

To the Editor.—Recent recommendations suggest booster doses of toxoid in previously immunized individuals and use of tetanus immune globulin (human) otherwise. This is adequately summarized in the *AMA Drug Evaluations 1971*. Present day courts might well question any use of tetanus antitoxin with adverse effects, should either of the other two alternatives be available.

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Tetanus immune globulin is not always readily available, and if a patient had not previously received tetanus toxoid, tetanus antitoxin would be indicated. At the same time, a dose of tetanus toxoid should be given as a start toward immunization.—

Ed.

Glomerulopathy Associated With Filarial Loiasis

To the Editor.—Glomerulonephritis is commonly associated with the formation in the plasma of antigen antibody complexes, which when trapped on the glomerular capillary membrane, initiate an inflammatory response. It is likely that the number of antigens with the potential for this series of events is extremely large, although the number of antigens identified is still small. Parasitic diseases,

such as malaria¹ and schistosomiasis,² may cause complex glomerulonephritis. Though not widely appreciated the filarial disease, loiasis, is also associated with glomerular disease. An example is presented here.

Report of a Case.—A 25-year-old Nigerian student arrived in the United States in 1969, in good health. In January 1972 he developed mumps. In July 1972 he noted polyuria and nocturia and was found to have proteinuria and urinary infection.

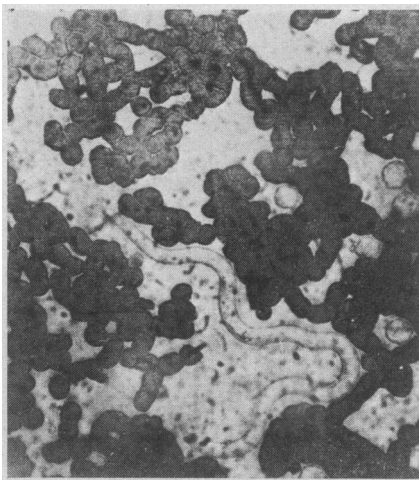


Fig 1.—Unstained blood smear showing microfilaria of *Loa loa*, (Original magnification $\times 1000$).

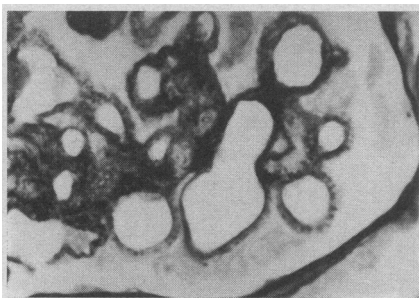


Fig 2.—Methanamine silver staining of renal biopsy showing thickening of basement membrane and the characteristic spikes, (Original magnification $\times 1000$).

The symptoms cleared after treatment with phenazopyridine hydrochloride. In August 1972 he developed itching in the left eye and was told that he had a worm in that eye. In December 1972 he was admitted to the University of Illinois Hospital for investigation of proteinuria. Physical examination was unremarkable. His blood pressure was 130/84 mm Hg, his eyes were normal, and there was no peripheral edema. The hematocrit reading was 42% with normal white blood cell count and platelets. Hemoglobin electrophoresis revealed 20% hemoglobin S and 80% hemoglobin A. Urinalysis revealed 2+ protein. Urinary sediment revealed 3 to 5 red blood cells per high power field. Serum creatinine level was 1.2 mg/100 ml, creatinine clear-

ance was 150 ml/min, 24-hour urinary protein excretion averaged 2.5 gm. Serum albumin level was 3.0 gm/100 ml, the globulins were 2.9 gm/100 ml, and the serum cholesterol level was 260 mg/100 ml. A peripheral blood smear was teeming with microfilaria belonging to the species *Loa loa* (Fig 1); no malarial parasites were seen. Microfilaria were not seen in the urine. A renal biopsied specimen contained four glomeruli that showed uniform thickening of capillary basement membranes. The silver stain showed prominent spikes (Fig 2). There were no proliferative changes. The biopsy was considered consistent with the diagnosis of membranous glomerulopathy. Unfortunately insufficient tissue was obtained for electron microscopic and immunofluorescent examination. The patient was given a three-week course of treatment with diethylcarbamazine citrate. The dose on the first day was 25 mg and was gradually increased until after a week's treatment the patient received a full daily dose of 6 mg/kg. This dose was then continued for 14 days. Three weeks after the last dose, the blood smear was negative for microfilaria; 24-hour urine protein excretion was 2.3 gm/100 ml.

Comment.—The renal changes associated with *Loa loa* infestation have recently been reviewed in a Dutch publication. Proteinuria is the main finding in *L. loa* nephropathy. The urinary sediment frequently contains red blood cells; occasionally microfilaria (usually dead) may be seen in the sediment. The few cases that have included renal biopsy as part of the workup,³ have shown pathologic changes described as "chronic glomerulonephritis," "swelling of some or all of the capillary walls, sometimes with endothelial proliferation," or "some infiltration of the renal interstitium with mononuclear cells." Our patient had proteinuria and membranous glomerulopathy. His renal function was well preserved.

While it seems most likely that the glomerulopathy associated with this disease is secondary to the formation of antigen-antibody complexes initiated by the parasitemia, in view of the finding of spikes on biopsy, definitive demonstration of these complexes by electron microscopy and immunofluorescent microscopy remains to be done.

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