A 66-year-old lady of Asian descent presented with hypertension of 1-year duration, heart murmurs, and hypercholesterolemia. She had frontal headaches, syncope, and 10 lb weight loss over the past few months. Physical examination revealed an elderly well-oriented lady in no distress. Her pulse was 82 min⁻¹, blood pressure 140/90, neck supple with no bruits or venous distention, skin showed no rashes, lower limbs revealed no edema, cardiovascular examination showed normal S1 and S2 with III/VI systolic murmur heard over the left sternal border, lungs were clear to auscultation, and abdomen was soft and non tender. Her medications included an angiotensin-converting enzyme inhibitor and a statin. Laboratory findings were significant for a serum creatinine level of 1.3 mg per 100 ml, creatinine clearance of 44 ml min⁻¹, and proteinuria (3.2 g 24 h⁻¹). Lipid profile showed total cholesterol of 179 mg per 100 ml, triglycerides 84 mg per 100 ml, high-density lipoprotein 37 mg per 100 ml, low-density lipoprotein 129 mg per 100 ml, and very-low-density lipoprotein 16.8 mg per 100 ml. Remaining work up, including serologies, was negative. A renal biopsy was performed to explain the proteinuria and declining renal function.

**RENAL BIOPSY**

A total of 16 glomeruli were present, two of which were globally sclerosed. Glomeruli showed distention and plugging of the capillary loops by numerous periodic acid–Schiff-positive amorphous ‘thrombus’-like material (Figure 1a and b). The ‘thrombi’ showed lamellations, did not polarize, and did not stain for fibrin (red on trichrome). Immunofluorescence microscopy was negative for immune deposits. Electron microscopy showed that the thrombi consisted of lipid material with vacuoles of different sizes (Figure 2). Foam cells were also noted in some capillaries. The capillary walls showed thickening and new basement membrane formation (double contours, arrows), indicating endothelial injury and repair.

**What is your diagnosis? What additional stain would help confirm the diagnosis?**
DISCUSSION
The diagnosis of lipoprotein glomerulopathy is based on the finding of lamellated lipid-like material within glomerular capillaries by light microscopy; electron microscopy showed the presence of lipid vacuoles of varying sizes within glomerular capillaries. The lipid ‘thrombi’ are limited to the glomerulus. Lipoprotein glomerulopathy on renal biopsy was confirmed using immunohistochemical stains against apolipoprotein E (Apo E) that stain the lipid ‘thrombi’ (Figure 3).

Lipoprotein glomerulopathy is a rare disease seen mostly in patients of Asian ancestry. The patient typically presents with hypertension and proteinuria; hematuria is rare. Renal biopsy is required for diagnosis. The disease is associated with mutations in the Apo E gene, resulting in elevated serum Apo E levels. The disease can be confirmed by DNA sequencing for Apo E mutations. Other systemic manifestations of hyperlipoproteinemia, such as xanthomas, corneal arcus, and so on, are rare. Management is currently focused toward intensive lipid lowering with statins and fibrates. There is a high degree of recurrence of the disease in patients treated with renal transplantation.1–3

REFERENCES