A 72-year-old woman was referred for an eye examination because of visual changes in the left eye. Her medical history was notable for end-stage kidney disease requiring a kidney transplant at age 50 years and a second transplant at age 65 years. She had received her medical care at outside facilities, the records of which were not available. She could not recall being given any precise diagnosis but reported having proteinuria since age 7 years. Her other medical conditions included hyperlipidemia, arterial hypertension, and pulmonary Mycobacterium avium complex infection. She was taking tacrolimus, amlodipine, atorvastatin, ethambutol, rifampin, and clarithromycin. Her family history was unremarkable. She had 2 healthy adult children.

One year after the first kidney transplant, she noted an inferior visual field defect in the left eye and was diagnosed with nonarteritic anterior ischemic optic neuropathy. Several years later, she underwent uncomplicated bilateral cataract procedures.

Visual acuity with distance correction was 20/25 OD and 20/30 OS. Color vision was 14/14 plates for each eye. There was a left relative afferent pupillary defect. Confrontation visual field was full for the right eye and demonstrated an inferior defect for the left eye. External and slitlamp examinations were notable only for bilateral pseudophakia. Both optic discs were anomalous in appearance, with central excavation in the left eye greater than the right eye (Figure). The remainder of the posterior pole examination, including the retinal vessels, appeared normal in both eyes.

What Would You Do Next?

1. Cranial magnetic resonance imaging
2. Genetic testing
3. Vasculitis serological evaluation
4. Positron emission tomography