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1670 Living Donor Kidney Transplantation: Facilitating Education about Live Kidney Donation—Recommendations from a Consensus Conference
Jane C. Tan, Elisa J. Gordon, Mary Amanda Dew, Dianne LaPointe Rudow, Robert W. Steiner, E. Steve Woodle, Rebecca Hays, James R. Rodrigue, and Dorry L. Segev
What’s the diagnosis? A previously healthy 18 year old male presented with leg swelling for three months, progressing to anasarca and hypertension. There was no family history of renal disease. Urinalysis revealed hemoglobin +2/+4 and the 24 hour urine collection contained 6.1g of protein. His serum creatinine was 0.80 mg/dl, cholesterol 268 mg/dl, high-density lipoprotein 18 mg/dl, and albumin 2.7 g/dl. Serum complement levels were normal and evaluation of secondary causes of the nephrotic syndrome was negative. Renal biopsy was performed. Light microscopy showed global endocapillary hypercellularity, mainly due to mesangial cells and eosinophilic deposits in subendothelial and subepithelial (granular pattern) locations. With silver stain, these deposits were negative and double contour of the basement membrane and spikes were seen (cover image). There were a few foci of interstitial fibrosis with rare atrophic tubules and no vascular lesions. Direct immunofluorescence was negative except for C3, which had strong mesangial staining and granular pattern in capillary loops, both subendothelial and subepithelial, global and diffuse in the glomeruli, and in vascular walls. Electron microscopy showed glomerular basement membrane vacuoles containing electron-lucent and electron-dense deposits (image below) compatible with Lecithin Cholesterol Acyltransferase Deficiency. The patient was treated with furosemide, captopril, prednisone and simvastatin with improvement of the edema and reduction of daily proteinuria to 2g. LCAT deficiency is a rare genetic disease with estimated prevalence of 1:100000 population. There are 88 genes mutations associated with the disease. This is an autosomal recessive disorder characterized by abnormalities in lipoprotein metabolism, leading to diminished formation of high-density lipoproteins and production of lipoprotein-X. These lipoproteins are deposited in the cornea and kidney, leading to progressive dysfunction. Other clinical findings include severe anemia and dyslipidemia. In the kidney, the deposits may be seen in glomeruli, arteries and arterioles. The ideal treatment would be enzyme replacement or LCAT gene replacement therapy however these options are not yet available in clinical practice. The current treatment is based in the management of nephrotic syndrome with statins, angiotensin II receptor blockers or angiotensin converting enzyme inhibitors and corticosteroids. The recurrence of the renal abnormalities after kidney transplantation is inevitable. (Images and text provided by Precil Diego Miranda de Menezes Neves, Federal University of Triângulo Mineiro - Nephropathology Service Uberaba, Minas Gerais, Brazil and colleagues) A complete list of authors is available as Supplemental Information.