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On the Cover

What's the diagnosis? Sickle cell anemia occurs due to a mutation in the globin gene that causes the replacement of one pair of amino-acid residues, resulting in drastically reduced solubility of deoxyhemoglobin S and its precipitation in the red blood cells. Affected red blood cells become elongated and rigid; this deformation and stiffness is the distinguishing feature of sickle cell anemia and is the primary cause of the symptoms. The pictured electronmicrograph shows a glomerular capillary loop filled with distorted red blood cells in a patient during sickle cell crisis. Normal red cells show dark and homogenous cytoplasm ultrastructurally, while sickled cells are of elongated and sometimes bizarre shapes, and reveal intracytoplasmic fibers organized in bundles; occasional vacuoles can also be seen in the cytoplasm of the abnormal red blood cells. These changes in the structure and shape of red blood cells result in injury to the endothelium and, over time, the glomerular capillary walls can show signs of remodeling, reduplication of the basement membranes, and a membranoproliferative pattern of glomerular injury, without electron-dense deposits - changes typically seen in patients with repetitive endothelial cell injury and chronic thrombotic microangiopathies. (Image and text provided by Dr. Vanesa Bijol, Brigham and Women's Hospital.)