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What's the diagnosis? A 28-year-old-man presented with normocytic normochromic anemia, thrombocytopenia, and acute kidney injury with a serum creatinine of 6.7mg/dl. A kidney biopsy was performed. Light microscopy of the biopsy revealed unremarkable glomeruli. The proximal tubular epithelial cells showed coarse golden-brown pigment (top image), which gave a positive reaction on Perls Prussian blue stain for iron (bottom image), consistent with deposition of hemosiderin. There were changes of acute tubular injury, including dilatation of tubules, cytoplasmic vacuoles, and cellular debris in the lumen. There was no significant interstitial inflammation or fibrosis or vascular abnormalities.

Renal hemosiderosis occurs as a complication of intravascular hemolysis. Hemolytic anemias, mismatched blood transfusions, paroxysmal nocturnal hemoglobinuria and mechanical hemolysis from prosthetic cardiac valves are the common causes. Intravascular haemolysis releases hemoglobin (Hb) into plasma, where it binds to plasma haptoglobin (Hp), forming Hb–Hp complex. This complex is not filtered by the glomerulus and is degraded by the liver. In chronic hemolytic states, plasma haptoglobin is consumed. Unbound (free) Hb accumulates in plasma and is filtered by the glomerulus. It is reabsorbed by the proximal tubule cells, resulting in the accumulation of ferric ions (hemosiderin) and subsequent cell injury.

Renal hemosiderosis seldom causes renal dysfunction and is usually an incidental radiologic or autopsy finding. In cases with clinical involvement, it causes reversible acute dysfunction to chronic irreversible damage. Treatment is usually directed to the underlying disease. Iron chelation therapy may be helpful.

(*Image and text provided by Anila Kurien, Center for Renal and Urological Pathology Pvt Ltd Chennai, Tamil Nadu, India, and Edwin Fernando, Government Stanley Medical College, Nephrology Chennai, Tamil Nadu, India*)