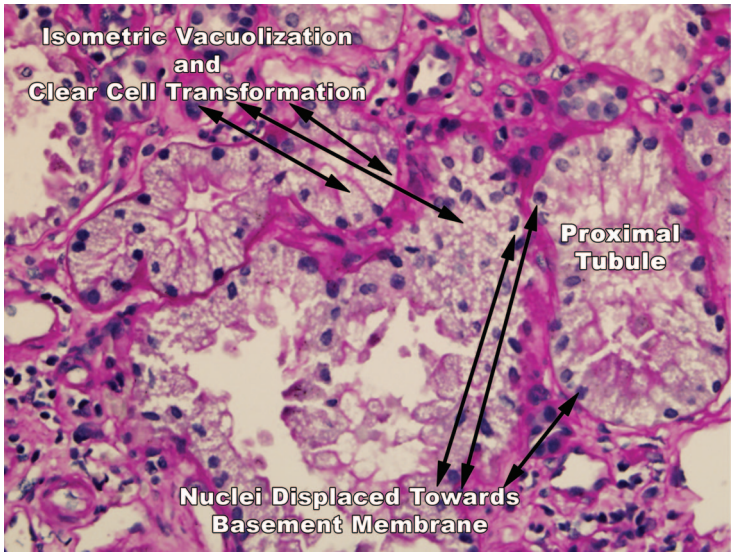
Hetastarch-induced Osmotic Nephrosis

Avinash B. Kumar, M.D., F.C.C.P., Manish Suneja, M.D.
University of Iowa Hospitals and Clinics, Iowa City, Iowa.
avinash-kumar@uiowa.edu



A 63-YR-OLD male (weighing approximately 83 kg) with no significant past medical history underwent an open reduction and internal fixation of his femur fracture under general anesthesia. He had been nil per os for 14 h and his preoperative blood pressure was 136/74 mmHg. Intraoperative course was uneventful, except for a 5–7 min episode of relative hypotension with systolic pressures 80–85 mmHg. He received 500 ml of 6% Hetastarch (Hospira, Lake Forest, IL) during this episode, and another 500 ml of 6% Hetastarch was started and completed during the case. His baseline serum creatinine was 1.1 mg/dl. He was oliguric on postoperative day 3 and his serum creatinine increased to 3.8 mg/dl. Urinalysis was consistent with acute tubular necrosis (muddy casts and tubular epithelial cells). A kidney biopsy was performed (image) considering a differential diagnosis of acute tubular necrosis, drug-induced nephrotoxicity, and interstitial nephritis. However, a histopathologic diagnosis of osmotic nephrosis was made.

Osmotic nephrosis is characterized by extensive vacuolization, clear cell transformation, and swelling of the proximal tubular epithelial cells with nuclei being displaced toward the basal membranes. The vacuoles in the proximal tubules are formed by the fusion of pinocytic vesicles (often containing the offending agent) and lysosomes containing hydrolytic enzymes. Distal tubules and collecting ducts are usually not affected.[1] Osmotic nephrosis has been reported with other osmotically active compounds, including mannitol, dextran-40, and iodinated contrast media.[2] Osmotic nephrosis (biopsy diagnosis) is an often unrecognized and frequently reversible cause of acute kidney injury. The choice of colloid may play a role in the pathophysiology of acute kidney injury and should be considered in the differential diagnosis.[3] This patient's creatinine returned to baseline at the time of discharge from the hospital.

References

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