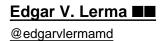
Twitter Thread by <u>Edgar V. Lerma</u> ■■





Low Flow AKI: Pathophysiology of Prerenal Azotemia ca. 2022 from @CJASN #Nephpearls #PathophysiologyPearls

https://t.co/8pEmCLePjy

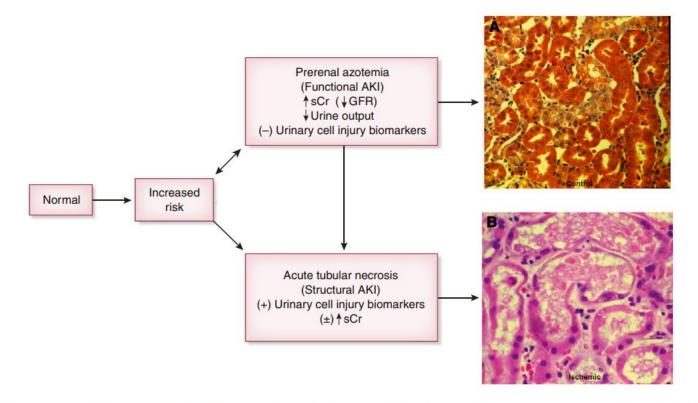


Figure 1. | Acute kidney injury divided into prerenal azotemia (functional AKI) and acute tubular necrosis (structural AKI) on the basis of serum creatinine (sCr) and urinary biomarkers. Prerenal azotemia has negative urinary cell injury biomarkers, whereas acute tubular necrosis has positive urinary cell injury biomarkers, indicating proximal tubule cell injury or dysfunction. (A) An image of normal human cortex, and prerenal azotemia appears the same. Courtesy of Jim Hasbargen. (B) A human kidney biopsy specimen 24 hours after injury. Note the flattened proximal tubule cells and shed brush border membrane in the lumen. The peritubular capillaries are filled with white blood cell and rouleauxs, and a mitotic cell is visible. Reprinted from ref. 67, with permission.