Hey everyone, this is Zoya Qureshy, fourth year medical student at UCSF! I'm so excited to share with you our schema for interpreting urinalysis.

Urinalysis, along with urine chemistry studies, provide information about the gross appearance and the levels of proteins, cells, and debris in the urine. Using this diagnostic tool, we can differentiate among several types of insults to the kidney, many of which can lead to acute kidney injury. We will separate the urine studies into three major categories: (1) Urinalysis or UA which provides information on whether protein (specifically albumin), blood, and nitrites, among other things can be detected in the urine; (2) urine protein to creatinine ratio, which is used to quantify proteinuria; and (3) urine sediment microscopy, which allows us to see cells and casts. Urine total protein and urine creatinine concentrations are measured in urine chemistry analysis. The UA ONLY detects albumin, whereas the urine chemistry detects ALL protein.

Let's start with prerenal kidney injury. This is generally as a result of hemodynamic perturbances such as hypovolemia, CHF and hepatorenal syndrome, leading to decreased perfusion of the kidney. Our UA, protein-to-creatinine ratio, and microscopy are generally normal, especially in early stages. And fun fact about hepatorenal syndrome: it can cause dark stained hyaline casts on microscopy due to the high bilirubin in the urine; these can sometimes be mistaken for "muddy brown casts", which is the hallmark of acute tubular necrosis or ATN.

In post-renal causes of kidney injury, especially early on, the UA, protein to creatinine ratio, and microscopy are similarly normal. One of these causes is some sort of obstruction, such as a tumor, and can be most efficiently evaluated by using a bladder scan, renal ultrasound or other imaging.

Persistent pre-renal injury, or the use of certain nephrotoxic agents, can lead to an intrarenal injury known as ATN. ATN is a disorder that is caused by damaged kidney tubule cells. When the epithelial cells lining tubules slough off, they can collect and obstruct the tubules. The UA is often normal. The protein to creatinine ratio can be mildly elevated but is generally less than 1 gram, as there is protein loss from the tubule cells; these proteins generally do not include albumin, which is usually lost into the urine in glomerular diseases. The most remarkable and confirmatory finding of ATN are the presence of "muddy brown casts" on microscopy. These are granular casts that are formed from the solidified epithelial cells that sloughed into the tubules.

Injury to small intrarenal vessels can also lead to decreased kidney function due to decreased perfusion and blood supply in the kidney. Some causes include hypertension, emboli from atherosclerosis or endocarditis, or vasculitis. Our urine studies will also have a mildly elevated protein to creatinine ratio less than one mostly containing non-albumin. The UA and microscopy will be normal.

Interstitial nephritis, inflammation in the interstitium of the kidney, is another intrarenal insult that can also lead to acute kidney injury. Causes of acute interstitial nephritis are medications such as penicillin or Bactrim, infections by organisms such as legionella, and systemic disorders such as sarcoidosis or lupus. In interstitial nephritis there might be mild non-albumin protein to creatinine ratio elevations. Microscopy is generally normal, however there can be occasional

white blood cells or white blood cell casts. Occasionally, a biopsy will be needed to differentiate among the different causes of interstitial nephritis.

Glomerular disease can also be a cause of intrarenal injury. Inflammatory glomerular disease, or glomerulonephritis, is caused by inflammation of the glomerulus resulting in hematuria and proteinuria. While the urine studies can be helpful, diagnosing what is causing the glomerulonephritis often requires a biopsy. On UA, blood and protein is detected. The protein to creatinine ratio is elevated but mildly. And the protein is mostly albumin, which is often lost in glomerular diseases. On microscopy, red blood cells and red blood cell casts are seen. Remember casts are formed from dead cells collecting and solidifying in the tubule; their presence would indicate that there is bleeding into the tubules. If RBC casts are present, there is a very low chance that they do not have glomerulonephritis, meaning it has a high specificity. However not having RBC casts does not rule out glomerulonephritis, meaning this finding has a low sensitivity. The presence of dysmorphic RBC confers both a low sensitivity and specificity, as they can show up in a number of other renal conditions.

Other non-inflammatory glomerular conditions mostly consist of nephrotic syndromes. In these diseases, there is no blood detected in the UA, but there is protein, mainly albumin. In fact, nephrotic syndromes are characterized by hypoalbuminemia, or low blood albumin, since it is lost in the urine. The protein to creatinine ratio is severely elevated; nephrotic syndrome is also characterized by a protein to creatinine ratio that is greater than or equal to 3.5. Lipiduria can also occur in nephrotic syndromes, so on microscopy sometimes fatty casts can be seen.

Urine studies provide a lot of information and can often be useful for creating a differential for what is causing kidney injury. Thank you everyone for listening in today and I hope you all enjoyed this video!