**Case 1:** A55-year-old woman with HCV cirrhosis presenting with fever and increased confusion. She reports increased abdominal girth but no pain, decreased UOP. Denies SOB, cough or dysuria. She has been taking lactulose as directed. On exam, she is afebrile, HR 92, BP 92/60, 98% on RA. She is diffusely edematous with a distended, nontender abdomen. Labs are notable for:

* Na 127, BUN 30, Cr 2.4 (baseline 1.2)
* WBC 12k, Hct 30%, Plt 90k
* Tbili 4 (baseline 2-3), alb 2
* UA: RBCs, hyaline casts
* Urine Na <10

**What further work-up do you want?**

Her low urine Na suggests a prerenal etiology of AKI. The differential includes GIB, volume loss, sepsis, medications or HRS. She is coming in with acute decompensation of her chronic liver disease (worsening Cr, Tbili and worsening encephalopathy) and should be evaluated for spontaneous bacterial peritonitis (SBP). This can present without significant abdominal pain.

* Diagnostic paracentesis for SBP – showed 500 cells, 90% PMNs, gram stain shows PMNs only, culture is pending.
* An albumin challenge can be initiated to evaluate for HRS while awaiting results of other testing.

**What is the cause of AKI?**

SBP! Greater than 250 PMNs on ascitic fluid cell count is suggestive of SBP. Empiric treatment should be started.

**How would you manage this patient?**

IV cefoxtaxime or ceftriaxone x 5 days. IV fluoroquinolones can be used in penicillin-allergic patients.

Albumin infusion 1.5 g/kg on day 1 and 1 g/kg should be used in patients with advanced liver or renal dysfunction – Tbili > 4, Cr > 1.0, BUN > 30. This has been shown to improve mortality and decrease risk of renal dysfunction.

**Case 2:** A 48-year-old man with HCV/EtOH cirrhosis actively listed for transplant presents from clinic with several lab abnormalities – worsening Cr, Tbili and INR. He reports increased fatigue, LE swelling and abdominal girth despite taking his diuretics. He reports mild abdominal diffuse abdominal discomfort but denies fever, chills, cough, SOB or dysuria. On exam, he is afebrile, HR 60, 92/45, 96% RA. He is diffusely edematous with a tight distended abdomen. Labs are notable for:

* Na 126, BUN 40, Cr 2.4 (baseline 1.0 from 1 month prior)
* WBC 8, Hct 28, Plt 80
* Tbili 11 (baseline 4), INR 5 (baseline 2)
* UA: 1+ protein, hyaline casts, occasional granular casts
* Urine Na>65, FeUrea 30%

**What further work-up do you want?**

FeUrea should be used in place of FeNa when patients are on diuretic therapy (which will cause urinary sodium excretion, resulting in high urine Na). FeUrea <35% is suggestive of prerenal cause of AKI.

Of note, ATN can be difficult to distinguish from other causes of AKI in patients with ESLD. Granular casts can be seen in the setting of severe hyperbilirubinemia and are not specific to ATN. FeNa and urine Na can be low even in the setting of ATN given persistent activation of renin-angiotensin-aldosterone system in advanced cirrhosis (aldosterone reclaims Na from urine).

He is presenting with acute decompensation of her chronic liver disease (worsening Cr, Tbili, Tbili and worsening ascites). He should be further evaluated with:

* Diagnostic paracentesis for SBP – showed 450 cells, 10% PMNs
* RUQ US with duplex for portal vein thrombosis (PVT) given worsening ascites – negative for PVT
* Consider Foley placement to measure bladder pressure to evaluate for abdominal compartment syndrome – bladder pressure 12 mmHg (normal)
* Albumin challenge – no improvement in Cr after 1 g/kg albumin x 48 hr.

**What is the cause of AKI?**

He has HRS given non-response to albumin challenge, likely type II given Cr < 2.5. Reviewing trend of Cr can point towards type I or type II. Type II HRS is commonly associated with refractory ascites.

**How would you manage this patient?**

* Stop his diuretics, his volume can be managed with periodic paracenteses
* Albumin up to 50 g/day for additional volume expansion
* Midodrine 5 – 7.5 mg TID as a starting dose with goal increase in MAP > 15 mmHg
* Octreotide 100 μg SQ TID

His cirrhosis is too advanced to consider TIPS should he not respond to this therapy.

Definitive therapy is a liver transplant (fortunately, he’s already listed!). Nephrology should be consulted to help manage his HRS. In some cases, patients require a concurrent liver-kidney transplant for severe HRS.

**Case 3:** A 45-year-old man with PSC cirrhosis presenting to the ED with increased abdominal pain typical of his PSC flares. The pain has been going on for about a week and he was initially managing it with OTC pain medications (Tylenol and ibuprofen) but has progressively worsened. He denies fevers/chills, cough or abdominal pain. He reports decreased UOP and darker colored urine. On exam, he is AF, HR 90, BP 120/80, 98% RA. He has a non-distended abdomen with mild RUQ tenderness without a Murphys’ sign. He has no CVA tenderness. Labs are notable for:

* Na 132, K 5.8, Cr 3.6 (baseline of 0.9)
* WBC 6, Hct 32, Plt 110
* Tbili 4 (baseline of 3), INR 1.4
* UA: hyaline casts, + WBC casts, no bacteria
* Urine Na 28, Urine Cr 60

**What is the likely cause of AKI?**

He has an intrinsic/intrarenal cause of AKI. His FeNa is 1.5%. With + WBC casts and recent heavy NSAID use, we have a high suspicion for AIN (acute interstitial nephritis).

**How would you manage this patient?**

* Stop offending medications – NSAIDs
* Volume challenge – reasonable to give a volume challenge to patients with AKI without signs of significant volume overload to rule out any concurrent prerenal etiology. This is especially useful in this case given that NSAIDs also cause a pre-renal AKI and he may have had decreased PO in the setting of recent PSC flare.