Lightning Lecture: Cirrhosis (Dr. Kelesis)

- Definition → hepatic disease characterized by distortion of normal hepatic tissue with fibrosis and regenerative nodules
- Most commonly caused by alcohol
- Compensated vs decompensated
    - Compensated usually asymptomatic
    - Decompensated → development of major complications, infection leading cause of mortality
- Varices
    - 50% are gastroesophageal
    - Treatment: ABCs, NG tube, fluids/blood, antibiotics, GI/IR consult early
- Ascites
    - Most common complication, and most common cause of admission
    - Avoid ACEs/ARBs, NSAIDs, ASA
- Infection (SBP)
    - Primary cause of mortality/decompensation
    - Usually E. coli or klebsiella
    - Fever and abdominal pain
    - Paracentesis → greater than 250 PMNs
    - Treatment: Ceftriaxone/cefotaxime (fluoroquinolones if allergic)
- Hepatic encephalopathy
    - Elevated ammonia metabolism → increased glutamine → astrocyte swelling and impaired neurotransmission
    - AMS, asterixis, impaired motor function
    - Treat with lactulose, rifaximin
- Hepatorenal Syndrome
    - Decreased renal blood flow due to splanchnic dilation > decreased GFR > decreased waste excretion
    - Diagnosis of exclusion. Will not be something you diagnose in the ED.
    - Treatment: call nephro and GI, d/c diuretics, increase MAP.
- Hepatopulmonary Syndrome
    - Triad of liver disease, intrapulmonary vasodilation, arterial O2 deficit
    - Can have dyspnea, tachypnea, hypoxemia, platypnea
    - Treatment: support with O2, may need tube

Lightning Lecture: Hernias (Dr. Coffman)

- Definition: when organ, intestine, or fatty tissue squeeze through weak spot in muscle/connective tissue
- Presents with bulge +/- reducible, pain, obstruction, systemic symptoms
- Locations
    - Inguinal → 75%
    - Ventral
    - Femoral
    - Obturator
- Three types
    - Reducible
    - Incarcerated → not easily reduced but no overlying skin changes, obstruction
    - Strangulated → skin changes, systemic symptoms, obstruction
- Inguinal → Direct vs indirect
- Ventral
    - Epi/hypogastric
    - Incisional
    - Spigelian
    - Umbilical
- Femoral
    - Female predilection
    - Prone to complications
    - Urgent intervention due to high complication rates
- Obturator
    - Urgent intervention
    - Howship-Romberg sign → inner thigh pain due to nerve impingement
- Richter hernia → antimesenteric border, no vomiting or obstruction, found late (ischemic)
- Diagnosis
    - US → can see defect, can use doppler
    - CT → gold standard
- Treatment
    - Can you reduce it?
    - NPO, pain control, apply ice packs early
    - Grasp and elongate hernia neck, apply firm, steady pressure proximally
    - Can place in trendelenburg position to help
    - Do not attempt if strangulated, concern for ischemia. Reintroduction of ischemic bowel into peritoneum can lead to sepsis.

PEM: GI Emergencies (Dr. Kopp)

- Case 1 (Appendicitis)
    - CC: CHI, vomiting, abdominal pain
    - Two days prior, CTH normal, given Zofran, did well since then.
    - No abdominal pain until fall, vomiting (NBNB) continued, no PO, BM yesterday.
    - Exam: vitals with temp 102.5F, slight tachy, TTP in RLQ
    - Pediatric Appendicitis Score
        - History (all 1 point each)
            - Anorexia
            - Fever > 38 C
            - NV
            - Migration of pain
        - Physical Exam (2 points each)
            - Peritoneal signs
            - RLQ tenderness
        - Labs
            - WBC > 10k or CRP > 1
            - ANC > 7500
    - Algorithm → imaging, surgery consult
    - Preschool children
        - More likely to have fever, abdominal tenderness
        - Less likely to have RLQ localization, anorexia
    - Ultrasound
        - Grade 1 → normal
        - Grade 2 → can't see, no secondary signs
        - Grade 3 → can't see, have secondary signs
        - Grade 4 → appendicitis
- Case 2 (Foreign body ingestion)
    - CC: influenza
    - Vitals okay
    - Fevers last week, dx with flu. Productive cough, with post-tussive emesis.
    - Abdomen soft, NTND.
    - Failed PO challenge
    - CXR with foreign body (button battery) in esophagus
        - Reportedly there for two days
    - Treatment (if in esophagus)
        - Urgent removal
        - If at home → honey 10 mL q10 minutes for six doses
        - At ER → sucralfate 1g q10 minutes
    - Treatment if past esophagus → depends on institution but symptoms usually gets removed
- Case 3 (Intussusception)
    - 19 month old with cc of "fussiness" since last night, started one hour after bedtime

    - No PMH, fully vaxxed. Previous episodes of constipation but not similar to this. Had normal BM earlier.
    - KUB → moderate stool burden, no dilated bowel, scattered gas pattern
    - US → target sign (intussusception)
    - Treatment → air enema
        - Need surgery present due to risk of perforation.
        - Six hour observation following
        - 10% recurrence rate overall
- Case 4 (Pyloric Stenosis)
    - 8 week old female with apneic episodes, happening frequently.
    - Vitals: RR 9, SpO2 61%
    - Responses to stimulation, pupils not pinpoint.
    - No co-sleeping, seemed today to have pauses in breathing.
    - No recent sick symptoms otherwise. Endorses may have been more sleepy today. No wet diapers today.
    - Cachectic appearing, nontoxic, no jaundice.
    - Born via C section at 36 weeks.
    - 100% with BVM, without apneic again.
    - Labs
        - VBG pH 7.59, CO2 59, HCO3 62
        - Lactate 9.6
        - Na 128, K 2.7, Cl 73
    - Seen previously for vomiting two weeks ago, sent home. Reportedly projectile vomiting.
    - Ultrasound → donut sign (pyloric stenosis)
        - Thickened muscle of the pylorus
    - Tidbits
        - Usually 3 to 5 weeks, males more likely
        - Family history
        - Forceful vomiting with each feed → HUNGRY
        - Exam: palpable “olive” in 1/7, “waves” over abdomen
        - Labs: hypochloremic, hypokalemic, metabolic alkalosis
        - IVF: NS bolus (es) → D5 ½NS + 20 KCl @ 1.5M
        - Surgery when electrolytes have stabilized

Liv-Er Let Die (Dr. Vanvleet)

- Case
  - 68 yo M to ED with AMS, abdominal pain, fever of 101.2
  - Exam: Jaundiced, abdomen tender to palpation
  - Fill: Nadolol, lactulose, rifaximin
- SBP
  - Infection of ascitic fluid
  - Mortality 20%
  - Translocation of bacteria + impaired immune system
  - Presents with pain, AMS, fever, hypotension
  - Para → 250 PMNs
  - Most are gram negative, can have healthcare associated SBP which are G+
  - Treatment: CTX if community acquired, zosyn if risk for MDRO, add MRSA coverage if history.
  - Albumin should be given, decreases mortality rate and incidence of renal failure
- Varices
  - Octreotide → splanchnic vasoconstriction, given as bolus followed by infusion
  - Vasoactive drugs decrease mortality, improve hemostasis.
  - Antibiotic prophylaxis shown to reduce mortality, bacterial infections, and rebleeding.
  - Acid reducing therapy should be given. No difference in mortality, rebleeding, transfusion, hospital LOS between BID and continuous infusion PPI.
- HRS
  - AKI seen in advanced liver disease.
  - Median survival is around 3 months.
  - Diagnosis of exclusion.
  - Portal hypertension causes release of NO, causing hypoperfusion that then leads to NE release which leads to renal vasoconstriction in the setting of global vasodilation.
  - Treatment: vasoactives with albumin.
  - Albumin shown to increase rates of response with vasoactive medications.

APAP/Acetaminophen/Tylenol/Paracetamol/Goodies Powder… (Dr. Eisenstat)

- History
    - APAP/phenacetin from late 1800s
    - Phenacetin removed from market 1970s because of renal injury, carcinogenesis, hemolysis
    - APAP first non-opioid, became primary analgesic/antipyretic following phenacetin removal
- Why APAP is the perfect toxin to treat
    - Easy to get, lots of data on it, antidote is cheap, can be bound by AC, can be dialyzed
- Tylenol comes in a billion formulations.
- How much is too much?
    - FDA max of 4 g in 24 hours, manufacturers have lowered to 3 g daily
    - 150 mg/kg in peds (acute)
    - 7.5 grams in adults (acute)
        - These are highly conservative
    - Chronic ingestions less understood
- Kinetics
    - Most absorbed in 1-2 hours
    - Peak serum by 4 hours, even in ER
    - Massive OD or opioid/anticholinergics may peak later
- Pathophysiology in overdose
    - Normally APAP undergoes addition of groups and metabolizes into non-toxic products
    - If these are overwhelmed, CYP2E1 produces NAPQI which is broken down. If this accumulates, it will cause hepatocyte necrosis.
        - NAPQI broken down with glutathione dependent pathway
        - NAC repletes glutathione stores.
- Signs and symptoms
    - Acute ingestion often asymptomatic
    - Nausea, vomiting most common
    - AST/ALT will rise before symptoms of liver failure
    - Early acidosis is BAD. Means likely massive acute ingestion
    - Chronic overdoses can present in fulminant liver failure
- Four stages of acetaminophen toxicity
- Rumack-Matthew Nomogram
- Cyanide killings in Chicago bring about safety seals.
- NAC - how it works
    - Works best in first 8 hours
    - Repletes glutathione stores lost in toxicity
    - Glutathione binds NAPQI and allows you to eliminate before it does damage
    - Also reduces oxidative stress
- Be careful with NAC dosing. Should be protocol. Can overdose
- There are both IV and PO formulations.

- Massive Ingestions
    - Treatment failure 5-10% with APAP over 300
    - Treatment failure 10-20% with APAP over 500
    - Not well defined
        - 30-50g
        - 250-500 mcg/ml APAP level
    - Several ways to increase NAC
        - Double the 16 hour maintenance bag
        - Give IV and PO at the same time (???)
- Fomepizole Usage
    - CYP2E1 inhibitor
    - Only useful earlier
    - Single loading 15 mg/kg
- Can dialyze
- Liver Transplant → Kings College Criteria