

GI BCPS Review—Key Points

GERD

- Alarm symptoms (dysphagia, odynophagia, bleeding, wt. loss, choking, chest pain) warrant immediate referral/invasive testing
- Response to appropriate tx considered diagnostic
- Most people with sx have normal esophageal mucosa on endosc.
- Do endoscopy in > 45 y.o., alarm sx, refractory to initial tx
- Ambulatory ph monitoring useful when no mucosal changes on endo but have cont. sx, when refractory to tx, or to monitor while on tx
- Tx
 - Nonpharmacologic counseling—unlikely to solely help but should do for all pts.—foods, positioning, avoid precipitant meds
 - Pharmacologic
 - Antacids and H2RA's ok but efficacy 20%
 - PPI's preferred if erosion seen
 - Watch forms that can be put in IV, NG, crushed, etc.

PUD

- Duodenal ulcer
 - H. pylori causes 95%, if present should always eradicate (increases risk of cancer)
 - more likely to have pain relieved by eating compared to gastric
- Gastric ulcer more likely pain exacerbated by eating
 - NSAIDs more common cause
- Risk factors for NSAID ulcer
 - Age > 60, use of steroids or warfarin or ASA, hx of ulcer or complications, hx of CV dz, RA, higher NSAID dosages and longer duration of use
- H. pylori testing
 - Invasive—if CLO test, should be off anti-acids for 1 week
 - Non-invasive
 - Serological—doesn't distinguish btwn active or past infection
 - Urea breath—need to be off anti-acids or antibiotics for 2 weeks before or 4 weeks following tx
 - Stool antigen—can be used to confirm eradication (wait for 4 weeks after tx to use)
- Primary prevention of NSAID-ulcers
 - Risk factor modification
 - PPI plus NSAID
 - Misoprostol
 - COX-2
 - Use of lower GI toxic NSAID (e.g. IBU or Diclofenac)
- Secondary prevention
 - Discontinue or lower dose
 - Tx h.pylori
 - PPI first choice for healing and preventing future
 - Misoprostol as effective as PPI for healing and preventing
 - H2RA's inferior to previous two options

UGIB (non-variceal)

- Mostly due to PUD, high mortality
- Predictors of persistent or recurrent GIB: age \geq 65, shock, comorbidities, initial low Hgb, coagulopathy, endo findings of active bleeding, clots, ulcer $>$ 2 cm
- Management:
 - Hemodynamics
 - Endo within 24 hrs.
 - Sclerotherapy + heat coagulation best
 - Either + drugs even better
 - IV pantoprazole CI fro 72 hrs.
 - H2RAs and octreotide not recommended!

IBD (UC and Crohn's)

- NSAIDs worsen, tobacco worsens Crohn's but betters UC
- General presenting sx: fever, abd pain, diarrhea (could be bloody), wt. loss
- Study 1st Table on page IX-15
- Remember that UC is more superficial and involves rectum and colon only while Crohns can be transmural and can extend throughout GI tract
- Both UC and Crohns classified in severity by mild, mod/severe, and fulminant. UC more based on amt. of stools per day and how bloody, while Crohns more based on severity of fever, abd pain, vomiting, and wt. loss
- High rates of relapse
- Managing UC
 - Classify distal dz (to splenic flexure) which can use topical only vs. extensive dz (requires systemic tx)
 - Mild dz
 - 1. topical aminosalicylates $>$ oral aminosalicylates + topical steroids
 - 2. Mesalamine PR
 - 3. refractory to above use PO prednisone
 - Maintenance—use aminosalicylates, NO steroids
 - Mod dz
 - 1. oral aminosalicylates, can use with topical therapy if distal dz OR infliximab
 - 2. add steroids PO
 - 3. refractory to steroids, use AZA or 6-MP (can produce steroid-reducing effects)
 - Maint.—all of the above except NO steroids
 - Severe
 - 7-10 days IV steroids
 - Infliximab
 - Refractory to steroids, use IV cyclosporine
 - Colectomy
- Managing Crohns
 - Mild
 - 1st line oral aminosalicylates
 - Flagyl, cipro, oral budesonide
 - Mod

- Oral steroids
- Infliximab
- Severe
 - IV steroids or cyclosporin or tacrolimus
- Maint.
 - NO STEROIDS
 - AZA/6-MP
 - Infliximab
- Drug issues
 - Watch sulfa allergy with sulfasalazine, no problem with mesalamine
 - Budesonide WAY more potent than prednisone, watch overlap when switch to budesonide
 - AZA/6MP take awhile to work, not good for acute tx, may have steroid-sparing effect, possible increased toxicity in those with reduced expression of TPMT, can cause pancreatitis, bone marrow suppression, and hepatotoxicity
 - Infliximab—works against TNF, good for fistula closure, can have infusion-related ADRs, delayed hypersensitivity, activation of latent infection (R/O TB!), contraindicated in Class III/IV HF, bone marrow suppression

Ascites

- SAAG > 1.1 indicative of ascites secondary to portal hypertension
- Can give albumin 8-10 gm/L removed >4L paracentesis

Encephalopathy

- Asterixis classic sign
- NH₃ levels will be high but don't follow for tx monitoring....doesn't correlate well with degree of CNS sx
- 1st line lactulose 45 mL q 1-2 hrs. until BM, titrate to 2-3 loose BM per day; also used for prevention
- Neomycin as effective as lactulose. Some is absorbed systemically so watch allergy and renal impairment
- Can use flagyl

Varices

- Band therapy better than sclerotherapy > nothing
- Vasopressin (constricts splanchnic blood flow) + NTG (to prevent some of effects on coronary constriction)—watch with CAD
- Octreotide and somatostatin
 - Reduces splanchnic blood flow by preventing vasodilation
 - Preferred with endoscopy x 5 days
- Can use TIPS, watch increase in encephalopathy and rebleeding
- Prim prevention
 - Non-selective BB significantly reduce first bleed incidence, no sig effect on mortality
 - Aim for HR 55 or 25% decr from baseline
 - Long acting nitrates possible alternative but not great evidence
- Sec prevention
 - BB 20% reduction in rebleed (not great), no effect on mortality

- Band ligation + BB best

SBP

- Ascetic fluids rarely positive, but + WBCs and PMNS > 250 diagnostic
- Mostly enterobacteriaceae (e.coli and kleb), some strept and staph
- High mortality, poorer outcomes with hepatorenal syndrome, low albumin, UGIB, encephalopathy
- Use 3rd gen cep (cefotaxime and ceftriaxone most studied), can use FQ
- Tx 5-10 days, avoid AG with incr SCr
- Albumin has been shown to decrease ARF and mortality when given with SBP (one study-1999)
- Prevention
 - If have one case, should have secondary prevention with bactrim or FQ

HAV

- RNA virus, mostly fecal-oral, mostly self-limited, supportive care
- Dx anti-HAV IgM or IgG
- Can pre-exposure vaccinate
- Post-exposure with IgG within 2 weeks

HBV

- DNA virus, mostly IV or sexual contact
- Higher risk of getting chronic with lower age
- Table on serologies on page IX-26, worry most about E Ag and Ab
- Dx of chronic if HbsAg + for 6 mo. And HBV DNA > 10⁵ /mL
- If HBeAg + and not decompensated, watch for 3-6 mo. To see if will self-convert to HBeAb +
- In general treat HBeAg (-) patients for longer than (+) patients
- If decompensated, don't use interferons
- If fail interferon, use RTIs (new one indicated not mentioned in PSAP is entecavir)
- Lamivudine has fast onset but high rate of resistance, can use adefovir if become resistant to lamivudine
- Use higher dose lamivudine for co-infected HIV patients
- Watch nasty ADRs of interferon (like depression, thyroid problems, diabetes, infections, myelosuppression)
- Pre-exposure—vaccination
- Post-exposure—vaccine and HBIG (within 7 days)

HCV

- RNA virus, mostly through blood
- Genotype 1 hardest to treat, have to assess treatment response at 12 weeks, if no response then stop treatment, and have to treat them longer than types 2 and 3 (48 weeks total vs. 24 weeks total)
- First-line is peg-interferon + ribavirin (higher doses used for type 1 vs. 2 or 3)
- Dx by HCV RNA viral load, anti-HCV Ab doesn't confer protection
- Tend to treat patients who have advanced histologic features on liver bx and high ALTs
- Watch anemias and renal function with ribavirin
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No vaccine or IG available for HCV

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