Common problems in Gastroenterology for Internal medicine

Julajak Limsrivilai
Siriraj Hospital
Outline: GI tract

Upper tract
- Dyspepsia
- GERD

Pancreaticobiliary
- Gall stone and CBD stone
- Acute cholecystitis
- Ascending cholangitis
- Acute pancreatitis

Lower tract
- IBS
- Chronic diarrhea
- FOBT
- CRC screening
Outline: Liver disease

Chronic liver disease
- Viral hepatitis
- Alcoholic hepatitis
- NAFLD
- Autoimmune liver disease
- Metabolic liver disease

Cirrhosis
- Portal hypertension
- Variceal bleeding
- Ascites and complication
- Hepatorenal syndrome
- Hepatocellular carcinoma
Ms YL, a 47 y/o woman, presents with dyspepsia for 2 months. She has no anemia or weight loss. Her brother is going to die from CA stomach. She has never taken any medication for this symptoms.

What’s the most appropriate management next?

1. Life style modification
2. PPI
3. Urea breath test and treat H.pylori if test +ve
4. EGD with rapid urease test
Dyspepsia

- pain or discomfort centered in the **upper abdomen**
  - epigastric pain
  - postprandial fullness
  - early satiation
  - Belching
  - nausea and vomiting
  - upper abdominal bloating
Dyspepsia

- Etiology

- Guideline
Etiology

Functional dyspepsia vs Organic dyspepsia

- Normal finding
- Nonerosive gastritis
- Erosive/Hemorrhagic gastritis
- Peptic ulcer
- Polyp
- Gastric cancer
Etiology: other causes of upper abdominal pain

- Chronic pancreatitis
- Pancreatic cancer
- Gallstones
- Liver tumor
Approach

Dyspepsia

R/O biliary colic

Alarming features (at any age)
Alarming features: if +ve → EGD

1. GI blood loss
   - Hematemesis
   - Melena
   - IDA

2. Weight loss

3. Dysphagia

4. Persistent vomiting

Familial history of CA stomach
Approach

Dyspepsia

R/O biliary colic

Alarming features (at any age)

Yes

Extra-luminal

US/CT or refer to GI

Luminal organ

EGD/refer to GI

Structural disease

Eg. PU, cancer

Treat appropriately

No
Approach

Dyspepsia

R/O biliary colic

Alarming features (at any age)

Yes

Extra-luminal
US/CT or refer to GI
Structural disease
Eg. PU, cancer
Treat appropriately

No

Luminal organ
EGD/refer to GI

review med/diet
Medications and Diet

- **NSAIDs and ASA**
- ATB: penicillin, sulphonamides, macrolides, doxycycline, tetracycline etc.
- Hormone: Oral anti-diabetics, estrogen, corticosteroids
- CV drugs: digoxin, CCB
- K supplement
- Alendronate
- Bronchodilators: theophylline
Approach

Dyspepsia

R/O biliary colic

Alarming features (at any age)

Extra-luminal

Yes

US/CT or refer to GI

Luminal organ

EGD/refer to GI

Age of onset > 55

Yes

Not resolved

No

review med/diet

Resolved

Uncomplicated dyspepsia

• Diet advice
• LSM
• Empirical Rx or Test and treat H.pylori

Not improved

Normal/
Gastritis –
Dx NUD

Treat appropriately

Structural disease
Eg. PU, cancer

Reassure
# Management of functional dyspepsia

> Reassure = most important

<table>
<thead>
<tr>
<th>Treatment</th>
<th>NNT</th>
<th>Grade quality</th>
</tr>
</thead>
<tbody>
<tr>
<td>TCA: amitriptyline</td>
<td>2</td>
<td>Very low</td>
</tr>
<tr>
<td>Prokinetic: domperidone</td>
<td>5</td>
<td>Low</td>
</tr>
<tr>
<td>H2RA</td>
<td>8</td>
<td>Low</td>
</tr>
<tr>
<td>PPI</td>
<td>9</td>
<td>Moderate</td>
</tr>
<tr>
<td>H. Pylori eradication</td>
<td>14</td>
<td>High</td>
</tr>
</tbody>
</table>

Moayyedi P, Curr Opinion 2012
Who should be received H. pylori eradication?
### H. Pylori: Indication for eradication

<table>
<thead>
<tr>
<th>Condition</th>
<th>Level of Evidence</th>
<th>Level of Control</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ptoolที่พบหลักฐานว่ามีผลที่กระทำต่ออาหารหรือลำไส้เล็กในตัวุน (ไม่ว่าผลจะหายแล้วหรือไม่)</td>
<td>ระดับ 1</td>
<td>ระดับ 1</td>
</tr>
<tr>
<td>Atrophic gastritis</td>
<td>ระดับ 2</td>
<td>ระดับ 2</td>
</tr>
<tr>
<td>Hemorrhagic / erosive gastritis</td>
<td>ระดับ 2</td>
<td>ระดับ 2</td>
</tr>
<tr>
<td>MALToma</td>
<td>ระดับ 1</td>
<td>ระดับ 1</td>
</tr>
<tr>
<td>After gastric cancer resection</td>
<td>ระดับ 2</td>
<td>ระดับ 2</td>
</tr>
</tbody>
</table>

**Additional Indications**

<table>
<thead>
<tr>
<th>Condition</th>
<th>Level of Evidence</th>
<th>Level of Control</th>
</tr>
</thead>
<tbody>
<tr>
<td>ผู้ที่ต้องรับจำหนายยาแอสไตรินในระยะยาว (หลังจากเกิดผลหรือการแพร่กระจายจากผลที่กระทำต่ออาหารหรือลำไส้เล็กในตัวุน)</td>
<td>ระดับ 2</td>
<td>ระดับ 2</td>
</tr>
<tr>
<td>ผู้ที่ต้องรับจำหนายยาแอสไตรินในระยะยาว ในอัตราที่ต่ำกว่าหรือเท่ากันกับการรับยาแอสไตรินในการรักษาโรคต่างๆ</td>
<td>ระดับ 2</td>
<td>ระดับ 2</td>
</tr>
<tr>
<td>ผู้ที่ต้องรับจำหนายยา NSAIDs ในระยะยาว</td>
<td>ระดับ 1</td>
<td>ระดับ 1</td>
</tr>
<tr>
<td>ผู้ที่มีเมตาบอลิซึมยาแอสไตรินในระยะยาว</td>
<td>ระดับ 2</td>
<td>ระดับ 2</td>
</tr>
<tr>
<td>ผู้ป่วยแสดงค่าของจำนวน (หลังจากได้รับค่าอินชัยและแนะนำจากการแพทย์แล้วได้ผลยิ่ง)</td>
<td>ระดับ 4</td>
<td>ระดับ 4</td>
</tr>
</tbody>
</table>
Mrs YL, a 47 y/o st_pid woman, presents with dyspepsia for 2 months. She has no anemia or weight loss. Her brother is going to die from CA stomach. She has never taken any medication for this symptoms.

What’s the most appropriate management next?

1. Life style modification
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**Lower tract**
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- Chronic diarrhea
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- CRC screening

**Pancreaticobiliary**
- Gall stone and CBD stone
- Acute cholecystitis
- Ascending cholangitis
- Acute pancreatitis

**CRC screening**
GERD

A condition which develops when the reflux of stomach contents causes troublesome symptoms and/or complications

**Esophageal syndromes**
- Symptomatic syndromes
  - Typical reflux syndrome
  - Reflux chest pain syndrome
- Syndromes with esophageal injury
  - Reflux esophagitis
  - Reflux stricture
  - Barrett’s esophagus
  - Adenocarcinoma

**Extra-esophageal syndromes**
- Established association
  - Reflux cough
  - Reflux laryngitis
  - Reflux asthma
  - Reflux dental erosions
- Proposed association
  - Sinusitis
  - Pulmonary fibrosis
  - Pharyngitis
  - Recurrent otitis media
GERD

Esophageal Manifestation

NERD 50-70%

24-hr pH

Acid reflux

Nonacid reflux

Esophageal hypersensitivity

Reflux esophagitis 30-40%

Barrett’s & CA 5%

Extraesophageal Manifestation
Symptoms suggestive of GERD

Alarm

Yes

EGD/Re-evaluation
Alarm features

Complication of GERD

Erosive esophagitis
Odynophagia
GI blood loss

Stricture
Dysphagia

Cancer
Dysphagia
Weight loss
Alarming features

Persistent vomiting
Symptoms suggestive of GERD

- Typical
  - LSM plus Standard dose PPI 4 wk
  - Symptoms free
    - Stop
    - Recurrent symptoms
      - On-demand/Intermittent Rx
  - No
- Atypical
  - LSM plus Double dose PPI 2 wk (consider 4-12 wks for atypical GERD)
  - Symptom persist
    - Maintain for at least 4 wks
      - No symptom
      - Re-evaluation
- Alarm
  - Yes
    - • Dysphagia
    - • Odynophagia
    - • Frequent vomiting
    - • GI bleed / anemia
    - • Weight loss
    - EGD/Re-evaluation
Outline: GI tract

**Upper tract**
- Dyspepsia
- GERD

**Pancreaticobiliary**
- Gall stone and CBD stone
- Acute cholecystitis
- Ascending cholangitis
- Acute pancreatitis

**Lower tract**
- Irritable bowel syndrome
- Chronic diarrhea
- FOBT
- CRC screening
Irritable bowel syndrome (IBS)

IBS ≠ IBD

IBS = inflammatory bowel syndrome
IBD = irritable bowel disease

???
Irritable bowel syndrome

• Recurrent abdominal pain or discomfort at least 3 days a month in the past 3 months

• Associated with 2 or more of:
  • Improved with defecation
  • Onset associated with a change in frequency of stool
  • Onset associated with a change in form of stool

• Constipation-predominant IBS (IBS-C)
• Diarrhea-predominant IBS (IBS-D)
• Mixed bowel pattern IBS (IBS-M)

Longstreth GF. *Gastroenterology* 2006;130:1480-91
Differential diagnosis

CA colon

- Age > 50 yr or have Familial Hx of CRC
- Bloody diarrhea, bowel habit change
- Anemia
- Weight loss

Chronic colitis eg. IBD

- Mucous bloody diarrhea
- Fever
- Anemia
- Weight loss
Alarming features

- New onset of symptoms at 50 years or older
- Bloody stools
- Nocturnal diarrhea
- Anemia
- Fever
- Unintentional weight loss
- Family history of colon cancer or IBD
Investigation

Routine diagnostic testing:
- CBC, serum chemistries, TFT
- Stool for ova and parasites, FOBT
- Abdominal imaging

No alarming features

<table>
<thead>
<tr>
<th>Organic disease</th>
<th>IBS</th>
<th>Normal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Colitis/IBD</td>
<td>0.51-0.98</td>
<td>0.3-1.2</td>
</tr>
<tr>
<td>Colon cancer</td>
<td>0-0.51</td>
<td>0-6</td>
</tr>
<tr>
<td>Thyroid dis</td>
<td>4.2</td>
<td>5-9</td>
</tr>
<tr>
<td>Lactose maldigestion</td>
<td>38</td>
<td>26</td>
</tr>
</tbody>
</table>
Investigation

Alarming features

• Blood test: CBC, serum chemistries, TFT
• Stool test: ova, parasite

Diarrhea

Colonoscopy

Constipation

BE or colonoscopy
## Treatment

<table>
<thead>
<tr>
<th>Drug class</th>
<th>Generic name and dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Antispasmodics</td>
<td>Hyoscine sulfate (0.125 mg sublingually or by mouth up to four times daily)</td>
</tr>
<tr>
<td>Dicyclomine (10–20 mg by mouth twice daily or up to four times daily)</td>
<td></td>
</tr>
<tr>
<td>Clidinium + chlordiazepoxide (2.5 mg/5 mg, 1–2 tablets up to three or four times daily)</td>
<td></td>
</tr>
<tr>
<td>Hyoscine + scopolamine + atropine + phenobarbital (1–2 tablets up to three or four times daily)</td>
<td></td>
</tr>
<tr>
<td>Tricyclic antidepressants</td>
<td>Amitriptyline (10–150 mg at night)</td>
</tr>
<tr>
<td>Doxepin (10–150 mg at night)</td>
<td></td>
</tr>
<tr>
<td>Imipramine (10–150 mg at night)</td>
<td></td>
</tr>
<tr>
<td>Clomipramine (25–100 mg at night)</td>
<td></td>
</tr>
<tr>
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<td></td>
</tr>
<tr>
<td>Desipramine (10–150 mg at night)</td>
<td></td>
</tr>
<tr>
<td>Nortriptyline (10–150 mg at night)</td>
<td></td>
</tr>
<tr>
<td>Selective serotonin reuptake inhibitors</td>
<td>Fluoxetine (10–40 mg daily)</td>
</tr>
<tr>
<td>Citalopram (20 mg daily)</td>
<td></td>
</tr>
<tr>
<td>Paroxetine (20–50 mg daily)</td>
<td></td>
</tr>
<tr>
<td>Sertraline (25–100 mg daily)</td>
<td></td>
</tr>
<tr>
<td>Escitalopram (10 mg daily)</td>
<td></td>
</tr>
<tr>
<td>5-HT₄ agonist</td>
<td>Tegaserod (6 mg twice daily)</td>
</tr>
</tbody>
</table>
## Treatment

<table>
<thead>
<tr>
<th>Drug class</th>
<th>Generic name (dose)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Antidiarrheals</td>
<td>Loperamide (1–8 mg four times daily in divided doses)</td>
</tr>
<tr>
<td></td>
<td>Diphenoxylate (5 mg up to four times daily)</td>
</tr>
<tr>
<td>5-HT₃ antagonist</td>
<td>Alosetron (0.5–1.0 mg twice daily)</td>
</tr>
<tr>
<td>Tricyclic antidepressants</td>
<td>Amitriptyline (10–150 mg at night)</td>
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<tr>
<td></td>
<td>Desipramine (10–150 mg at night)</td>
</tr>
<tr>
<td></td>
<td>Nortriptyline (10–150 mg at night)</td>
</tr>
<tr>
<td>Antibiotics</td>
<td>Rifaximin (400–550 mg three times daily)</td>
</tr>
</tbody>
</table>
# Treatment

## Constipation

<table>
<thead>
<tr>
<th>Drug class</th>
<th>Generic name (dose)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bulking agents</td>
<td>Psyllium (2.5–30 g daily in divided doses)</td>
</tr>
<tr>
<td></td>
<td>Methylcellulose (500 mg, 1–2 tablespoons daily or up to three times daily)</td>
</tr>
<tr>
<td></td>
<td>Calcium polycarbophil (1,250 mg twice or four times daily)</td>
</tr>
<tr>
<td>Osmotic laxatives</td>
<td>Milk of magnesia (400 mg/5 ml, 10–20 ml up to four times daily)</td>
</tr>
<tr>
<td></td>
<td>Lactulose (10–20 g/15–30 ml daily)</td>
</tr>
<tr>
<td></td>
<td>Polyethylene glycol (17 g in 237 ml solution daily)</td>
</tr>
<tr>
<td>Stimulant laxatives</td>
<td>Senna (15 mg daily)</td>
</tr>
<tr>
<td></td>
<td>Diphenylmethane derivatives (for example, bisacodyl at a dose of 10 mg, 1–2 tablets daily or 1 suppository daily)</td>
</tr>
<tr>
<td>Emollient laxatives</td>
<td>Docusates (100 mg, 1–3 tablets daily)</td>
</tr>
<tr>
<td></td>
<td>Mineral oil (5–10 cm³ daily)</td>
</tr>
<tr>
<td>5-HT₄ agonist</td>
<td>Tegaserod (6 mg twice daily)</td>
</tr>
<tr>
<td>Chloride channel activator</td>
<td>Lubiprostone (8 µg twice daily with meals)</td>
</tr>
</tbody>
</table>
Outline: GI tract

**Upper tract**
- Dyspepsia
- GERD

**Pancreaticobiliary**
- Gall stone and CBD stone
- Acute cholecystitis
- Ascending cholangitis
- Acute pancreatitis

**Lower tract**
- IBS
- Chronic diarrhea
- FOBT and occult GI bleeding
- CRC screening
Chronic diarrhea
SIBO

Drug

Endocrine
Small bowel lesion

**Clue**

- Voluminous watery diarrhea
- Malabsorption, severe
- Hypoalbuminemia
- Mid abdominal colicky pain
- Hypokalemia
**Small Bowel lesions**

<table>
<thead>
<tr>
<th>Infections</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Infection</strong></td>
</tr>
<tr>
<td>Malignancy: IPSID, lymphoma</td>
</tr>
<tr>
<td><strong>Inflammation</strong></td>
</tr>
<tr>
<td>Tropical sprue</td>
</tr>
<tr>
<td>Crohn’s disease</td>
</tr>
<tr>
<td>Eosinophilic enteritis</td>
</tr>
<tr>
<td>Drugs: colchicine, ponstand</td>
</tr>
<tr>
<td><strong>Infiltrative</strong></td>
</tr>
<tr>
<td>Amyloidosis</td>
</tr>
<tr>
<td><strong>Lymphangiectasia</strong></td>
</tr>
</tbody>
</table>

**Infections**

- **Bacteria**
  - MAC
  - MTB
- **Virus**
  - CMV
- **Helminths**
  - *Capillaria philippinensis*
  - *Strongyloides stercoralis*
- **Protozoa**
  - *Cryptosporidium parvum*
  - *Cyclospora cayetanensis*
  - *Encephalitozoon intestinalis*
  - *Enterocytozoon bieneusi*
  - *Giardia lamblia*
  - *Isospora belli*
Suspected SB lesion

Chronic diarrhea: Approach

Stool exam, endoscopy (Push entero, EGD) with biopsy
Colonic and Terminal Ileal lesion

**Clue**

- Chronic mucous bloody diarrhea (common in left-sided lesion)
- Tenemus (rectal involvement)
- Lower abdominal pain
- WBC in stool
Colonic and Terminal Ileal Lesion

- **Infection**
  - Bacteria: C. difficile, TB
  - Virus: CMV, herpes simplex
  - Parasite and protozoa: amebic

- **Cancer**
  - Adenocarcinoma, lymphoma

- **Inflammation**
  - IBD: UC, CD, microscopic colitis
  - Eosinophilic colitis
  - Autoimmune, vasculitis esp. Behcet

- **Radiation**
- **Drug induced**

**Stool exam, colonoscopy with biopsy**
Suspected SB lesion

Stool exam, endoscopy (Push entero, EGD) with biopsy

Suspected LB lesion

Stool exam, colonoscopy with biopsy
SIBO

Endocrine

Drug

LB

SB

P
Pancreatic disease

Clue

• Frank steatorrhea
• Upper abdominal pain
• DM
• Weight loss
• Alcohol drinking
Diagnosis of CP

Plain Film
- Sensitivity (S) ~50%
- Specificity (Sp) 95%

US
- Sensitivity (S) 48-96%
- Specificity (Sp) 75-90%

CT Scan
- Sensitivity (S) 56-95%
- Specificity (Sp) 85-100%

MRCP
- Sensitivity (S) 88-91%
- Specificity (Sp) 92-98%

ERCP
- Sensitivity (S) 68-100%
- Specificity (Sp) 89-100%

EUS
- Sensitivity (S) 85-100%
- Specificity (Sp) 85-100%
Chronic diarrhea: Approach

- Suspected SB lesion: Stool exam, endoscopy (Push entero, EGD) with biopsy
- Suspected LB lesion: Stool exam, colonoscopy with biopsy
- Suspected pancreatic lesion: Imaging
Small Intestinal Bacterial Overgrowth (SIBO)

Definition:
Jejunal bacteria > $10^5$ bact/ml

Stomach:
H. pylori

Prox SB:
$10^2$-$10^3$ bact/ml

Ileum:
$10^8$ bact/ml

Colon:
$10^{10}$-$10^{11}$ bact/ml

400-500 species including
- Bacteroides
- Eubacterium
- Peptostreptococcus
- Bifidobacterium
- Ruminococcus
- Bacillus
- Fusobacterium
- Clostridium
- Lactobacillus
- Enterococcus
- Enterobacter

Oral cavity:
200 species
## SIBO: Etiology

### Intestinal stasis

**Anatomic:**
- stricture (e.g. Crohn’s disease, radiation enteritis)
- Diverticulosis
- End-to-side enteroenteric anastomosis

**Motility disorder**
- Scleroderma
- Diabetic autonomic neuropathy

### Loss IC valve

- Fistulae
- Resection of IC valve

### Hypochlorhydria

- Chronic atrophic gastritis
- Medication

### Immunodeficiency

- Primary
- Secondary: AIDS
SIBO: Pathophysiology

- ↑ SB bacteria
  - Deconjugate bile acid
    - Steatorrhea
  - Bact consume B12
    - Megaloblastic anemia
  - SB mucosal injury
    - Carbo intolerance, hypoprotein

- Spare K, folate
Chronic diarrhea: Approach

- Suspected SB lesion: Stool exam, endoscopy (Push entero, EGD) with biopsy
- Suspected LB lesion: Stool exam, colonoscopy with biopsy
- Suspected pancreatic lesion: Imaging
- Suspected SIBO: Lactulose/glucose $\text{H}_2$ breath test
Chronic diarrhea: Approach

**Suspected SB lesion**
- Stool exam, endoscopy (Push entero, EGD) with biopsy

**Suspected LB lesion**
- Stool exam, colonoscopy with biopsy

**Suspected pancreatic lesion**
- Imaging

**Suspected SIBO**
- Lactulose/glucose $H_2$ breath test

**Suspected drug/endocrine**
- Stop drug, endocrine w/o
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Question

Which of the following statements about gFOBT (guiac test) is true?

1. Avoid Fe supplement before testing
2. Lower sensitivity than immunohistochemistry FOBT for upper GI bleeding
3. More than one sample of stools can increased test sensitivity
4. Vitamin C ingestion causes false-positive result
5. is recommended for annually surveillance in HNPCC family
FOBT
Pancreatic protease

**Hb**

Gastric pepsin protease

Reabsorption in prox intestine

15%

Having peroxidase activity

intestinal converted fraction

Colonic bacteria (1-99%)

No peroxidase activity

Porphyryns

Iron

Pancreatic & intestinal protease

Colonic bacteria

Immunochemical

globin

Heme-porphyryin

digestion

### Fecal occult blood

<table>
<thead>
<tr>
<th></th>
<th>Guaiac</th>
<th>Heme-Porphyrin</th>
<th>iFOBT</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>False positives</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Animal Hb</td>
<td>++++</td>
<td>++++</td>
<td>0</td>
</tr>
<tr>
<td>• Dietary peroxidase</td>
<td>+++</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td><strong>False negatives</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Vitamin C</td>
<td>++</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

High false +ve and –ve
FOBT and bleeding site

Sites of Gastrointestinal Bleeding

Upper gastrointestinal tract
Porphyrins, partially degraded heme, degraded globin

Middle gastrointestinal tract
Porphyrins, partially degraded heme, partially degraded globin

Lower gastrointestinal tract
Intact heme and intact globin

Relative Likelihood of a Positive Fecal Occult-Blood Test

- Guaiac-based
- Heme–porphyrin
- Immunochemical

Rockey DC, NEJM 1999
Fecal occult blood test: indication

Colorectal cancer screening:
Decreased mortality from CRC

Not indicate in symptomatic patients:
IDA, abdominal pain

- Low sensitivity
- Low specificity
### Role of FOBT in patients with IDA

<table>
<thead>
<tr>
<th>Blood Ingestion</th>
<th>CA Colon</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>60 cc blood ingestion</strong></td>
<td><strong>CA colon</strong></td>
</tr>
<tr>
<td>gFOBT</td>
<td>16%</td>
</tr>
<tr>
<td>iFOBT</td>
<td>2%</td>
</tr>
<tr>
<td>gFOBT</td>
<td>33-50%</td>
</tr>
<tr>
<td>iFOBT</td>
<td>50-75%</td>
</tr>
</tbody>
</table>

*No role of FOBT in the evaluation of IDA*

- Rockey DC, Am J Gastroenterol 1999
- Lieberman DA. NEJM 2009
Iron deficiency anemia

IDA

Men, postmenopausal women

GI tract evaluation

Premenopausal women

GYN consultation

GI tract evaluation if +ve risk
183 patients met criteria for IBS

15 patients had positive FOBT

4 patients (2.2%) had lesions found

- 1 Hemorrhoids
- 2 anal fissures
- 1 Melanosis coli

Tolliver BA, Am J Gastroenterol 1994

+ve FOBT with Abdominal pain
1. Globus sensation (Dysphagia)
2. Evidence of gastrointestinal blood loss + melena (Malignancy, hemorrhage, ulceration)
3. Unexplained weight loss
4. Persistent vomiting

No role of FOBT

False positives and false negatives are common in the diagnosis of IBS (see Table 1). The accuracy of alarm features is disappointing. For example, weight loss, nocturnal pain offer little discriminative value in distinguishing patients with IBS from those with organic diseases. Whereas anemia and weight loss have poor sensitivity for organic diseases, they offer very good specificity. As such, in patients who fulfill symptom-based criteria of IBS, the absence of selected alarm features, including anemia, weight loss, and a family history of colorectal cancer, inflammatory bowel disease, or celiac sprue, should reassure the clinician that the diagnosis of IBS is correct.
Which of the following statement about gFOBT (guiac test) is true?

1. Avoid Fe supplement before testing
2. Lower sensitivity than immunohistochemistry FOBT for upper GI bleeding
3. More than one sample of stools can increased test sensitivity
4. Vitamin C ingestion causes false-positive result
5. is recommended for annually surveillance in HNPCC family
Outline : GI tract

Upper tract
- Dyspepsia
- GERD

Pancreaticobiliary
- Gall stone and CBD stone
- Acute cholecystitis
- Ascending cholangitis
- Acute pancreatitis

Lower tract
- IBS
- Chronic diarrhea
- FOBT
- CRC screening
Mr. CHL, a 57 year-old man, has recently been diagnosed with advance stage colon cancer. He concerns this malignant disease might have genetic transmission. What would you recommend to his 30 yr-old son?

1. Colonoscopy now
2. Colonoscopy at age 40
3. Colonoscopy at age 50
4. FOBT at age 40
5. FOBT at age 50
Colon cancer screening
Clinical manifestation of CRC

- Polyp
- Advanced polyp
- Early stage CA
- Late stage CA

- Asymptomatic
- Occult bleed
- Occult/Overt bleed
- Obstruction
Benefit of early detection

Polyp removal: prevent cancer

5 yr survival

90%  60%  10%

Polyp  Advanced polyp  Early stage CA  Late stage CA
Screening tools

**Stool test**
- FOBT
- Fecal DNA test

**Endoscopy**
- Flexible sigmoidoscopy
- Colonoscopy
- Capsule colonoscopy

**Imaging**
- CT colonoscopy
- Barium enema
## Average risk: Conclusion

<table>
<thead>
<tr>
<th>Test</th>
<th>Sensitivity</th>
<th>Convenient, safety</th>
<th>Cost</th>
<th>Study decrease CRC/mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Advanced adenoma</td>
<td>Cancer</td>
<td></td>
<td></td>
</tr>
<tr>
<td>gFOBT (3 samples)</td>
<td>11</td>
<td>33-50</td>
<td>+++</td>
<td>30 B/sample</td>
</tr>
<tr>
<td>iFOBT (1-3 samples)</td>
<td>20-50</td>
<td>60-85</td>
<td>+++</td>
<td>70 B/sample</td>
</tr>
<tr>
<td>Sigmoidoscopy</td>
<td>70</td>
<td>&gt; 95 (distal)</td>
<td>++</td>
<td>6,000 B</td>
</tr>
<tr>
<td>Colonoscopy</td>
<td>88-98</td>
<td>&gt; 95</td>
<td>+</td>
<td>8,000 B</td>
</tr>
<tr>
<td>CT colonography</td>
<td>80 : &gt; 9 mm</td>
<td>&gt; 90</td>
<td>+, ++</td>
<td>13,000 B</td>
</tr>
<tr>
<td></td>
<td>60 : 6-9 mm</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
## Average risk: guideline recommendation

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>gFOBT 3 specimens</td>
<td>ทุก 1 ปี</td>
<td>ทุก 1 ปี</td>
<td>ทุก 1 ปี (ทางเลือก)</td>
<td>ทุก 1-2 ปี</td>
</tr>
<tr>
<td>iFOBT 1-2 specimens</td>
<td>ทุก 1 ปี</td>
<td>ทุก 1 ปี</td>
<td>ทุก 1 ปี (แนะนำ)</td>
<td>ทุก 1-2 ปี</td>
</tr>
<tr>
<td>Sigmoidoscopy</td>
<td>ทุก 5 ปี</td>
<td>ทุก 5 ปี</td>
<td>ทุก 5-10 ปี (ทางเลือก)</td>
<td>ทุก 5 ปี</td>
</tr>
<tr>
<td>+ FOBT ทุก 3 ปี</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Colonoscopy</td>
<td>ทุก 10 ปี</td>
<td>ทุก 10 ปี</td>
<td>ทุก 10 ปี (แนะนำ)</td>
<td>ทุก 10 ปี</td>
</tr>
<tr>
<td>CT colonography</td>
<td>ทุก 5 ปี</td>
<td>ทุก 5 ปี</td>
<td>ทุก 5 ปี (ทางเลือก)</td>
<td>-</td>
</tr>
<tr>
<td>Barium enema</td>
<td>-</td>
<td>ทุก 5 ปี</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

---

[^6]: USPSTF 2008

[^7]: Multi-society Joint Guideline 2008

[^8]: ACG 2008

[^9]: Asia Pacific 2008

---

**Start at 50 yr**

+ve colonoscopy

---
Familial colorectal cancer

![Relative risk of CRC](chart1)

- no FH of CRC or adenoma
- FH adenoma
- 1st degree relative with CRC
- Dx CRC < age 45
- > 1 relative with CRC

![Cumulative incidence](chart2)

Cumulative incidence (cases/10,000)

<table>
<thead>
<tr>
<th>Age (yr)</th>
<th>Family history</th>
<th>No family history</th>
</tr>
</thead>
<tbody>
<tr>
<td>30</td>
<td></td>
<td></td>
</tr>
<tr>
<td>35</td>
<td></td>
<td></td>
</tr>
<tr>
<td>40</td>
<td></td>
<td></td>
</tr>
<tr>
<td>45</td>
<td></td>
<td></td>
</tr>
<tr>
<td>50</td>
<td></td>
<td></td>
</tr>
<tr>
<td>55</td>
<td></td>
<td></td>
</tr>
<tr>
<td>60</td>
<td></td>
<td></td>
</tr>
<tr>
<td>65</td>
<td></td>
<td></td>
</tr>
<tr>
<td>70</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
# Familial colorectal cancer

<table>
<thead>
<tr>
<th>RR</th>
<th>Age to begin</th>
<th>Test</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Either CRC or AP in</td>
<td>4x</td>
<td>• 40 years, or Colonoscopy</td>
<td>Every 5 years</td>
</tr>
<tr>
<td>• 1&lt;sup&gt;st&lt;/sup&gt; degree &lt; 60 yr or</td>
<td>• 10 years</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• 2 or more 1&lt;sup&gt;st&lt;/sup&gt; degree at any age</td>
<td>before the youngest</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Either CRC or AP in</td>
<td>2-2.5x</td>
<td>• 40 years</td>
<td>Screening options as recommended for average-risk</td>
</tr>
<tr>
<td>• 1&lt;sup&gt;st&lt;/sup&gt; degree age &gt; 60 yr or</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• 2 or more 2&lt;sup&gt;nd&lt;/sup&gt; degree</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
## Hereditary colon cancer

<table>
<thead>
<tr>
<th>Who should be screened</th>
<th>Age to begin</th>
<th>Tool for screening and age to stop</th>
</tr>
</thead>
<tbody>
<tr>
<td>FAP</td>
<td>Age 10 to 12 years</td>
<td>- Annual FSIG or colonoscopy until age 30 years, every 3-5 years thereafter until age 60</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HNPCC</td>
<td>Age 20 to 25 years, or 10 years before the youngest case</td>
<td>- At least biennial colonoscopy, until age 40 years, annually thereafter to age 70-75 years or until co-morbidity makes it clinically inappropriate</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
# Hereditary colon cancer

<table>
<thead>
<tr>
<th>Who should be screen</th>
<th>Age to begin</th>
<th>Tool for screening and age to stop</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>FAP</strong></td>
<td><strong>Age 10 to 12 years</strong></td>
<td><strong>Annual FSIG or colonoscopy</strong> until age 30 years, -every 3-5 years thereafter until age 60</td>
</tr>
<tr>
<td>-mutation carrier</td>
<td></td>
<td></td>
</tr>
<tr>
<td>-family members at 50% risk if no mutation identified</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>HNPCC</strong></td>
<td><strong>Age 20 to 25 years, or 10 years before the youngest case</strong></td>
<td><strong>At least biennial colonoscopy</strong>, until age 40 years, -annually thereafter to age 70-75 years or until co-morbidity makes it clinically inappropriate</td>
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<tr>
<td>-mutation carrier</td>
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<td></td>
</tr>
<tr>
<td>-family members at 50% risk if no mutation identified</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**AD pattern**
## Hereditary colon cancer

<table>
<thead>
<tr>
<th>Who should be screened</th>
<th>Age to begin</th>
<th>Tool for screening and age to stop</th>
</tr>
</thead>
</table>
| **FAP**                | Age 10 to 12 years | -Annual FSIG or colonoscopy until age 30 years,  
every 3-5 years thereafter until age 60 |
| -mutation carrier      |              |                                   |
| -family members at 50% risk if no mutation identified | | |
| **HNPCC**              | Age 20 to 25 years, or 10 years before the youngest case | -At least biennial colonoscopy, until age 40 years,  
annually thereafter to age 70-75 years or until co-morbidity makes it clinically inappropriate |
| -mutation carrier      |              |                                   |
| -family members at 50% risk if no mutation identified | | |

**AD pattern**
FAP
HNPCC

Not polyposis

Extracolonic cancers

Overall 2-20% of cases

Most common: endometrial carcinoma (60% of female)

Extra-colonic cancer

GI: stomach, small bowel (stomach to rectum)

GU (sex): endometrium, ovary

GU (uro): transitional cell carcinoma of the renal pelvis or ureter

Hepatobiliary system, pancreas
Mr. CHL, a 57 year-old man, has recently been diagnosed with advance stage colon cancer. He concerns this malignant disease might have genetic transmission. What would you recommend to his 30 yr-old son?

1. Colonoscopy now
2. Colonoscopy at age 40
3. Colonoscopy at age 50
4. FOBT at age 40
5. FOBT at age 50
Outline: GI tract

**Upper tract**
- Dyspepsia
- GERD

**Pancreaticobiliary**
- Gall stone and CBD stone
- Acute cholecystitis
- Ascending cholangitis
- Acute pancreatitis

**Lower tract**
- IBS
- Chronic diarrhea
- FOBT
- CRC screening
A 40 year-old woman presents with acute epigastric pain for 4 hr. LFTs: TB 0.6, DB 0.4, AST 350, ALT 320, ALP 120, A/G 4.4/2.6. U/S reveals gall stone, no biliary duct dilatation.

What’s the most appropriate next management after controlling her abdominal pain?

1. check for HAV, HBV, and HEV infection
2. LC
3. ERCP
4. Endoscopic ultrasonography
Gall stone and complications
Asymptomatic GS 60-80%
Acute cholecystitis 10%
Biliary pain (with ↑ AST/ALT)
Obstructive jaundice
Cholangitis
5%
GS pancreatitis <5%
Gall stone and CBD stone
<table>
<thead>
<tr>
<th>References</th>
<th>Characteristic</th>
<th>No. of cases</th>
<th>Average follow-up period (years)</th>
<th>No. of acute cholecystitis cases (%)</th>
<th>Only those with remarkable jaundice cases (%)</th>
<th>Cholangitis</th>
<th>Cholecystitis</th>
<th>Gallbladder cancer</th>
</tr>
</thead>
<tbody>
<tr>
<td>Comfort et al.</td>
<td>Asymptomatic</td>
<td>112</td>
<td>15</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Lund</td>
<td>Asymptomatic</td>
<td>95</td>
<td>13</td>
<td>?</td>
<td>?</td>
<td>1(?)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Gracie et al.</td>
<td>Asymptomatic</td>
<td>123</td>
<td>11</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>McSherry et al.</td>
<td>Asymptomatic</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Friedman et al.</td>
<td>Asymptomatic</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Thistle et al.</td>
<td>Asymptomatic</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Wenckert et al.</td>
<td>Mild symptomatic</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>3</td>
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<tr>
<td>Ralston et al.</td>
<td>Mild symptomatic</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>2</td>
<td></td>
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<tr>
<td>Friedman et al.</td>
<td>Mildly symptomatic</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Newman et al.</td>
<td>Symptomatic</td>
<td>332</td>
<td>10</td>
<td>38 (11.4)</td>
<td>?</td>
<td>1</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>McSherry et al.</td>
<td>Symptomatic</td>
<td>556</td>
<td>7</td>
<td>47 (8.5)</td>
<td>19</td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
</tbody>
</table>

Do only in symptomatic cases

Kimura Y, J Hepatobiliary Pancreas Sci 2013
CBD stone

Difficult to diagnose

up to 50% developing complication

Removal

<table>
<thead>
<tr>
<th>Test</th>
<th>Sensitivity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Transabdominal U/S</td>
<td>75% dilated ducts 50% non-dilated</td>
</tr>
<tr>
<td>CT scan</td>
<td>70-75%</td>
</tr>
<tr>
<td>MRCP</td>
<td>88-92% all comers 90-95% dilated ducts</td>
</tr>
<tr>
<td>EUS</td>
<td>90-97%</td>
</tr>
</tbody>
</table>
CBD stone: clinical predictors in who have GS

<table>
<thead>
<tr>
<th>Predictor</th>
<th>Very strong</th>
</tr>
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<tbody>
<tr>
<td>CBD stone on US</td>
<td>Presence of any very strong</td>
</tr>
<tr>
<td>clinical ascending cholangitis</td>
<td>Presence of both strong</td>
</tr>
<tr>
<td>bilirubin &gt; 4 mg/dL</td>
<td>No predictors present</td>
</tr>
<tr>
<td></td>
<td>All other patients</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Predictor</th>
<th>Strong</th>
</tr>
</thead>
<tbody>
<tr>
<td>dilated CBD on US (&gt;6 mm)</td>
<td>Obstruction</td>
</tr>
<tr>
<td>bilirubin 1.8-4 mg/dL</td>
<td>AST or ALT</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Predictor</th>
<th>Moderate</th>
</tr>
</thead>
<tbody>
<tr>
<td>abnormal LFT other than bilirubin</td>
<td>ALP</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Predictor</th>
<th>Low</th>
</tr>
</thead>
<tbody>
<tr>
<td>age &gt; 55 yr</td>
<td></td>
</tr>
<tr>
<td>GS pancreatitis</td>
<td></td>
</tr>
</tbody>
</table>

ASGE guideline 2010
CBD stone

Predictor

Very strong
- CBD stone on US
- clinical ascending cholangitis
- bilirubin > 4 mg/dL

Strong
- dilated CBD on US (>6 mm)
- bilirubin 1.8-4 mg/dL

Moderate
- abnormal LFT other than bilirubin
- age > 55 yr
- GS pancreatitis

Presence of any very strong
- High

Presence of both strong
- High

No predictors present
- Low

All other patients
- Intermediate

ASGE guideline 2010

Low

Intermediate

High

LC

LC with IOC or Preop EUS/MRCP

ERCP

ASGE guideline 2010
A 40 year-old woman presents with **acute epigastric pain** for 4 hr. LFTs: TB 0.6, DB 0.4, **AST 350, ALT 320, ALP 120, A/G 4.4/2.6**. U/S reveals **gall stone**, no biliary duct dilatation.

What’s the most appropriate next management after controlling her abdominal pain?

1. check for HAV, HBV, and HEV infection
2. LC
3. ERCP
4. **Endoscopic ultrasonography**
Acute cholecystitis
# Acute cholecystitis

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>RUQ pain, RUQ tenderness</td>
<td>90%</td>
</tr>
<tr>
<td>Fever</td>
<td>50%</td>
</tr>
<tr>
<td>Prior history of biliary colic</td>
<td>75%</td>
</tr>
<tr>
<td>Murphy’s sign</td>
<td>Sens 65%</td>
</tr>
<tr>
<td></td>
<td>Spec 87%</td>
</tr>
<tr>
<td>Jaundice</td>
<td>20%</td>
</tr>
<tr>
<td>• Sepsis</td>
<td></td>
</tr>
<tr>
<td>• CBD stone</td>
<td></td>
</tr>
<tr>
<td>• Mirizzi’s syndrome</td>
<td></td>
</tr>
</tbody>
</table>
Cholecystitis: Diagnosis

**US**
- Sens 88%, Spec 80%
- US Murphy sign
- Sens 63%, Spec 94%

**Distention (41%), wall thickening (59%), fat strand (52%), pericholecystic fluid (31%)**

**Tc-HIDA scan**
- Sensitivity 80-90%
- False positive 10-20%
# Cholecystitis: Diagnosis

<table>
<thead>
<tr>
<th>Item</th>
<th>Signs</th>
<th>Details</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Local signs of inflammation</td>
<td>Murphy’s sign</td>
</tr>
<tr>
<td></td>
<td></td>
<td>RUQ mass/pain/tenderness</td>
</tr>
<tr>
<td>B</td>
<td>Systemic sign of inflammation</td>
<td>Fever</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Elevated CRP (&gt; 3 mg/dl)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Elevated WBC (&gt;10,000 mm$^3$/dl)</td>
</tr>
<tr>
<td>C</td>
<td>Imaging findings</td>
<td>Characteristic of acute cholecystitis</td>
</tr>
</tbody>
</table>

**Suspected diagnosis:** A + B

**Definitied diagnosis:** A + B + C

Sens 91.2%, Spec 96.9%
Cholecystitis: Treatment

- IV ATB and supportive care
- Specific Rx
  - Cholecystectomy
  - Cholecystostomy
Acute cholecystitis: management

Grade I: not II and III
- ATB and general support care

Grade II: 1/4 of
- Duration > 72 hr,
- palpable tender mass,
- WBC > 18,000/mm³,
- Mark local inflam by image
- ATB and general support care

Grade III: organ failure
- ATB and general support care
  - Urgent/early GB drainage
    - Percutaneous cholecystostomy

In pt with Sx risk
- Observation
  - Advanced laparoscopic technique available
  - Early LC
  - Emergency Sx
  - Successful Rx
  - Delayed/elective LC
  - Failure Rx
    - Urgent/early GB drainage
      - Percutaneous cholecystostomy

Yokoe M, J Hepatobiliary Pancreas Sci 2013
Ascending cholangitis
### Ascending cholangitis

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fever</td>
<td>95%</td>
</tr>
<tr>
<td>RUQ tenderness</td>
<td>90%</td>
</tr>
<tr>
<td>Jaundice</td>
<td>80%</td>
</tr>
<tr>
<td>Confusion</td>
<td>15%</td>
</tr>
<tr>
<td>Hypotension</td>
<td>15%</td>
</tr>
</tbody>
</table>

1+2+3 = Charcot’s triad (20-70% of patients)

1+2+3+4+5 = Reynolds’ pentad
## Ascending cholangitis: Diagnosis

<table>
<thead>
<tr>
<th>Item</th>
<th>Signs</th>
<th>Details</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Systemic inflammation</td>
<td>Fever (&gt; 38 c) and/or shaking chills</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Laboratory data evidence of inflammation</td>
</tr>
<tr>
<td>B</td>
<td>Cholestasis</td>
<td>Jaundice (&gt; 2 mg/dL)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Laboratory data: abnormal LFTs (&gt; 1.5x STD)</td>
</tr>
<tr>
<td>C</td>
<td>Imaging findings</td>
<td>Biliary dilatation</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Evidence of the etiology on imaging (stricture, stone, stent, etc.)</td>
</tr>
</tbody>
</table>

**Suspected diagnosis**: A + (B or C)

**Definitied diagnosis**: A + B + C

Sens 91.8%, Spec 77.7%

Kiriyama S, J Hepatobiliary Pancreas Sci 2013
Cholangitis: Treatment

IV ATB and supportive care +

Specific Rx

ERCP

PTBD

Surgery
Ascending cholangitis: Treatment

**Grade I:** not II and III
- ATB and general support care

**Grade II:** 2/5 of
- Age ≥ 75 yr, T ≥ 39°C, TB ≥ 5mg/dl, Alb ≤ 2.5g/dl
- WBC <4,000 or >12,000
- Early biliary drainage
  - ATB and general support care

**Grade III:** organ failure
- Urgent biliary drainage
  - Organ support, ATB

- Biliary drainage
- Finish course of ATB
- ERCP, PTBD, Sx

Treatment for etiology if still needed (endoscopic Rx, percutaneous Rx, or surgery)

Yokoe M, J Hepatobiliary Pancreas Sci 2013
Acute pancreatitis
Acute Pancreatitis

- ALT ≥ 3x ULN or GS detected by US
  - TG ≥ 1000 mg/dl or elevated Ca
    - Definite drugs or other causes
      - Alcohol ≥ 80 g/day ≥ 5 years
        - CT scan if age ≥ 40
          - EUS
            - Idiopathic AP
          + Identified cause
        + Identified cause
      + Alcoholic pancreatitis
    + Drug-induced or Others
      + Hyper TG or Hypercalcemia
    + GS pancreatitis
**Dx of AP**

**Dx of etiology esp. GS**

**Assess severity**

- **Mild**
  - Supportive Rx

- **Severe**
  - Enteral Nutrition
  - CT at 3-7 d
  - ERCP within 72 hr

  - **No necrosis**
    - Improved
    - CT/US-guided FNA

  - **Necrosis**
    - Gas
    - Conservative Rx ± ATB 5-7 d
      - Sterile
      - Not improved
        - Infected
        - Surgery

  - **ABP + cholangitis**
Outline: Liver disease

### Chronic liver disease
- Viral hepatitis
- Alcoholic hepatitis
- NAFLD
- Autoimmune liver disease
- Metabolic liver disease

### Cirrhosis
- Portal hypertension
- Variceal bleeding
- Ascites and complication
- Hepatorenal syndrome
- Hepatocellular carcinoma
<table>
<thead>
<tr>
<th>Chronic liver disease</th>
<th>Cirrhosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Viral hepatitis</td>
<td>Portal hypertension</td>
</tr>
<tr>
<td>Alcoholic hepatitis</td>
<td>Variceal bleeding</td>
</tr>
<tr>
<td>NAFLD</td>
<td>Ascites and complication</td>
</tr>
<tr>
<td>Autoimmune liver disease</td>
<td>Hepatorenal syndrome</td>
</tr>
<tr>
<td>Metabolic liver disease</td>
<td>Hepatocellular carcinoma</td>
</tr>
</tbody>
</table>
A 27 year-old man consults you because he missed the 3rd dose of hepatitis B vaccine. He received second dose at 12 months ago.

What would you recommend?

1. Restart full course of hepatitis B vaccine
2. Continue 3rd dose as soon as possible
3. Check anti-HBs, and restart full course of hepatitis B vaccine if result is negative
4. Check anti-HBs, and continue 3rd dose as soon as possible if result is negative
Viral hepatitis B

<table>
<thead>
<tr>
<th>Immune tolerance</th>
<th>Immune clearance</th>
<th>Immune control</th>
<th>Immune escape</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>HBeAg+ve</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HBV-DNA</td>
<td>10^9–10^10 cp/mL</td>
<td>&lt;10^5 cp/mL</td>
<td>&gt;10^5 cp/mL</td>
</tr>
<tr>
<td></td>
<td>10^7–10^8 cp/mL</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ALT</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

monitor: HBeAg +ve chronic hepatitis

monitor: Inactive-carrier state

monitor: HBeAg –ve chronic hepatitis

treat

Inactive-carrier state
Viral hepatitis B

HbeAg +ve

ALT < 1 x ULN
Q 3-6 mo ALT
Q 6-12 mo HBeAg

ALT 1-2 x ULN
Q 3 mo ALT
Q 6 mo HBeAg
Bx if persistent to age > 40, Rx as needed

ALT > 2 x ULN
Q 1-3 mo ALT, HBeAg
Treat if persist 3-6 mo
Bx optional
Immediate Rx if Jx or decompensated

Imm tolerance

Imm clearance

Lok AS, AASLD guidelines; Chronic hepatitis B 2009
Viral hepatitis B

**Inactive**
- ALT < 1x ULN
- VL < 2,000 IU/mL
- Q 3 mo ALT x 3, Then Q 6-12 mo if ALT still < 1x ULN

**HbeAg -ve**
- ALT 1-2x ULN
- VL 2,000-20,000
- Q 3 mo ALT & HBV VL
- Consider biopsy if persistent
- Rx as needed

**Reactivation**
- ALT > 2x ULN
- VL > 20,000 IU/mL
- Treat if persistent,
- Liver biopsy optional

Lok AS, AASLD guidelines; Chronic hepatitis B 2009
Viral hepatitis B: vaccine

Different manufacturer between doses?  OK

Interrupt:

Interrupt at 1\textsuperscript{st} dose:
- 2\textsuperscript{nd} dose as soon as possible,
- 3\textsuperscript{rd} dose at 8 wk interval from 2\textsuperscript{nd}

Interrupt at 2\textsuperscript{nd} dose:
- 3\textsuperscript{rd} dose as soon as possible

http://www.cdc.gov
A 27 year-old man consults you because he forgets to receive the 3$^{rd}$ dose of hepatitis B vaccine. He received second dose at 12 months ago.

What would you recommend?

1. Restart full course of hepatitis B vaccine
2. Continue 3$^{rd}$ dose as soon as possible
3. Check anti-HBs, and restart full course of hepatitis B vaccine if result is negative
4. Check anti-HBs, and continue 3$^{rd}$ dose as soon as possible if result is negative
Viral hepatitis C

Age > 18 years with HCV RNA +ve

Genotype 1, 4, 6
- Significant fibrosis
  - F2 on Metavir
  - Fibroscan > 7 kPa

Consider Rx with PegIFN + RBV 6 mo

Genotype 2, 3
- Consider Rx with PegIFN + RBV 12 mo

Advise and F/U

Thailand Practice Guideline for management of chronic hepatitis B and C 2012
Viral hepatitis C

**Contraindication**

- Decompensated cirrhosis
- Allergy to IFN, RBV
- Severe uncontrolled depression
- Pregnancy or intend to pregnancy
- Organ transplantation except liver transplantation
- Poor underlying medical illness

Thailand Practice Guideline for management of chronic hepatitis B and C 2012
**Alcoholic liver disease: Acute alcoholic hepatitis**

- **Establish disease severity**
  - **High risk:**
    - MDF $> 32$, presence of HE, or MELD $> 18$
  - **Low risk:**
    - MDF $< 32$ and $1^{st}$ week decrease in bilirubin, or MELD $< 18$ and $1^{st}$ wk decrease in MELD by 2 pt

- **Consider liver Bx if diagnosis is uncertain**
  - Prednisolone
  - Pentoxifyline (early renal failure)

- **Nutritional assessment/intervention**
  - Not include:
    - Pancreatitis
    - GI bleeding
    - Active infection
  - Supportive care and close follow up
Non alcoholic fatty liver disease (NAFLD)

Metabolic syndrome

- Fatty liver
  - rare

- NASH
  - 20-25%

Cirrhosis

- Subacute liver failure
  - 2%

- Liver-related death
  - 40%

- Hepatocellular carcinoma
  - ?
NAFLD: predictor of fibrosis

**NAFLD fibrosis Score**
- Age
- BMI
- Hyperglycemia
- Plt count
- Albumin
- AST/ALT ratio

**BARD score**
- BMI > 28 kg/m²
- AST/ALT Ratio > 0.8
- DM
- Score ≥ 2 significant fibrosis 17x, NPV 96%

**Consider liver biopsy**
# NAFLD: Treatment

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Recommendation</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight reduction</td>
<td>Yes</td>
<td>• 3-5%: improve steatosis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• 10%: improve necroinflammation</td>
</tr>
<tr>
<td>Metformin</td>
<td>No</td>
<td>6-12 mo of MF did not improve aminotransferases or liver histology</td>
</tr>
<tr>
<td>Pioglitazone</td>
<td>Yes</td>
<td>Improve histology in biopsy-proven NASH, esp in non-diabetic</td>
</tr>
<tr>
<td>Vitamin E</td>
<td>Yes</td>
<td>Dose 800 IU/d improves histology in non-diabetic adults with biopsy-proven NASH</td>
</tr>
<tr>
<td>UDCA</td>
<td>No</td>
<td>No histological effect</td>
</tr>
<tr>
<td>Omega-3 fatty acid</td>
<td>N/A</td>
<td>1st line agent to Rx hyperTg in NAFLD</td>
</tr>
<tr>
<td>Statin</td>
<td></td>
<td>Can be used to Rx dyslipidemia in NAFLD</td>
</tr>
</tbody>
</table>
Autoimmune liver disease

Liver parenchyma: AIH

Small duct: PBC

Large duct: PSC
## AIH: Clinical clues

<table>
<thead>
<tr>
<th>Type 1</th>
<th>Type 2</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age</strong></td>
<td>Infants to elderly</td>
</tr>
<tr>
<td>Female</td>
<td>78%</td>
</tr>
<tr>
<td>Concurrent immune diseases</td>
<td>38%</td>
</tr>
<tr>
<td>Typical concurrent autoimmune diseases</td>
<td>Autoimmune thyroiditis</td>
</tr>
<tr>
<td></td>
<td>Graves’ disease</td>
</tr>
<tr>
<td></td>
<td>Ulcerative colitis</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td>Organ-specific antibodies</td>
<td>4%</td>
</tr>
<tr>
<td>Serum gamma globulin elevation</td>
<td>+++</td>
</tr>
</tbody>
</table>

### Clinical presentation

- **Acute hepatitis on top chronic hepatitis**: 40-50%
- **Chronic hepatitis**: 40-50%
- **Acute hepatitis**: 10%
## AIH: Investigation

<table>
<thead>
<tr>
<th>Antibody</th>
<th>Type</th>
<th>Prevalence</th>
</tr>
</thead>
<tbody>
<tr>
<td>ANA</td>
<td>1</td>
<td>60-80%</td>
</tr>
<tr>
<td>SMA</td>
<td>1</td>
<td>60-80%</td>
</tr>
<tr>
<td>LKM-1</td>
<td>2</td>
<td>4%</td>
</tr>
</tbody>
</table>

- **Plasma cell infiltration**
- **Interface hepatitis**
- **Hepatocyte rosette formation**
## AIH treatment

<table>
<thead>
<tr>
<th>FINDINGS</th>
<th>Indications</th>
<th>RELATIVE</th>
<th>NONE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clinical</td>
<td>Incapacitating symptoms</td>
<td>Mild or no symptoms</td>
<td>Asymptomatic with minimal laboratory changesPrevious intolerance of prednisone and/or azathioprine</td>
</tr>
<tr>
<td>Laboratoy</td>
<td>AST ≥10-fold ULN</td>
<td>AST 3- to 9.9-fold ULN</td>
<td>AST &lt;3-fold ULN</td>
</tr>
<tr>
<td></td>
<td>AST ≥5-fold ULN and gamma globulin ≥ 2-fold ULN</td>
<td>AST ≥ 5-fold ULN and gamma globulin &lt;2-fold ULN</td>
<td>Severe cytopenia</td>
</tr>
<tr>
<td>Histologic</td>
<td>Bridging necrosis</td>
<td>Interface hepatitis</td>
<td>Inactive cirrhosis</td>
</tr>
<tr>
<td></td>
<td>Multilobular necrosis</td>
<td></td>
<td>Portal hepatitis</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Decompensated cirrhosis with variceal bleeding</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Combination</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prednisone Only (mg/d)</td>
</tr>
<tr>
<td>-------------</td>
</tr>
<tr>
<td>60</td>
</tr>
</tbody>
</table>
Primary biliary cirrhosis (PBC)

Small duct (interlobular bile ducts) destruction by autoimmune inflammatory process
# PBC : Clinical

<table>
<thead>
<tr>
<th>Finding</th>
<th>Frequency (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fatigue</td>
<td>21-85</td>
</tr>
<tr>
<td>Pruritus</td>
<td>19-55</td>
</tr>
<tr>
<td>Jaundice</td>
<td>3-10</td>
</tr>
<tr>
<td>Right upper quadrant pain</td>
<td>8</td>
</tr>
<tr>
<td>Hyperpigmentation</td>
<td>25</td>
</tr>
<tr>
<td>Hepatomegaly</td>
<td>25</td>
</tr>
<tr>
<td>Splenomegaly</td>
<td>15</td>
</tr>
<tr>
<td>Xanthelasma</td>
<td>10</td>
</tr>
<tr>
<td>None</td>
<td>25-61</td>
</tr>
</tbody>
</table>
PBC: Diagnosis

- AMA +ve
- ALP > 1.5x
- AST < 5x

PPV > 98%

Ductopenia
Granuloma
PBC: Treatment

**UDCA**
- 13-15 mg/kg/d

**Cirrhosis complication**

**Cholestatic complication**
- Pruritus: Cholestyramine, rifacimin, opioid antagonist
- Steatorrhea: MCT
- Bone disease: Vit D + Ca
- Coagulopathy: Vit K
Primary sclerosing cholangitis

Large duct destruction by autoimmune inflammatory process
PSC: Clinical features

**Intermittent biliary obstruction**

**Secondary biliary cirrhosis**

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>Rate (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fatigue</td>
<td>65-75</td>
</tr>
<tr>
<td>Abdominal pain</td>
<td>24-72</td>
</tr>
<tr>
<td>Pruritus</td>
<td>15-69</td>
</tr>
<tr>
<td>Fever/night sweats</td>
<td>13-45</td>
</tr>
<tr>
<td>Asymptomatic</td>
<td>15-44</td>
</tr>
<tr>
<td>Weight loss</td>
<td>10-34</td>
</tr>
</tbody>
</table>

**Signs**

- Jaundice: 30-73
- Hepatomegaly: 34-62
- Splenomegaly: 32-34
- Hyperpigmentation: 14-25
- Ascites: 4-7
PSC: Treatment

- UDCA
- Immunosuppressive and other agents

Less Benefit

- ERCP and endoscopic therapy
- Liver transplantation
Outline: Liver disease

Chronic liver disease
- Viral hepatitis
- Alcoholic hepatitis
- NAFLD
- Autoimmune liver disease
- Metabolic liver disease

Cirrhosis
- Portal hypertension
- Variceal bleeding
- Ascites and complication
- Hepatorenal syndrome
- Hepatocellular carcinoma
Which condition can cause varix only at fundus of stomach?

1. Primary biliary cirrhosis
2. Essential thrombocytosis with portal vein thrombosis
3. Alcoholic chronic pancreatitis
4. Budd-Chiari syndrome
5. Schistosomiasis
Portal hypertension

Left sided portal HT:
- Isolated fundal varices

Caput medusa:
- Cirrhosis

Left sided portal HT:
- Isolated fundal varices
Portal hypertension: Etiologies

**Prehepatic**
- Portal V thrombosis
- Splenic V thrombosis

**Posthepatic**
- Budd-Chiari syndrome
- Constrictive pericarditis
- Rt-sided HF

**Intrahepatic**
- Presinusoidal: Schistosomiasis
- Sinusoidal: cirrhosis
- Postsinusoidal: VOD

**Variceal bleeding**

**Ascites (high prot)**

**hepatomegaly**

**Ascites (low prot)**
Which condition can cause varix only at fundus of stomach?

1. Primary biliary cirrhosis

2. Essential thrombocytosis with portal vein thrombosis

3. **Alcoholic chronic pancreatitis**

4. Budd-Chiari syndrome

5. Schistosomiasis
Variceal bleeding
Variceal hemorrhage

\[ T = tp \times \frac{r}{w} \]

\[ V = I \times R \]

No varices
- 8% per year
- No hemorrhage

Small varices
- 8% per year
- No hemorrhage

Med/Large varices
- 5-15% per year
- No hemorrhage

Variceal hemorrhage
- 60%

Recurrent hemorrhage

Variceal growth \( \rightarrow \) rupture

OLT
- cirrhosis

TIPS, Shunt
- ↑Resistance to portal flow

Variceal bleeding

\[ V = I \times R \]

↑ Portal pressure

↑ Portal blood flow

Somatostatin, telipressin, Non-select BB

Splanchnic vasodilatation

EVL

Splanchnic vasodilatation
No varices

- 8% per year

Small varices
- No hemorrhage

- 8% per year

Med/Large varices
- No hemorrhage

- 5-15% per year

Variceal hemorrhage

- 60%

Recurrent hemorrhage

- 60%

EGD in every cirrhotic pt

- Prevent variceal formation: no benefit
- BB

- Prevent variceal progression:
  - CTP-B, C or presence of red wale sign
- BB

- Repeat EGD:
  - CTP-A: 3 year
  - CTP-B, C: 1 year

- Prevent variceal rupture
  - BB or EVL

- Stop variceal bleeding
  - Somatostatin/telipressin + EVL,
  - if fail → TIPS or OLT

- Prevent rebleeding
  - BB & EVL, if fail → TIPS or OLT

- 8% per year
Ascites
Ascites: What’s SAAG

Exudate VS Transudate

High protein vs Low protein

Right-sided CHF: High protein
SBP: Low protein
Ascites: What’s SAAG

\[ S_H - S_O = PC_H - PC_O \]
\[ S_H = S_O + PC_H - PC_O \]

Portal pressure = Serum Alb – Ascites Alb
Ascites

1. Cirrhotic ascites
2. Cardiac ascites
3. Peritoneal malignancy

Serum – ascites albumin gradient (g/dL)

- Cirrhotic ascites: 1.0
- Cardiac ascites: 0.0
- Peritoneal malignancy: 0.0

Ascitic fluid total protein (g/dL)

- Cirrhotic ascites: 2.0
- Cardiac ascites: 0.0
- Peritoneal malignancy: 0.0


97% accuracy
Ascites

High gradient > 1.1 g/dl
- Sinusoid
  - Cirrhosis
  - Alcoholic hepatitis
  - Mixed ascites
- Post sinusoid
  - Veno-occlusive disease Budd-Chiari syndrome
  - Cardiac ascites
- Myxedema

Low gradient ≤ 1.1 gm/dl
- Peritoneal disease
  - Peritoneal carcinomatosis
  - TB peritonitis
  - Pancreatic ascites
  - Bowel obstruction / infarction
  - Biliary ascites
  - Postop lymphatic leak
  - Serositis in CNT diseases
- Nephrotic syndrome

## Ascites

<table>
<thead>
<tr>
<th>High gradient &gt; 1.1 g/dl</th>
<th>Low gradient ≤ 1.1 gm/dl</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sinusoid</td>
<td>Peritoneal disease</td>
</tr>
<tr>
<td>Cirrhosis</td>
<td>Peritoneal carcinomatosis</td>
</tr>
<tr>
<td>Alcoholic hepatitis</td>
<td>TB peritonitis</td>
</tr>
<tr>
<td>Mixed ascites</td>
<td>Pancreatic ascites</td>
</tr>
<tr>
<td>Post sinusoid</td>
<td>Bowel obstruction /infarction</td>
</tr>
<tr>
<td>Veno-occlusive diseaseBudd-Chiari syndrome</td>
<td>Biliary ascites</td>
</tr>
<tr>
<td>Cardiac ascites</td>
<td>Postop lymphatic leak</td>
</tr>
<tr>
<td>Myxedema</td>
<td>Serositis in CNT diseases</td>
</tr>
<tr>
<td></td>
<td>Nephrotic syndrome</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Grade</th>
<th>Definition</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Only detectable by US</td>
<td>No treatment</td>
</tr>
<tr>
<td>2</td>
<td>Moderate symmetrical distension of abdomen</td>
<td>Restriction of salts and diuretic</td>
</tr>
<tr>
<td>3</td>
<td>Large or gross ascites with marked abdominal distention</td>
<td>LVP</td>
</tr>
</tbody>
</table>
Grade 2 ascites

Low-sodium diet (70-90 mEq/day)
Diuretics

No peripheral edema
Spironolactone
or Amiloride

Goal:
Weight loss 0.5 kg/day

Peripheral edema
Spironolactone + furosemide

Goal:
Weight loss 1 kg/day

Maintenance therapy

Grade 3 ascites

Large-volume Paracentesis/TIPS

< 5 L

Synthetic plasma expanders
(8 g/L of ascites tapped)

> 5 L

Albumin
(8 g/L of ascites tapped)

Low sodium diet (70-90 mEq/day)
Diuretics

<table>
<thead>
<tr>
<th>Type of infection</th>
<th>PMN (/mm3)</th>
<th>Culture result</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spontaneous bacterial peritonitis</td>
<td>≥ 250</td>
<td>Positive</td>
<td>ATB + alb</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(usually 1 Organism)</td>
<td></td>
</tr>
<tr>
<td>Culture-negative neutrocytic ascites</td>
<td>≥ 250</td>
<td>Negative</td>
<td>ATB + alb</td>
</tr>
<tr>
<td>Monomicrobial nonneutrocytic bacterascites</td>
<td>&lt; 250</td>
<td>Positive</td>
<td>Symp : as SBP</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(1 Organism)</td>
<td>Asymp : repeat</td>
</tr>
<tr>
<td>Secondary bacterial peritonitis</td>
<td>≥ 250</td>
<td>Positive</td>
<td>Surgery</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(polymicrobial)</td>
<td></td>
</tr>
</tbody>
</table>
SBP: Albumin infusion

- 1.5 g/kg within 6 hr and 1 g/kg on day 3
- ↓ type I HRS (30%->10%) and MR (29%->10%)
- Indicated in
  1. Cr >1mg/dL
  2. BUN >30 mg/dL
  3. TB > 4mg/dL
Ascites infection

TREATMENT INDICATED

PMN>250?

NO

Culture Positive?

YES

TREATMENT NOT INDICATED

NO

Repeat Paracentesis

PMN>250?

YES

Culture Positive?

NO

YES
Primary VS Secondary peritonitis

<table>
<thead>
<tr>
<th>Ascitic profiles</th>
<th>Sensitivity*</th>
<th>Specificity*</th>
</tr>
</thead>
<tbody>
<tr>
<td>2/3 of</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Total protein &gt; 1g/dL</td>
<td>100</td>
<td>45</td>
</tr>
<tr>
<td>• LDH &gt; ULN of serum LDH</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Glucose &lt; 50 mg/dL</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• CEA &gt; 5 ng/mL or</td>
<td>92</td>
<td>88</td>
</tr>
<tr>
<td>• ALP &gt; 240 units/L</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Only for free perforation

Akriviadis EA, Runyon BA. Gastroenterology 1990
Wu SS et al. J Hepatol 2001
Hepatorenal syndrome

- Functional renal failure in cirrhosis
- Decompenated cirrhosis
  - NO, CO, endogenous cannabinoids
  - Splanchnic vasodilatation
  - ↓ effective circulatory volume
  - RAAS \( \rightarrow \) Renal vasoconstriction
  - Renal failure

- Type 1
- Type 2

Infection: SBP, UTI
Hepatorenal syndrome: Diagnosis

1. Cirrhosis with ascites
2. Serum creatinine > 1.5 mg/dL
3. No improvement after at least 2 days with diuretic withdrawal and volume expansion with albumin (1g/kg/d, max 100 g/d)
4. Absence of other causes
   1. shock
   2. recent treatment with nephrotoxic drugs
   3. parenchymal renal disease (proteinuria > 0.5 g/d, rbc > 50/HF, abnormal U/S)

Type 1
Rapidly progressive (Cr >2.5 mg/dL or Ccr <20 ml/min in 2 wk)

Type 2
Slow (months)
Refractory ascites
## Hepatorenal syndrome: Treatment

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Type I</th>
<th>Type II</th>
</tr>
</thead>
<tbody>
<tr>
<td>Correct precipitating factor</td>
<td>+++</td>
<td></td>
</tr>
<tr>
<td>Vasoactive drugs plus albumin</td>
<td>+++</td>
<td>+</td>
</tr>
<tr>
<td>Renal replacement therapy</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>TIPS</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Liver dialysis</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>Liver transplantation</td>
<td>+++</td>
<td>+++</td>
</tr>
</tbody>
</table>

Vasoactive agents: Terlipressin, Norepinephrine, Midodrine
Mr KTR, a 56 year-old man with HBV cirrhosis is detected a 4.5 cm arterial enhancing mass with portovenous wash out in left lobe liver by 3-phase liver CT scan. PV is patent.

Lab: Hb 12 g/dL, Platelet 50,000, TB 2.2 g/dL, DB 1.7 g/DL, AST 50 IU/L, ALT 40 IU/L, ALP 110 IU/L, Alb 3.0 g/dL, Glob 4.0 g/dL.

What is the most appropriate management next?

1. Request serum Alpha fetoprotein to confirm diagnosis of HCC
2. Mass biopsy to confirm diagnosis of HCC
3. Hepatectomy with mass resection
4. Liver transplantation
5. Transarterial chemoembolization
Hepatocellular carcinoma

Screening

- US + AFP q 6-12 months
- Cirrhosis
- Chronic HBV infection
  - Asian male > 40 yr
  - Asian female > 50 yr
  - Family history of HCC

Bruix J, AASLD practice guideline 2010
Diagnosis

Cirrhosis/CH-B with liver nodule

< 1cm

Repeat US q 3 mo

Growing

Ix according to size

Stable for 2 yr

Routine surveillance

> 1cm

4-phase MDCT/dynamic CE MRI

Typical lesion

Bruix J, AASLD practice guideline 2010
Typical HCC on 3-phase CT

A. hypervascular

AND

V. or delayed phase wash out

A phase 30 sec

V phase 80 sec
Diagnosis

Cirrhosis/CH-B with liver nodule

< 1cm

Repeat US q 3 mo

Growing

Ix according to size

Stable for 2 yr

Routine surveillance

> 1cm

4-phase MDCT/dynamic CE MRI

Typical lesion

HCC

Y

Other CE study

N

Bx

Bruix J, AASLD practice guideline 2010
Mr KTR, a 56 year-old man with HBV cirrhosis is detected a 4.5 cm arterial enhancing mass with portovenous wash out in left lobe liver by 3-phase liver CT scan. PV is patent.

Lab : Hb 12 g/dL, Platelet 50,000, TB 2.2 g/dL, DB 1.7 g/DL, AST 50 IU/L, ALT 40 IU/L, ALP 110 IU/L, Alb 3.0 g/dL, Glob 4.0 g/dL.

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1. Request serum Alpha fetoprotein to confirm diagnosis of HCC
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3. Hepatectomy with mass resection
4. Liver transplantation
5. Transarterial chemoembolization
ทำดี...ได้ดี
Thank you for your attentions and hope to see you again ....
วันเสาร์ที่ 8 มีนาคม 2557

<table>
<thead>
<tr>
<th>Time</th>
<th>Session</th>
</tr>
</thead>
<tbody>
<tr>
<td>08:30-08:40</td>
<td>Opening Remark นพ. สาวนวล ภายในสีลึก นร.</td>
</tr>
<tr>
<td>08:40-09:00</td>
<td>Dysphagia นพ. สาวนวล อิศราพิบูลย์สัมพันธ์</td>
</tr>
<tr>
<td>09:00-09:30</td>
<td>Dyspepsia นพ. สาหร่าย มณีรัตน์สุรเศรษฐี</td>
</tr>
<tr>
<td>09:30-09:50</td>
<td>Nausea / Vomiting นพ. สาหร่าย มณีรัตน์สุรเศรษฐี</td>
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<tr>
<td>09:50-10:20</td>
<td>Diarrhea นพ. สาวนวล ภายในสีลึก</td>
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<tr>
<td>10:20-10:40</td>
<td>Break</td>
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<tr>
<td>10:40-11:00</td>
<td>Constipation นพ. สาหร่าย พระภิรมย์สุรเศรษฐี</td>
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<tr>
<td>11:00-11:20</td>
<td>Jaundice นพ. สาหร่าย โยธินรัตน์</td>
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<tr>
<td>11:20-11:40</td>
<td>Ascites นพ. สาหร่าย โยธินรัตน์</td>
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<tr>
<td>11:40-12:50</td>
<td>Lunch</td>
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</tbody>
</table>

วันอาทิตย์ที่ 9 มีนาคม 2557

<table>
<thead>
<tr>
<th>Time</th>
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<tr>
<td>08:30-09:00</td>
<td>Cholangiocarcinoma นพ. สาหร่าย ปริญญา</td>
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<tr>
<td>09:00-09:30</td>
<td>Acute Viral Hepatitis and นพ. สาหร่าย ปริญญา</td>
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<tr>
<td></td>
<td>Fulminant Hepatitis นพ. สาหร่าย ปริญญา</td>
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<tr>
<td>09:30-10:00</td>
<td>Chronic Viral Hepatitis นพ. สาหร่าย ปริญญา</td>
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<td>10:00-10:20</td>
<td>Alcoholic Liver Disease นพ. สาหร่าย ปริญญา</td>
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<td>10:20-10:40</td>
<td>Break</td>
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<tr>
<td>10:40-11:00</td>
<td>NAFLD นพ. สาหร่าย ปริญญา</td>
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<tr>
<td>11:00-11:20</td>
<td>Wilson’s Disease นพ. สาหร่าย โยธินรัตน์</td>
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<tr>
<td>11:20-12:00</td>
<td>AIH, PBC and PSC นพ. สาหร่าย ปริญญา</td>
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<td>12:00-13:00</td>
<td>Lunch</td>
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<td>13:00-13:40</td>
<td>Cirrhosis and Complications นพ. สาหร่าย โยธินรัตน์</td>
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<td>13:40-14:10</td>
<td>Hepatocellular Carcinoma นพ. สาหร่าย โยธินรัตน์</td>
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<td>14:10-14:40</td>
<td>Vascular Disorders of the Liver นพ. สาหร่าย โยธินรัตน์</td>
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<td>14:40-15:00</td>
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<tr>
<td>15:00-15:30</td>
<td>Inflammatory Bowel Disease นพ. สาหร่าย โยธินรัตน์</td>
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<td>15:30-15:50</td>
<td>Endoscopic Findings You Need to Know นพ. สาหร่าย ปริญญา</td>
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<td>15:50-16:30</td>
<td>Common Inpatient GI Consultation นพ. สาหร่าย โยธินรัตน์</td>
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<td>16:30</td>
<td>Closing Remark</td>
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