GENERAL SURGERY

Dr. S. Gallinger
Melanie Altas, Chad Ball and Jamie Newman, chapter editors
Gilbert Tang, associate editor

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ACUTE ABDOMEN

Definition
- an abdomen with immense pain and/or peritonitis.

Note: most cases of acute abdomen do not need an immediate operation

APPROACH TO THE CRITICALLY ILL SURGICAL PATIENT

ABC, I'M FINE

ABC (see Emergency Medicine Chapter)
I - IV: two large bore IV’s with normal saline, wide open
M - Monitors: O₂ sat, EKG, BP
F - Foley catheter to measure urine output
I - Investigations: see above
N - +/- NG tube
E - Ex rays (3 views, CXR)

Table 1. Causes of an Acute Abdomen

<table>
<thead>
<tr>
<th>Vascular</th>
<th>Gastrointestinal</th>
</tr>
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<tbody>
<tr>
<td>ruptured abdominal aortic aneurysm (RAAA)</td>
<td>pancreatitis, gastritis</td>
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<tr>
<td>mesenteric embolus/thrombus</td>
<td>penetrating/perforated peptic ulcer</td>
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<tr>
<td></td>
<td>acute cholecystitis</td>
</tr>
<tr>
<td>Urological</td>
<td>biliary colic</td>
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<tr>
<td>pyelonephritis</td>
<td>appendicitis</td>
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<tr>
<td>renal colic</td>
<td>diverticulitis</td>
</tr>
<tr>
<td>cystitis</td>
<td>small/large bowel obstruction/perforation</td>
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<tr>
<td></td>
<td>intestinal ischemia</td>
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<tr>
<td>Gynecological</td>
<td>inflammatory bowel disease (IBD)</td>
</tr>
<tr>
<td>ruptured ectopic pregnancy</td>
<td>Extraperitoneal</td>
</tr>
<tr>
<td>acute pelvic inflammatory disease (PID)</td>
<td>myocardial infarction (MI)</td>
</tr>
<tr>
<td>ruptured ovarian cyst</td>
<td>pleuritis</td>
</tr>
<tr>
<td>torsion of ovarian cyst</td>
<td>lower lobe pneumonia</td>
</tr>
<tr>
<td>Mittelschmerz</td>
<td>rectal sheath or abdominal wall hematomata</td>
</tr>
<tr>
<td>Other</td>
<td></td>
</tr>
<tr>
<td>diabetes mellitus (DM)</td>
<td></td>
</tr>
<tr>
<td>lead poisoning</td>
<td></td>
</tr>
<tr>
<td>porphyria</td>
<td></td>
</tr>
<tr>
<td>Herpes Zoster</td>
<td></td>
</tr>
<tr>
<td>tertiary syphilis</td>
<td></td>
</tr>
</tbody>
</table>

EVALUATION

History

Pain
- location of pain (see Table 2)
- include potential causes above and below the actual site of pain
- onset, quality, radiation, severity, timeline of pain, relieving/aggravating factors
- history of similar pain
- referred pain
  - biliary colic: right shoulder or scapula
  - renal colic: to groin
  - appendicitis: periumbilical to right lower quadrant (RLQ)
- pancreatitis: to back
- ruptured aortic aneurysm: to back or flank
- perforated ulcer: to RLQ (right paracolic gutter)
- hip pain: to groin

Associated Symptoms
- systemic: fevers, chills, weight loss, jaundice, pruritis
- gastrointestinal: anorexia, nausea, vomiting, diarrhea, constipation, tenesmus, obstipation, melena, hematochezia, hematemesis, pale stools, steatorrhea
- urinary: dysuria, hematuria, urinary frequency, dark urine
- gynecological: 1st day last menstrual period (LMP), vaginal discharge, previous sexually transmitted disease (STD), intrauterine device (IUD) use, sexual history, libido
- extra-intestinal features: eyes, skin, joints
- other: food intolerances, time since last meal, travel history, contact history, usual bowel habits, drug history (NSAIDS, EtOH, laxatives, steroids, ulcer medications), family history (IBD, stones, cancers)
Physical Examination
1) general observation: patient position (i.e. lying still vs. writhing), facial expression
2) vitals: postural changes, fever
3) status of hydration: vitals, mucous membranes, skin, urinary output, jugular venous pressure (JVP), mental status
4) cardiovascular/respiratory examination
5) abdominal examination
   observation: guarding, distention, bulging flanks, scars, visible peristalsis, liver stigmata
   auscultation: absent, decreased, normal, increased or tinkling bowel sounds, bruits
   percussion: hypertympanic sounds in bowel obstruction, hepatosplenomegaly, ascites, percussion tenderness indicative of peritonitis
   palpation: tenderness, abdominal masses, hepatosplenomegaly, ascites
6) costovertebral angle (CVA) tenderness, cough tenderness (peritonitis)
7) specific signs (see below)
8) hernias, male genitalia
9) rectal/pelvic exam

Specific "Signs" on Physical Examination
   Blumberg's sign (rebound tenderness): constant, held pressure with sudden release causes severe tenderness (peritoneal irritation)
   Courvoisier's sign: palpable, non-tender gall bladder with jaundice (pancreatic or biliary malignancy)
   Cullen's sign: blue discoloration around umbilicus (peritoneal hemorrhage)
   Grey Turner's sign: flank discoloration (retroperitoneal hemorrhage)
   Iliopsoas sign: flexion of hip against resistance or passive hyperextension of hip causes pain (retrocecal appendix)
   Murphy's sign: inspiratory arrest on deep palpation of RUQ (cholecystitis)
   McBurney's point tenderness: 1/3 from anterior superior iliac spine (ASIS) to umbilicus; indicates local peritoneal irritation (appendicitis)
   Obturator sign: flexion then external or internal rotation about the right hip causes pain (pelvic appendicitis)
   Percussion tenderness: often good substitute for rebound tenderness
   Rovsing's sign: palpation pressure to left abdomen causes McBurney's point tenderness (appendicitis)
   Shake tenderness: peritoneal irritation (bump side of bed in suspected malingerers)
   Boas’s sign: right subscapular pain due to cholelithiasis
   Fox’s sign: ecchymosis of inguinal ligament seen with retroperitoneal bleeding
   Dance’s sign: empty right lower quadrant in children with ileocecal intussusception

Investigations laboratory
   CBC and differential (possible group and screen/type and cross if necessary)
   electrolytes, BUN, creatinine
   amylase, lipase levels
   liver enzymes, liver function tests
   urinalysis (plus C&S, R&M if necessary)
   stool for occult blood
   others - ECG, ß-hCG, ABG, septic workup, lactate (ischemic bowel)
ACUTE ABDOMEN... CONT.

radiology
- 3 views abdomen
- CXR
- others - U/S, CT, endoscopy, intravenous pyelogram (IVP), peritoneal lavage, laparoscopy

indications for urgent operation (i.e. surgical abdomen)
- physical findings
  - diffuse peritonitis (localized peritonitis is not always an indication)
  - severe or increasing localized tenderness
  - progressive distension
  - tender mass with fever or hypotension (abscess)
  - septicemia and abdominal findings
  - bleeding and abdominal findings
  - suspected bowel ischemia (acidosis, fever, tachycardia)
  - deterioration on conservative treatment
- radiologic
  - free air
  - massive bowel distention (colon > 12 cm)
  - space occupying lesion with fever
- endoscopic
  - perforation
  - uncontrollable bleeding
- paracentesis
  - blood, pus, bile, feces, urine

Figure 1. Abdominal Incisions
Illustration by Jackie Robers

Layers of the Abdominal Wall
skin (epidermis, dermis and subcutaneous fat)
superficial fascia
- Camper’s fascia —> dartos muscle
- Scarpa’s fascia —> Colles’ fascia
muscle (see Figure 1)
- external oblique —> inguinal ligament, external spermatic fascia, fascia lata
- internal oblique —> cremasteric muscle
- transversalis abdominus —> posterior inguinal wall
transversalis fascia —> internal spermatic fascia
supraperitoneal fat
peritoneum —> tunica vaginalis
at midline
- rectus abdominus muscle; in rectus sheath, divided by linea alba
- above semicircular line of Douglas (midway between symphysis pubis and umbilicus):
- anterior rectus sheath = external oblique aponeurosis and anterior leaf of internal oblique aponeurosis
- posterior rectus sheath = posterior leaf of internal oblique aponeurosis and transversus
- below semicircular line of Douglas:
- anterior rectus sheath = aponeurosis of external, internal oblique, transversus arteries:
  - superior epigastric (branch of internal thoracic), inferior epigastric (branch of external iliac);
  - both arteries anastomose and lie behind the rectus muscle
HIATUS HERNIA (HH)

Figure 2. Types of Hiatus Hernia

Illustrations by Bryce Hough

Sliding Hiatus Hernia (Type I)
- Herniation of both the stomach and the gastroesophageal (GE) junction into thorax
- Majority are asymptomatic
- GERD (heartburn 1-3 hrs post-prandial, chest pain, regurgitation)
- Relief with sitting, standing, water, antacids
- Complications:
  - Reflux, esophagitis, chronic occult GI blood loss with anemia, ulceration, dysphagia, esophageal stricture, Barrett's esophagus, adenocarcinoma, aspiration pneumonia, bleeding

Investigation:
- Gastroscopy with biopsy: document type and extent of tissue damage, rule out esophagitis, Barrett's esophagus and cancer
- 24 hour esophageal pH monitoring: often used if atypical presentation, gives information about frequency and duration of acid reflux, correlation of symptoms with signs
- Esophageal manometry: detects decreased lower esophageal sphincter (LES) pressure; may also diagnose motility disorder
- Upper GI series or barium swallow
- CXR globular shadow with air-fluid level over cardiac silhouette, visible shadow posterior mediastinum on lateral view

Treatment:
- Conservative:
  - Stop smoking
  - Weight loss
  - Elevate head of bed
  - No nocturnal meals (<3 hrs prior to sleeping)
  - Smaller and more frequent meals
  - Avoid alcohol, coffee, fat
- Medical:
  - Antacids
  - H2 antagonists (e.g. cimetidine, ranitidine)
  - Proton pump inhibitor (e.g. Losec, Pentaloc, Prevacid) x 8-12 weeks for esophagitis
  - Adjuvant prokinetic agents may play a role (metoclopramide, motilium)
- Surgical (<15%)
  - Nissen fundoplication (laparoscopic or open) where fundus of stomach is wrapped around the LES and sutured in place
  - 90% success rate
  - Indications for surgery:
    - Complications of sliding hernia or GERD (especially stricture, severe ulceration, fibrosis, bleeding, Barrett's)
    - Symptoms refractory to conservative and medical treatment
Paraesophageal Hiatus Hernia (Type II) (see Figure 2)
- herniation of all or part of the stomach through the esophageal hiatus into the thorax
- with an undisplaced gastroesophageal (GE) junction
- <10% of esophageal hernias
- clinical presentation
  - asymptomatic
  - heartburn/reflux uncommon (because normal GE junction)
  - pressure sensation in lower chest, dysphagia
- complications
  - hemorrhage
  - incarceration, obstruction, strangulation, gastric stasis ulcer
  - palpitations rarely
- treatment
  - surgery in almost every case to prevent severe complications
  - procedure: reduce hernia, suture to posterior rectus sheath (gastropexy), close defect in hiatus
  - excellent results

Mixed Hiatus Hernia (Type III)
- a combination of Types I and II

ESOPHAGEAL CARCINOMA
- epidemiology
  - 1% of all malignant lesions
  - male:female = 3:1
  - 50-60 years of age (onset)
  - squamous cell carcinoma (SCC) 5x more common in blacks
- risk factors
  - physical agents: alcohol, tobacco, nitrosamines, lye, radiation
  - structural: diverticula, hiatus hernia, achalasia, GERD
  - Barrett's epithelium (8-10% risk of adenocarcinoma, monitor every 1-2 years by endoscopy and biopsy)
  - chronic iron deficiency (Plummer-Vinson syndrome)
- pathology
  - upper 20-33%, middle 33%, lower 33-50%
  - squamous cell carcinoma: 80-85% (esophagus)
  - adenocarcinoma: 5-10% (GE junction) - associated with Barrett's esophagus
- differential diagnosis
  - leiomyoma, metastases, lymphoma, benign stricture, achalasia, GERD, spasms
- clinical presentation
  - frequently asymptomatic - late presentation
  - often dysphagia, first solids then liquids
  - weight loss, weakness, systemic symptoms
  - regurgitation and aspiration (aspiration pneumonia)
  - hematemesis, anemia
  - odynophagia then constant pain
  - tracheoesophageal (TE), bronchoesophageal fistula
  - spread directly or via blood and lymphatics - trachea (coughing), recurrent laryngeal nerves (hoarseness, paralysis), aorta, liver, lung, bone, celiac and mediastinal nodes
- investigations and diagnosis
  - barium swallow first - narrowing site of lesion (shelf or annular lesion) - localizes tumor
  - esophagoscopy - biopsy for tissue diagnosis and resectability/extent of tumour
  - bronchoscopy - for upper and mid esophageal lesions due to high incidence of spread to tracheobronchial tree
  - CT scan (chest/abdomen): for staging - adrenal, liver, lung, bone metastases
  - tracheosophageal U/S
  - CXR, bone scan, LFTs - for metastases staging (see Table 3)

Table 3. Staging of Esophageal Carcinoma

<table>
<thead>
<tr>
<th>Stage</th>
<th>Criteria</th>
<th>Prognosis (5 year survival)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Lamina propria or submucosa</td>
<td>80%</td>
</tr>
<tr>
<td>II</td>
<td>Extension to muscularis propria</td>
<td>33%</td>
</tr>
<tr>
<td>II</td>
<td>Extension to regional nodes</td>
<td>15%</td>
</tr>
<tr>
<td>I</td>
<td>Distant metastases or involvement of continuous structures</td>
<td>0%</td>
</tr>
</tbody>
</table>
treatment
- surgery
  - lower third
    - thoracic esophagectomy, pyloroplasty (or pyloromyotomy) and celiac lymph node resection
    - reconstruction of GI continuity with either stomach or colon
  - middle or upper third
    - esophagectomy extends to cervical esophagus
    - anastomosis performed through separate neck incision
    - contraindications: invasion of tracheobronchial tree or great vessels, lesion > 10 cm
    - radiation
      - if unresectable, palliation (relief of dysphagia in 2/3 of patients, usually transient)
    - chemotherapy
      - alone, or pre and post-operatively
    - multimodal - combined chemotherapy, radiation and surgery
      - palliative or cure, survival rates higher than surgery alone
    - palliative treatment
      - resection, bypass, dilation and stent placement, laser ablation
  - prognosis
    - 5-8% operative death rate
    - five-year survival rates post surgery (stage I = 80%, stage II = 33%, stage III = 14%, stage IV = 0%)
    - prognosis slightly better if squamous cell carcinoma

STRUCTURAL LESIONS (see Gastroenterology Chapter)

MOTILITY DISORDERS (see Gastroenterology Chapter)

OTHER DISORDERS
- esophageal varices (see Liver section)
- Mallory Weiss Tear (see Gastroenterology Chapter)

STOMACH AND DUODENUM

GASTRIC ULCERS (see Gastroenterology Chapter)
- surgical management
- indications for surgery
  - unresponsive to medical treatment (intractability)
  - dysplasia or carcinoma
  - hemorrhage - 3x risk of bleeding as compared to duodenal ulcers
  - obstruction, perforation, penetration
- procedures
  - distal gastrectomy with ulcer excision (Billroth I or Billroth II (see Figure 3))
  - always biopsy ulcer for malignancy
  - always operate if fails to heal completely, even if biopsy negative - could be primary gastric lymphoma
  - vagotomy and pyloroplasty only if acid hypersecretion (rare)

DUODENAL ULCERS (see Gastroenterology Chapter)
- most within 2 cm of pylorus (i.e. duodenal bulb)
- complications
  - perforation ulcer (typically on anterior surface)
    - sudden onset of pain
    - acute abdomen, rigid, diffuse guarding
    - initial chemical peritonitis followed by bacterial peritonitis
    - no bowel sounds, ileus
    - diagnosis: CXR - free air under diaphragm (70% of patients)
    - treatment: oversew ulcer (plication) and omental (Graham) patch or vagotomy and pyloroplasty/antrectomy
  - posterior penetration
    - into pancreas (elevated amylase)
    - constant mid-epigastric pain burrowing into back, unrelated to meals
  - hemorrhage (typically on posterior surface)
    - gastroduodenal artery involvement
    - initial resuscitation with crystalloids, blood transfusion for hypotension and hypovolemia
    - diagnostic and/or therapeutic endoscopy
      (i.e. laser, cautery, injection)
    - surgery if severe or recurrent bleeding
    - procedure: pyloroplasty, truncal vagotomy or vagotomy with antrectomy
STOMACH AND DUODENUM . CONT.

- gastric outlet obstruction
  - due to edema, spasm, fibrosis of pyloric channel
  - nausea and vomiting (undigested food, non-bilious), dilated stomach, crampy abdominal pain
  - surgery after NG decompression and correction of hypochloremic, hypokalemic metabolic alkalosis
  - procedure: vagotomy with antrectomy and gastroduodenostomy or vagotomy with drainage

indications for surgical management
- hemorrhage (massive: > 8 units or relentless), rebleed in hospital, perforation, gastric outlet obstruction, intractable despite medical management

procedures
- truncal vagotomy and drainage via pyloroplasty
  - best combination of safety and effectiveness
  - 5-10% recurrence, but low complication rate
- truncal vagotomy and antrectomy with Billroth I or II anastomosis
  - low recurrence (less than 2%)
  - highest morbidity (dumping, diarrhea) and mortality
  - highly selective vagotomy
  - high recurrence rate (up to 25%)

complications following surgery
- recurrent ulcer, retained antrum, fistula (gastrocolic/ gastrojejunal), dumping syndrome, anemia, postvagotomy diarrhea, afferent loop syndrome

Figure 3. Billroth I and II Gastrectomies

GASTRIC CARCINOMA

epidemiology
- male:female = 3:2
- most common age group 50-59 years
- decreased incidence by 2/3 in past 50 years

risk factors
- smoking
- alcohol
- smoked food, nitrosamines
- H. pylori causing chronic atrophic gastritis
- pernicious anemia associated with achlorhydria and chronic atrophic gastritis
- gastric adenomatous polyps
- previous partial gastrectomy (> 10 years post-gastrectomy)
- hypertrophic gastropathy
- hereditary nonpolyposis colon cancer (HNPCC)
- blood type A

pathology
- histology
  - 92% adenocarcinoma (8% lymphoma, leiomyosarcoma)
- morphology - Borrman classification
  - polypoid (25%)
  - ulcerative (25%)
  - superficial spreading (15%)
  - linitis plastica (10%): diffusely infiltrating
  - advanced/diffuse (35%): tumour has outgrown above 4 categories
STOMACH AND DUODENUM . CONT.

clinical presentation
• suspect when ulcer fails to heal or is on greater curvature of stomach or cardia
• asymptomatic (late onset of symptoms)
• insidious onset of: postprandial abdominal fullness, weight loss, burping, nausea, vomiting, dyspepsia, anorexia, dysphagia, vague epigastric pain, hepatomegaly, epigastric mass (25%), hematemesis, fecal occult blood, iron-deficiency anemia, melena
• rarely: Virchow’s node (left supraclavicular node), Blumer’s shelf (mass in pouch of Douglas), Krukenberg tumour (mets to ovary), Sister Mary Joseph nodule (umbilical nodule), malignant ascites, Irish’s node (left axilla)
• spread: liver, lung, brain

investigation
• EGD and biopsy, upper GI series with air contrast (poor sensitivity if previous gastric surgery)
• CT for distant metastases staging (see Table 4)

Table 4. Staging of Gastric Carcinoma

<table>
<thead>
<tr>
<th>Stage</th>
<th>Criteria</th>
<th>Prognosis (5 year survival)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>mucosa and submucosa</td>
<td>70%</td>
</tr>
<tr>
<td>II</td>
<td>extension to muscularis propria</td>
<td>30%</td>
</tr>
<tr>
<td>III</td>
<td>extension to regional nodes</td>
<td>10%</td>
</tr>
<tr>
<td>I</td>
<td>distant metastases or involvement of continuous structures</td>
<td>0%</td>
</tr>
<tr>
<td>V</td>
<td>overall</td>
<td>10%</td>
</tr>
</tbody>
</table>

Treatment
• surgery for adenocarcinoma
• proximal lesions
  • total gastrectomy and esophagojejunostomy (Roux-en-Y) (see Figure 4)
  • include lymph node drainage to clear celiac axis (may require splenectomy)
• distal lesions
  • distal radical gastrectomy (wide margins, en bloc removal of omentum and lymph node drainage)
• palliation
  • gastric resection to decrease bleeding and to relieve obstruction thus enabling the patient to eat
• lymphoma
  • chemotherapy ± surgery ± radiation

Figure 4. Roux-en-Y Anastomosis
Illustration by Janice Chan

BARIATRIC SURGERY
weight reduction surgery for morbid obesity
surgical options
• gastric bypass
  • staple off small gastric pouch with roux-en-y limb to pouch
• vertical banded gastroplasty
  • vertical stapled small gastric pouch with placement of silastic ring band
• complications
  • dumping syndrome
  • malnutrition

Ligament of Treitz
STOMACH AND DUODENUM. CONT.

COMPlications of GASTRIC surGERY

general
• anesthetic reaction
• post-op complications

specific
• alkaline reflux gastritis
  • duodenal contents (bilious) reflux into stomach
  • common postgastrectomy (25%)
  • postprandial epigastric pain, nausea, vomiting, weight loss, anemia
  • diagnosis: endoscopy and biopsy (gastritis, bile reflux)
  • treatment
    • medical: H2 blocker, metoclopramide, cholestyramine
    • surgical: conversion of Billroth I or II to Roux-en-Y
• afferent loop syndrome - occurs with Billroth II
  • early postprandial distention, RUQ pain, nausea, bilious vomiting, anemia
  • caused by intermittent mechanical obstruction and distension of afferent limb
    (accumulated bile and pancreatic secretions)
  • treated by increasing drainage of afferent loop by conversion to Roux-en-Y
• dumping syndrome - seen in postgastrectomy patients
  1. early
    • caused by hyperosmotic chyme release into small bowel
    resultiing in fluid accumulation and jejunal distention
    • post-prandial symptoms: epigastric fullness or pain, emesis, weakness,
      nausea, palpitations, dizziness, diarrhea, tachycardia, diaphoresis
    • treatment: small multiple low carbohydrate, low fat and high protein diet
      with avoidance of liquids at meals (last resort: delay gastric emptying by
      interposition of antiperistaltic jejunal loop between stomach and small bowel)
  2. late
    • large glucose load leads to large insulin release and hypoglycemia
    • treatment: small snack 2 hours after meals
• blind-loop syndrome – after Billroth II
  • bacterial overgrowth (colon-type gram negative bacteria (GNB)) in afferent limb; leads to
    anemia/weakness, diarrhea, malnutrition, abdo pain and hypocalcemia
  • broad spectrum antibiotics; may convert to Billroth I
• postvagotomy diarrhea (up to 25%)
  • because bile salts in colon inhibit water resorption
  • treatment
    • medical: cholestyramine
    • surgical: reversed interposition jejunal segment
  • usually improves

SMALL INTESTINE

SMALL BOWEL OBSTRUCTION (SBO)

disruption of the normal flow of intestinal contents —> dilation proximal to the blockage
(stomach and proximal bowel) —> decompression of distal bowel
complete or partial
non-strangulating or strangulating (usually in the setting of complete obstruction)
diagnosis is typically based upon clinical and radiological features
must differentiate from non-obstructive motility disorders (paralytic ileus and intestinal pseudo-obstruction)
(see table 5) as they may present with symptoms similar to that of an obstruction, but are not associated with
a mechanical obstruction
must characterize: acute vs. chronic, partial vs. complete, small vs. large bowel,
mechanical vs. functional in summarization

Etiology

Table 5. Causes of Small Bowel Obstruction

<table>
<thead>
<tr>
<th>Extrinsic Lesions</th>
<th>Intrinsic Lesions</th>
<th>Intraluminal Obstruction</th>
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<tbody>
<tr>
<td>Adhesions (60%)</td>
<td>Neoplasms (15%)</td>
<td>Gallstone ileus</td>
</tr>
<tr>
<td>- in patients with prior abdominal surgery</td>
<td>Inflammatory lesions/strictures:</td>
<td>Intussusception</td>
</tr>
<tr>
<td>Hernia (20%)</td>
<td>- Crohn’s, radiation enteritis/stricture</td>
<td>Feces/meconium</td>
</tr>
<tr>
<td>Volvulus</td>
<td>Duplication, atresia, stenosis</td>
<td>Bezoar</td>
</tr>
<tr>
<td>Annular pancreas</td>
<td>Congenital malformations</td>
<td>Intramural hematoma</td>
</tr>
<tr>
<td>Neoplasms</td>
<td>Cystic fibrosis (CF)</td>
<td>Foreign body (barium, worms)</td>
</tr>
<tr>
<td></td>
<td>Superior mesenteric artery (SMA)</td>
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<tr>
<td></td>
<td>sydrome (ischemia)</td>
<td></td>
</tr>
</tbody>
</table>

UpToDate Clinical Reference Library Release 9.2, Clinical Manifestations and Diagnosis of Small Bowel Obstruction

GS10 – General Surgery MCCQE 2006 Review Notes
pathogenesis
• intestinal secretions can pass distally
• swallowed air and gas from bacteria contribute to dilatation
• bowel wall edema and loss of absorptive function —> increased fluid in lumen
—> transudative fluid loss into peritoneal cavity

clinical presentation
• abdominal distention, nausea, vomiting, crampy abdominal pain, +/- obstipation
• flatus and passing of feces may occur after the onset of obstruction as the colon takes
  12-24 hours to empty
  1) non-strangulating obstruction
     • proximal obstruction
     • profuse early vomiting (often bilious)
       - dehydration
     • colicky abdominal pain
     • minimal abdominal distension
       (proximal bowel acts as a reservoir when dilated)
     • middle level obstruction
       • moderate vomiting after onset of pain
       • abdominal distension
       • intermittent colicky pain
       • obstipation
     • distal obstruction
       • late feculent vomiting
       • marked abdominal distension and peristaltic rushes
       • obstipation, variable pain
  2) strangulating obstruction (10% of bowel obstructions) - surgical emergency
     • edema and intraluminal pressure cause a decrease in perfusion,
       impaired blood supply leads to necrosis
     • early shock
     • fever, leukocytosis, tachycardia
     • cramping pain turns to continuous ache
     • vomiting gross or occult blood
     • abdominal tenderness or rigidity (peritonitis)
     • melena if infarcted

investigations
• radiological (see Colour Atlas G1)
  • upright CXR (r/o presence of free air) (left lateral decubitus (LLD) if unable to do an upright film)
  • abdominal x-ray (3 views) (air-fluid levels and dilated edematous loops of bowel
    (ladder pattern - plica circularae), colon devoid of gas unless partial obstruction
  • if ischemic bowel look for: free air, pneumatosis, thickened bowel wall, air in portal vein
    • adjuvant: 1) CT provides information on presence, level, severity, cause
      2) small bowel series detects and determines degree of obstruction
      3) ultrasound is useful as a bedside test or for pregnant patients
• laboratory
  • NOT diagnostic
  • normal early in disease course
  • BUN, creatinine, hematocrit (hemoconcentration) to assess degree of dehydration
  • strangulation (leukocytosis with left (L) shift, elevated serum lactate, LDH
    (sensitive, not specific)
  • increased amylase
  • metabolic alkalosis —> proximal SBO and frequent emesis
  • metabolic acidosis —> bowel infarction

prognosis
• mortality: non-strangulating 2%, strangulating 8% (25% if >36 hours), ischemic up to 85%

complications
• strangulation —> necrosis —> open perforation
• septicemia
• hypovolemia
Table 6. Small Bowel Obstruction (SBO) vs. Paralytic Ileus

<table>
<thead>
<tr>
<th></th>
<th>Small Bowel Obstruction (SBO)</th>
<th>Paralytic Ileus</th>
</tr>
</thead>
<tbody>
<tr>
<td>nausea and vomiting</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>abdominal distention</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>obstipation</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>abdominal pain</td>
<td>crampy</td>
<td>minimal or absent</td>
</tr>
<tr>
<td>bowel sounds</td>
<td>normal, increased</td>
<td>absent, decreased</td>
</tr>
<tr>
<td>abdominal x-ray (AXR)</td>
<td>ladder pattern, air fluid levels, no gas in colon</td>
<td>gas present throughout small and large colon</td>
</tr>
</tbody>
</table>

TUMOURS OF SMALL INTESTINE
very rare (1-5% of GI tumours)
theories for low incidence: liquid contents prevent mucosal irritation, rapid transit of contents, low bacterial load, increased lymphoid tissue
predisposing conditions
• exposure to carcinogens (dietary red meat)
• familial colonic polyposis, Peutz-Jeghres syndrome, Gardner’s syndrome
• Crohn’s disease, celiac disease
• immunodeficiency, autoimmune disorders
clinical presentation
• does not distinguish between benign vs. malignant
• usually asymptomatic until advanced
• most common: intermittent obstruction, intussusception, occult bleeding, palpable abdominal mass, abdominal pain
Benign
• usually asymptomatic until large
• 10 times more common than malignant
• most common sites: terminal ileum, proximal jejunum
• types:
  1. polyps
     • adenomas: 1) simple villous – significant malignant transformation
     2) tubular - low malignant potential
     3) Brunner’s gland adenomas – rare, hyperplasia of gland
     • familial adenomatous polyposis
     • multiple intestinal polyps in association with desmoid tumours, mandible or skull osteomas, sebaceous cysts
     • malignant degeneration of polyps common
     • hamartomatous
     • overgrowth and abnormal arrangement of normal cells
     • may be associated with Peutz-Jeghers syndrome
     • multiple polyoid hamartomas and pigmentation (perioral and palms of hands and soles of feet)
     • rarely malignant
     • autosomal dominant inheritance
     • treatment: surgical
  2. leiomyomas
     • arise from smooth muscle cells
     • usually enlarge extraluminally therefore present when they outstrip their blood supply
  3. lipomas
     • submucosal lesion arising from submucosal adipose tissue or serosal fat
  4. hemangiomas
Malignant
• usually asymptomatic until advanced stage
• 25 – 30% associated with distant metastases at time of diagnosis
• types
  1) adenocarcinoma 40% (most common primary tumour of small intestine)
     • usually 50 – 70 years old, with male predominance
     • usually in proximal small bowel, incidence decreases distally
     • higher risk in Crohn’s disease (lesion usually at the ileum) and with previous colorectal cancer (common etiology?)
     • early metastases to lymph nodes (80% metastatic at time of operation)
     • 5 year survival 25%
     • often asymptomatic, may cause SBO
     • diagnosis - small bowel follow through or enteroclysis

GS12 – General Surgery  MCCQE 2006 Review Notes
2) carcinoid 50%
   - increased incidence between 50–60 years old
   - originate from enterochromaffin cell in the crypts
   - most commonly 60 cm from the ileocecal (IC) valve
   - may be associated with multiple endocrine neoplasia (MEN) I and II
   - often slow-growing
   - classified by embryological origin; (correlate with morphology, behaviour)
     - foregut – stomach, duodenum, pancreas
     - midgut – jejunum, ileum, appendix ascending colon
     - hindgut – transverse, descending and sigmoid colon, rectum
   - common sites; appendix 46%, distal ileum 28%, rectum 17%
   - clinical presentation
     - usually asymptomatic and found incidentally
     - obstruction, bleeding, crampy abdominal pain, intussusception
     - carcinoid syndrome (< 10%)
       - requires liver involvement, +/- mets (bronchi, ovaries, testes)
       - lesion secretes serotonin, kinins and vasoactive peptides
directly to systemic circulation (normally inactivated by liver)
     - hot flushes, hypotension, diarrhea, bronchoconstriction (wheezing),
and tricuspid/pulmonic valve insufficiency, right heart failure
   - diagnosis: most found at surgery for obstruction or appendectomy,
elevated 5-HIAA (breakdown product of serotonin) in urine, or
increased 5-HT in blood
   - treatment: resect tumour and mets, +/- chemotherpay, treat carcinoid
syndrome (steroids, histamine, octreotide)
   - metastatic risk - 2% if size < 1 cm, 90% if > 2 cm
   - 5 year survival 70%, unless liver mets (20%)

3) lymphoma 20%
   - highest incidence at 70 years old, more common in males
   - usually non-Hodgkin’s lymphoma
   - usually distal ileum
   - proximal jejunum in patients with celiac disease
   - clinically: fatigue, weight loss, abdominal pain, fever, malabsorption,
   - rarely – perforation, obstruction, bleeding, intussusception
   - treatment
     - low grade: chemotherapy with cyclophosphamide
     - high grade: surgical resection, radiation
   - palliative: somatostatin, doxorubicin
   - prognosis: 65-80% overall; 95% if localized
   - survival: 40% at 5 years

4) metastatic
   - most common site for metastatic melanoma
   - hematogenous: breast, lung, kidney
   - direct extension: cervical ovarian, colon

5.) sarcomas (leiomyosarcoma)
   - most common in jejunum, ileum, Meckel’s diverticulum
   - enlarge extraluminally —> late obstruction

**MECKEL’S DIVERTICULUM**
remnant of the embryonic vitelline duct (on antimesenteric border of ileum)
most common true diverticulum of GI tract
several types of mucosa (heterotopic); gastric, pancreatic, colonic
rule of 2’s: 2% of the population, symptomatic in 2% of cases, found within 2 feet
(10-90 cm) of the ileocecal (IC) valve, 2 inches in length, often present within 2 years of age
clinical presentation (4% symptomatic): GI bleed, SBO, diverticulitis (mimic appendicitis)
note: painless bleeding due to ulceration caused by ectopic gastric mucosa (50% of
patients with this presentation are < 2 years old)
diagnosed with technetium Tc99 to identify the ectopic gastric mucosa
complications; fistula (umbilicus-ileum, umbilical sinus), fibrous cord between
umbilicus-ileum may cause SBO due to volvulus, intussusception, perforation
treatment: incidental finding —> surgical resection
symptomatic —> fluid and electrolyte restoration and surgical resection
(if broad based —> segmental resection to remove all the mucosal types)

**FISTULA**
abnormal communication between two epithelialized surfaces
etiology
- foreign object erosion (e.g. gallstone, graft)
- infection
- inflammatory bowel disease (IBD) (especially Crohn's disease)
- congenital
- trauma
- iatrogenic
types
- tracheoesophageal, aortoesophageal, aortoenteric
- biliaryenteric, cholecystogastric/duodenal/hepatic/colonic
- coloenteric, colonic, etc.

why fistulas stay open (FRIENDO)
- Foreign body
- Radiation
- Infection
- Epithelialization
- Neoplasm
- Distal obstruction (most common)
- Others: increased flow; steroids (may inhibit closure, usually will not maintain fistula)

bowel fistula management
- relieve obstruction
- fluid and electrolyte balance
- nutrition - elemental/low residue
- decrease flow - NPO, TPN
- decrease secretion - octreotide/somatostatin
- identify anatomy – fistulogram, sinogram
- surgical intervention dependent upon etiology, or uncertainty of diagnosis

APPENDICITIS

epidemiology
- 6% of population, higher incidence among men
- 80% between 5-35 years of age
- diagnosis may be difficult (atypical presentation in very young and very old)
- patients may not seek medical attention early

pathogenesis
- luminal obstruction of appendix
- children/young adult: hyperplasia of lymphoid follicles, initiated by infection
- adult: fibrosis/stricture, fecolith, neoplasm
- all ages: parasites, foreign body, neoplasm (rare)
- natural history: obstruction → bacterial overgrowth → inflammation/swelling
  → increased pressure → localized ischemia → gangrene/perforation
  → contained abscess or peritonitis

clinical presentation
- only reliable feature is progression of signs and symptoms
- low grade fever, anorexia
- nausea and vomiting after pain starts
- early (localized inflammation): constant dull, poorly localized abdominal pain, periumbilical (due to visceral innervation and embryological origins)
- late: well localized pain where the appendix irritates overlying parietal peritoneum
- inferior (to cecum) appendix → pain at McBurney's point: pain 1/3 of the distance along a line drawn between the anterior iliac spine and umbilicus,
  Rovsing's sign: pressure in the LLQ elicits pain in the RLQ, rectal exam may elicit pain as well
- retrocecal appendix → positive psoas sign: pain on extension of the right hip
- pelvic appendix → positive obturator sign: pain with passive rotation of the flexed right hip when in the supine position, +/- urinary frequency, dysuria, diarrhea
- perforation → peritonitis → rebound tenderness
- pregnancy: appendix may be superior at the level of the fundus, pain may be in the RUQ

diagnosis
- laboratory (not diagnostic, help to rule out other diagnoses i.e. UTI)
- mild leukocytosis (although many have normal WBC counts) with left shift
- higher leukocyte count with perforation
- radiology (not very helpful in establishing a diagnosis)
- x-rays: usually nonspecific; free air if perforated, look for calculus
- CT scan (standard or appendiceal CT with rectal contrast): thick wall, appendicolith
- consider ultrasound or laparoscopy in female

differential diagnosis
- cecal diverticulitis, Meckel’s diverticulitis
- ileitis, Crohn’s disease, pelvic inflammatory disease

treatment
- surgical (possible laparoscopy)
- 70-80% rate of true appendicitis is acceptable
- need to be aggressive with young females as perforation may cause infertility due to tubal damage
- hydration, correct electrolyte abnormalities
- perioperative antibiotics: non-perforated → cefazolin + metronidazole
  perforated → ceftriaxone + metronidazole
complications
• perforation: 25-30%, more common at extremes of age, increase in fever and pain
• peritonitis: local (if walled-off by omentum) or generalized
• appendiceal abscess (phlegmon)
  • presents as appendicitis plus RLQ mass
  • diagnosis by U/S or CT
  • interval appendectomy (6 weeks) as needed after optimal preparation (aspiration, antibiotics)
• morbidity/mortality 0.6% (uncomplicated), 5% if perforated

TUMOURS OF THE APPENDIX (rare)
carcinoid tumours (most common type)
• appendix is the most common location
• can be benign (90% in appendix) or malignant
• usually asymptomatic
• may produce carcinoid syndrome with liver metastases
• treatment: 1) appendectomy if < 2 cm and not extending into serosa
  2) right hemicolecotomy if > 2 cm, through the serosa, or nodal or base of appendix involvement (increased incidence of malignancy)
adenocarcinoma
• 50% present as acute appendicitis
• spreads rapidly to lymph nodes, ovaries, and peritoneal surfaces
• treatment: right hemicolecotomy
malignant mucinous cystadenocarcinoma
• usually present as abdominal distension and pain
• treatment: appendectomy
• prognosis: local recurrence is inevitable, mortality 50% at 5 years

INFLAMMATORY BOWEL DISEASE (IBD)

<table>
<thead>
<tr>
<th>Table 7. Differentiating Features of IBD</th>
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</thead>
<tbody>
<tr>
<td><strong>Area of involvement</strong></td>
</tr>
<tr>
<td>Colonic, always rectum</td>
</tr>
<tr>
<td><strong>Pattern of involvement</strong></td>
</tr>
<tr>
<td>Bloody</td>
</tr>
<tr>
<td><strong>Diarrhea</strong></td>
</tr>
<tr>
<td>Less often</td>
</tr>
<tr>
<td><strong>Abdominal pain</strong></td>
</tr>
<tr>
<td>Sudden</td>
</tr>
<tr>
<td><strong>Extraintestinal disease</strong></td>
</tr>
<tr>
<td><strong>RLQ mass</strong></td>
</tr>
<tr>
<td>Superficial ulceration, mucosal shine</td>
</tr>
<tr>
<td>erythema, continuous lesion, pseudopolyps</td>
</tr>
<tr>
<td><strong>Perianal disease</strong></td>
</tr>
<tr>
<td><strong>Fistula, Strictures</strong></td>
</tr>
<tr>
<td><strong>Endoscopic findings</strong></td>
</tr>
<tr>
<td><strong>Histological Features</strong></td>
</tr>
<tr>
<td><strong>Increased Cancer Risk</strong></td>
</tr>
</tbody>
</table>


Surgical Management
- can alleviate symptoms, address complications, improve quality of life and be lifesaving
- goal of surgery is to conserve bowel - resect as little as possible
  1) optimize medical status: correct anemia, fluids/electrolytes, acid-base disorders, nutrition (may require TPN and bowel rest)
  2) hold immunosuppressive therapy pre-op, corticosteroids must be tapered post-op
  3) bowel cleansing to decrease fecal contamination (contraindicated if obstructed or acute abdomen)
  4) antibiotic prophylaxis one day prior to surgery (i.e metronidazole) and IV antibiotics one hour prior to surgery (a cephalosporin)
  5) deep vein thromboembolism (DVT) prophylaxis – heparin (IBD patients at increased risk of thromboembolic events)
CROHN'S DISEASE (see Gastroenterology Chapter) (see Colour Atlas G4)
intervention required in 70-75% of patients when complications arise
not curable by surgery
surgery reserved for those with complications or refractory to medical therapy
indications for surgical management
• most common: SBO (due to stricture/inflammation) – indication in 50% of surgical cases,
abscess/fistula (enterocolic, vesicular, vaginal, cutaneous abscess)
• failure of medical management (intractable disease – more common with colonic involvement)
less common: perforation, hemorrhage, quality of life issues, chronic disability, toxic
megacolon, failure to thrive (especially children), perianal disease
procedures
• resection and anastomosis/ostomy: if active/subacute inflammation, perforation, fistula
• side-to-side anastomosis may decrease likelihood of symptom recurrence
• ileocecal resection with incidental appendectomy (unless base of appendix involved)
• strictureplasty - widens lumen in chronically scarred bowel – relieves obstruction
(not done if acute inflammation exists),
• exclusion bypass - bypass unresectable inflammatory mass, but later risk of
abscess, perforation, hemorrhage, malignancy
• others: balloon dilation (dilates strictures risk of perforation), stenting, laparoscopy
complications
• short gut syndrome (diarrhea, steatorrhea, malnutrition)
• fistulas
• biliary stones (as decreased bile salt absorption leads to increased
cholesterol precipitation)
• kidney stones (due to loss of Ca^{2+} in diarrhea leading to increased oxalate
absorption and hyperoxaluria → stones)
prognosis
• recurrence rate at 10 years: ileocolic (50%), small bowel (50%), colonic (40-50%)
• 80-85% of patients who need surgery lead normal lives
• mortality 15% at 30 years
• re-operation at 5 years: primary resection 20%, bypass 50%
• to decrease risk of recurrence: mesalamine (5-ASA) +/- metronidazole post-op

ULCERATIVE COLITIS (UC) (see Gastroenterology Chapter) (see Colour Atlas G5)
surgery improves quality of life and reduces the risk of malignancy
indications for surgical management
• hemorrhage
• obstruction, perforation
• elective, poor control, unable to taper steroids
• toxic megacolon (emergency) - leading cause of death in UC, 40% of cases fatal
• cancer risk, failure to thrive in children
procedures
• proctocolectomy and ileoanal anastomosis (operation of choice)
• proctocolectomy with permanent ileostomy (if not a candidate for ileoanal procedures)
• if emergency: total abdominal colectomy and ileostomy with Hartmann closure of the rectum,
rectal preservation
• colectomy, mucosal proctectomy, and ileal pouch-anal canal anastomosis (IPAA)
• colectomy and stapled ileal pouch distal rectal anastomosis (IPDRA)
complications
• early: bowel obstruction, pouch bleeding, transient urinary dysfunction,
derhydration (high output from ostomy)
• late: stricture, anal fistula/abscess, pouchitis, poor anorectal function, reduced fertility
prognosis
• mortality: 5% over 10 years
• 2% mortality with elective surgery, 8-15% mortality with emergency surgery
• total proctocolectomy will completely eliminate risk of cancer

ILEOSTOMIES AND COLOSTOMIES  connection of the GI tract to abdominal wall skin: a manmade fistula
ileostomies
• Brooke: incontinent, continuous drainage
• Kock’s continent ileostomy: constructing a reservoir with a valve made from
the small intestine increased complications (obstruction)
• Ileal conduit: loops of stapled-off ileum made into a pouch, anastamosed to
ureters and brought out to abdominal wall skin (to drain urine)
coloctomies
• loop colostomy: an opening created in a loop of bowel, brought to the skin surface
• end (terminal) colostomy: colon is divided and one end is brought out to the skin surface
• double barrel colostomy: end colostomy and a mucous fistula
complications (20%)
• obstruction: herniation, stenosis (skin and abdominal wall)
• peri-ileostomy abscess and fistula
• skin irritation
• prolapse or retraction
LARGE INTESTINE

LARGE BOWEL OBSTRUCTION (LBO)
10 – 15% of all intestinal obstruction
most commonly at sigmoid colon
etiology
• colon adenocarcinoma (65%)
• scarring associated with diverticulitis (20%)
• volvulus (rotation of intestinal segment on mesentery axis- typically sigmoid) (5%)
• other causes: IBD, benign tumours, fecal impaction, foreign body, adhesions, hernia (especially sliding type), intussusception (children), endometriosis
clinical presentation
• slower in onset, less pain, later onset of vomiting, less fluid/electrolyte disturbance than SBO
• abdominal distention, crampy abdominal pain in hypogastrium
• constipation, obstipation, anorexia
• nausea and late feculent vomiting
• high-pitched (borborygmi) or absent bowel sounds
• may have visible peristaltic waves
• complete obstruction: obstipation and no flatus/stool > 8 hours —> emergent operation
• partial obstruction: passage of some gas/stool —> NG decompression, IV fluids, then O.R.
• open loop (10-20%) (safe):
  • incompetant ileocecal valve allows relief of colonic pressure as contents reflux into ileum, therefore clinical presentation similar to SBO
• closed loop (80-90%) (dangerous):
  • ileocecal valve competent, allowing build up of colonic pressures to dangerous level
  • massive colonic distention
  • high risk of perforation if cecum diameter > 12 cm on AXR
  • compromise of lymphatic, venous and arterial circulation (infarction
  • cecum at greatest risk of perforation due to Laplace’s Law (pressure = wall tension/radius)
  • suspect impending perforation in the presence of tenderness over the cecum
• if obstruction at ileocecal valve symptoms of SBO
Ogilvie’s syndrome: pseudo-obstruction, distention of colon without mechanical obstruction
• associations: long term debilitation, chronic disease, immobility, narcotic use, polypharmacy, recent orthopedic surgery, post-partum, hypokalemia
• diagnosis: cecal dilatation on AXR, if diameter > 12 cm, increased risk of perforation
• treatment: decompression with colonoscopy or enema
• if unsuccessful - decompression with nasogastric tube, rectal tube
• if perforation or ischemia - surgery
investigation
• x-ray: "picture frame" appearance, distended proximal colon, air-fluid levels
• hypaque enema – confirms the diagnosis and identifies the location (no oral contrast)
• do not use contrast - may become inspissated and convert partial to complete LBO
• goal: decompression to prevent perforation
• correct fluid and electrolyte imbalance, NG suction, continuous observation
• surgical correction of obstruction (usually requires temporary colostomy)
• volvulus: sigmoidoscopic decompression or barium enema followed by operative reduction if unsuccessful
prognosis
• dependent upon age, general medical condition, vascular impairment of bowel, perforation, promptness of surgical management
mortality
• overall: 20%
• cecal perforation: 40%

DIVERTICULAR DISEASE (see Colour Atlas G3)
diverticulum - abnormal sac or pouch protruding from the wall of a hollow organ
diverticulosis - presence of diverticula
right sided (true) diverticula = contains all layers (congenital)
left sided (false) diverticula = contain only mucosal and submucosal layers (acquired)
Asymptomatic (70%)  
Diverticular bleed (5 – 15%)  
Diverticulitis (15 – 20%)  
  Simple (75%)  
  Complicated (25%)  
       - abscess  
       - obstruction  
       - perforation  
       - fistula

Figure 5. Natural History of Diverticulosis

epidemiology
• 35-50% of general population (M=F)
• increase incidence in 5th – 8th decades of life
• 95% involve sigmoid colon (site of highest pressure)
• majority are asymptomatic (approximately 80-90%)
• higher incidence in Western countries, related to low fibre content in diet

pathogenesis
• related to high intraluminal pressure and defects in the colonic wall
• factors: fibre-deficient diet increases gut transit time, thickening of the muscle layer due to elastin deposition and structural changes of collagen, result in decreased wall resistance, luminal narrowing and increased pressure
• muscle wall weakness from aging and illness
• diverticula occur at greatest area of weakness, most commonly at the site of penetrating vessels, therefore increased risk of hemorrhage

clinical presentation
• uncomplicated diverticula - asymptomatic (80%), usually an incidental finding
• recurrent abdominal pain (usually LLQ), bloating, flatulence
• alternating/irregular bowel habits
• absence of fever/leukocytosis
• no physical exam findings or poorly localized LLQ tenderness.
• bleeding - 2/3 of all massive lower gastrointestinal bleeds (see Diverticular Bleed section)
• may develop diverticulitis (see Diverticulitis section)

treatment
• medical: high fibre diet, education, reassurance
• surgical: treat massive hemorrhage, rule out carcinoma, treat complications

Diverticular Bleed
penetrating vessels create wall weakness and diverticulum form
vessel is separated from the lumen only by mucosa, vessel wall weakens as it is injured and rupture can occur usually a PAINLESS rectal bleed (maroon or bright red blood)
Diverticulitis ("left sided appendicitis")
erosion of the wall by increased intraluminal pressure or inspissated food particles
---> inflammation and focal necrosis --> perforation
usually mild inflammation with perforation walled off by pericolic fat
often involves sigmoid colon
clinical presentation
• depends on severity of inflammation and presence of complications
  • left lower quadrant (LLQ) pain/tenderness, present for several days before admission
  • constipation, diarrhea, urinary symptoms (dysuria if inflammation adjacent to bladder)
  • palpable mass if phlegmon or abscess, nausea, vomiting
  • low-grade fever, mild leukocytosis
  • occult or gross blood in stool less common
• generalized tenderness suggests free perforation and peritonitis
complcations
• abscess - on physical exam palpable abdominal mass
• fistula
  • colovesicular (most common) – pneumaturia, fecaluria, recurrent UTI’s
  • coloenteric – diarrhea
  • colovaginal – stool per vagina
  • colocutaneous – furuncle, stool drainage
• obstruction – due to scarring from repeated inflammation
• perforation
• peritonitis – rare
investigations
• to confirm the diagnosis and rule out other sources of abdominal signs
• plain film x-ray
  • localized diverticulitis: ileus, thickened wall, SBO, partial colonic obstruction
  • free air may be seen in 30% with perforation and generalized peritonitis
• barium enema - contraindicated during an acute attack
  • risk chemical peritonitis (if perforation)
  • interferes with subsequent investigations (colonoscopy) and treatment (anastomosis)
• hypaque (water soluble) contrast - SAFE
  • saw-tooth pattern (colonic spasm)
  • may show site of perforation
  • trickle of contrast out of colon
  • abscess cavities or sinus tracts
• compression ultrasound
  • look for abnormal colonic segment at point of maximal tenderness
  • useful to follow the resolution of an abscess
• sigmoidoscopy/colonoscopy
  • not during an acute attack, only done on an elective basis
  • rule out other lesions, polyps, cancer, take biopsies
  • mucosal edema, erythema --> cannot advance scope
• CT scan (may be the optimal method of investigation)
  • very helpful in localizing an abscess

<table>
<thead>
<tr>
<th>Mild</th>
<th>Uncomplicated</th>
<th>Uncomplicated</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oral ciprofloxacin +/- flagyl</td>
<td>Improved</td>
<td>IV antibiotics</td>
</tr>
<tr>
<td>clear fluids</td>
<td>Not improved</td>
<td>NPO</td>
</tr>
<tr>
<td>Improved</td>
<td>F/U barium enema or Colonoscopy</td>
<td>Uncomplicated</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Moderate</th>
<th>Complicated</th>
</tr>
</thead>
<tbody>
<tr>
<td>Uncomplicated</td>
<td>Complicated</td>
</tr>
<tr>
<td>IV antibiotics</td>
<td>Abscess – CT drainage</td>
</tr>
<tr>
<td>NPO</td>
<td>Obstruction, Fistula</td>
</tr>
<tr>
<td>Improved</td>
<td>Not improved</td>
</tr>
<tr>
<td>Not improved</td>
<td>Surgery 1 – 2 stage</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Severe/Diffuse Peritonitis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Surgery 2 stage</td>
</tr>
</tbody>
</table>

Figure 7. Treatment of Diverticulitis

*UpToDate Clinical Reference Library Release 9.1, Treatment of Acute Diverticulitis*
LARGE INTESTINE... CONT.

treatment (see Figure 7)
• conservative and medical (50% resolve)
  • localized (omentum has walled-off area)
  • NPO, IV, NG tube, antibiotics (ciprofloxacin, metronidazole) and analgesia
  • observe every 2-4 hours
• surgical indications for diverticulitis
  • complications - sepsis (secondary to perforation, abscess),
    hemorrhage, fistula (vesical, vaginal, cutaneous), obstruction
    (extra-luminal abscess, chronic fibrosis)
  • recurrent inflammation, persistent pain or mass, right sided diverticulitis,
    age < 40, clinical deterioration within 48 hours, rule out cancer
• surgical procedures
  • resection with colostomy and closure of distal rectal stump (Hartmann procedure)
    (see Figure 8), re-anastomosis 3 months later if acute diverticulitis
  • sigmoidectomy and primary colorectal anastomosis is an alternative procedure if non-acute

Figure 8. Hartmann Procedure

Illustration by Myra Rudakewich

ANGIODYSPLASIA (see Colour Atlas G17)
pathogenesis
• a vascular anomaly
• end result is focal submucosal venous dilatation and tortuosity
• theories:
  1) response to chronic low grade venous obstruction
  2) complication from chronic mucosal ischemia
  3) complication of local ischemia associated with cardiac, vascular, pulmonary disease
  4) congenital (likely in the young and those with congenital diseases)
histology: dilated thin-walled vessels in the mucosa and submucosa covered by a single layer of epithelium
most frequently in right colon of patients > 60 years old
bleeding typically intermittent (melena, anemia, guaiac positive stools) and in the elderly
diagnosis
• endoscopy (cherry red spots, branching pattern from central vessel)
• angiography (slow filling/early emptying mesenteric vein, vascular tuft)
• red cell technetium scan
• barium enema is contraindicated (obscures other x-rays, i.e. angiogram)
treatment if symptomatic, if incidental finding —> NO treatment
• electrocautery through colonoscope
• right hemicolectomy with ileostomy (if bleeding persists or recurs)
• endoscopic embolization or vasopressin infusion by angiography (temporary procedure,
  risk of colonic necrosis or perforation)
• other: sclerotherapy, band ligation, lasers, argon plasma coagulation, octreotide

ISCHEMIC COLITIS reduced blood flow due to occlusion, vasospasm, or hypoperfusional states
acute: occlusive (emboli/thrombosis) or nonocclusive (vasoconstriction) obstruction of artery or vein
chronic: usually due to atherosclerotic disease
• more common in the elderly, although cases in young adults have been reported
• associated with atherosclerosis, diabetes, vasculitis, estrogen use
• usually affects the “watershed” area of the colon (splenic flexure, right colon)
presentation: sudden onset of LLQ pain —> bright red blood per rectum (BRBPR)
• colonoscopy = diagnostic modality of choice; showing petechial hemorrhage,
  friability and ulceration, allowing for confirmatory biopsies to be obtained
treatment
• depends on severity
• supportive if no gangrene or perforation
• bowel rest and monitor recovery
• broad spectrum antibiotics if moderate to severe
• most subside in 24-48 hours, and resolve in 2 weeks
• if clinically deteriorate —> laparotomy and segmental resection
LARGE INTESTINE . . . CONT.

VOLVULUS

rotation of segment of bowel about its mesentery
50% of patients > 70 years old as stretching/elongation of bowel with age is a predisposing factor
symptoms due to bowel obstruction or bowel ischemia
clinical presentation
• sigmoid (70%)
  • intermittent crampy pains, obstipation, distension, anorexia, nausea, vomiting
  • secondary to high residue diets, elongated colon, chronic constipation, laxative abuse, pregnancy, elderly, bedridden, institutionalized
• cecum (30%) - congenital anomaly – hypermobile cecum as ascending colon is incompletely fixated (ideopathic)
  • like distal SBO presentation: colicky pain, vomiting, obstipation +/- distension
investigations
• plain x-ray
  • “omega or bent inner tube sign” of dilated bowel loop (sigmoid)
  • "coffee-bean" shape of dilated bowel loop (with no haustra) (cecum)
  • concavity of “bean” points right for cecal volvulus, left for sigmoid
• barium/gastrografin enema
  • “ace of spades” appearance due to contrast-filled lumen tapering of upper end of lower segment (funnel-like narrowing)
• sigmoidoscopy or colonoscopy as appropriate
treatment
• cecum
  • correct fluid and electrolyte imbalance
  • always operate - cecopexy (suture bowel to parietal peritoneum) or right colectomy with ileotransverse colonic anastomosis
• sigmoid
  • nonsurgical decompression by flexible sigmoidoscopy and insertion of rectal tube past obstruction
  • evidence of strangulation, perforation or unsuccessful decompression —> OPERATE (Hartmann procedure) (see Figure 8)
  • elective surgery recommended (recurrence = 50-70%)

COLORECTAL POLYPS

polyp = a small mucosal outgrowth which grows into the lumen of the colon or rectum
• can be sessile (flat) or pedunculated (round with a long thin neck attached to mucosa)
• prevalence: 30% at age 50, 40% at age 60, 50% at age 70
• 50% in the rectosigmoid region 50% are multiple
clinical presentation
• most asymptomatic
• rectal bleeding, change in bowel habits
• usually detected during routine endoscopy or family screening
pathology (types of polyps) (see table 8)
• adenomas (neoplastic)
  • 1) tubular 2) tubulovillous 3) villous
  • all premalignant
  • often carcinoma-in-situ
  • some have frank invasion into muscularis
  • hyperplastic
• asymptomatic
  • incidental finding on endoscopy
• benign
  • inflammatory (pseudopolyps) – associated with IBD, no malignant potential
• hamartomas
  • juvenile polyps
  • polyps associated with Peutz-Jeghers syndrome
• low malignant potential
  • may spontaneously regress or autoamputate
  • benign lymphoid polyps
increased risk of malignancy
• all neoplastic polyps
• size > 1 cm
• tubular (5%), tubulovillous (20%), villous (35%)
• occurrence of cancers and polyp in same segment
• malignant polyp syndromes: familial adenomatous polyps (FAP), hereditary non-polyps colorectal carcinoma (HNPPC)
**Table 8. Comparison of Colonic Polyps**

<table>
<thead>
<tr>
<th>Type</th>
<th>Occurrence</th>
<th>Location</th>
<th>% Malignant</th>
<th>Management</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tubular</td>
<td>10% of adults</td>
<td>rectosigmoid in 20%</td>
<td>7% malignant</td>
<td>endoscopic excision</td>
</tr>
<tr>
<td>Villous</td>
<td>common in elderly</td>
<td>rectosigmoid in 80%</td>
<td>33% malignant</td>
<td>surgical removal</td>
</tr>
<tr>
<td>Hamartoma</td>
<td>uncommon</td>
<td>small bowel</td>
<td>low</td>
<td>excise for bleed/obstruction</td>
</tr>
<tr>
<td>Inflammatory</td>
<td>uncommon</td>
<td>colon, rectum</td>
<td>none</td>
<td>observation</td>
</tr>
<tr>
<td>Hyperplastic</td>
<td>fairly common</td>
<td>stomach, colon, rectum</td>
<td>none</td>
<td>observation</td>
</tr>
</tbody>
</table>


**Large Intestine... Cont.**

diagnosis
- 60% within reach of flexible sigmoidoscope, or colonoscopy and biopsy

**Familial Adenomatous Polyposis (FAP)**
- Autosomal dominant (AD) inheritance, mutation in APC gene (deletion or mutation of second allele)
- Many colonic adenomas at an average age of 39
- Colon cancer in 90% if untreated by age 45

**Hereditary Non-Polyposis Colorectal Carcinoma (HNPCC)**
- AD inheritance, mutation in a DNA mismatch repair gene resulting in genomic instability and subsequent mutations
- Mutated genes: MSH2, MSH6, MLH1, PMS1, PMS2
- Early age of onset, usually involving the right colon
- Mean age of cancer presentation is 48 years

**Colorectal Carcinoma (CRC)**
- Epidemiology: Third most common carcinoma (after skin and lung)
- Mean age = 70 years

**Risk Factors**
- FAP, HNPCC
- Adenomatous polyps (especially if > 1 cm, villous, multiple)
- Age > 50 (dominant risk factor in sporadic cases)
- Family history of colon cancer
- IBD (especially UC)
- Previous colorectal cancer (also gonadal or breast)
- Diet (increased fat, decreased fiber) and smoking
- Diabetes mellitus (insulin is a growth factor for colonic mucosal cells) and acromegaly

**Diagnosis**
- By the Amsterdam Criteria
  1) At least 3 relatives with colorectal cancer, and one must be a first degree relative of the other two
  2) Two or more generations involved
  3) One case must be diagnosed before 50 years old
  4) FAP is excluded

**Treatment**
- Subtotal colectomy and ileorectal anastomosis or proctocolectomy
- +/- ileal pouch or ileostomy if many rectal polyps
pathogenesis
• most arise from adenomatous polyps
• some arise de novo
• primary: ?, diet (low fibre, high fat), genetic
• secondary: IBD (risk of cancer 1-2%/year if UC > 10 years, less risk if Crohn's)
clinical presentation: see Table 9
• usually a combination of hematochezia/melena, abdominal pain, change in bowel habits
• others: weakness, anemia weight loss
• more rare: fistula (if locally invasive), abscess,

<table>
<thead>
<tr>
<th>Table 9. Clinical Presentation of Colorectal Carcinoma</th>
</tr>
</thead>
<tbody>
<tr>
<td>Right Colon</td>
</tr>
<tr>
<td>Frequency</td>
</tr>
<tr>
<td>Pathology</td>
</tr>
<tr>
<td>Symptoms</td>
</tr>
<tr>
<td>Signs</td>
</tr>
</tbody>
</table>

spread
• direct extension
• regional nodes
• hematogenous: liver (most common distant met), lungs
• transperitoneal spread: ovary, blummers shelf
• intraluminal
diagnosis
• colonoscopy (gold standard) – visualize the entire colon, detects synchronous lesions, biopsy, polypectomy (see Colour Atlas G6, G11)
• sigmoidoscopy – not as good as colonoscopy alone
• metastatic work-up if no obvious metastases
• labs: CBC, urinalysis, liver function tests, CEA, CXR
• hemoccult
• digital rectal exam (10% are palpable)
differential diagnosis of colonic mass
• malignant
  • adenocarcinoma
  • lymphoma
  • carcinoid tumour
  • Kaposi’s sarcoma
  • prostate cancer
• benign
  • Crohn’s colitis
  • diverticulitis
  • rectal ulcer
  • lipoma, polyp
  • endometriosis
  • tuberculosis, amebiasis, cytomegalovirus (CMV), fungal infection,
extrinsic lesion
staging (see Table 10 and 11)

<table>
<thead>
<tr>
<th>Table 10. Duke-Astler-Coller Staging of Colorectal Carcinoma</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage</td>
</tr>
<tr>
<td>-----------------------------------------------</td>
</tr>
<tr>
<td>A T1-T2/N0/M0</td>
</tr>
<tr>
<td>B1 T3/N0/M0</td>
</tr>
<tr>
<td>B2 T4/N0/M0</td>
</tr>
<tr>
<td>C1 Any T/N1-N3/M0</td>
</tr>
<tr>
<td>C2 Any T/Any N/M1</td>
</tr>
<tr>
<td>D Any T/Any N/M1</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Table 11. TNM Classification System for Staging of Colorectal Carcinoma</th>
</tr>
</thead>
<tbody>
<tr>
<td>Primary Tumour (T)</td>
</tr>
<tr>
<td>-----------------------------------------------</td>
</tr>
<tr>
<td>T0 No primary tumour found</td>
</tr>
<tr>
<td>Tis Carcinoma in situ</td>
</tr>
<tr>
<td>T1 Invasion into submucosa</td>
</tr>
<tr>
<td>T2 Invasion into muscularis propria</td>
</tr>
<tr>
<td>T3 Invasion through muscularis and into serosa of named vascular trunks</td>
</tr>
<tr>
<td>T4 Invasion into adjacent structures or organs</td>
</tr>
</tbody>
</table>
treatment
• surgery
  • for all cases
  • curative: wide resection of lesion (5 cm margins) with nodes and mesentery
  • palliative: if distant spread, then local control for hemorrhage or obstruction
  • 80% of recurrences occur within 2 years of resection
  • improved survival if metastasis consists of solitary hepatic mass that is resected
• radiotherapy and chemotherapy
  • decrease recurrences only in rectal (Duke's B/C), not colon carcinoma
  • chemotherapy
  • 5-FU and levamisol or leucovorin (folinic acid) improve survival in Dukes C

screening for colorectal cancer
• at 50 years old for average risk people
• patients at increased risk should be screened earlier or more frequently depending on their individual risk profile
  • first degree relative with CRC/adenomatous polyp – Amsterdam Criteria:
    • if 3 relatives within 2 generations with at least 1 being a first degree relative:
      • start screening 10 years prior to the relative’s age with the earliest onset of carcinoma
    • family history of FAP – genetic counseling/testing, if + --> annual sigmoidoscopy
    • family history of HNPPC - genetic counseling/testing, if + --> colonoscopy yearly
    • surveillance (when polyps are found) – colonoscopy at least 3 years after initial finding
    • patients with past CRC – colonoscopy every 3–5 years
    • IBD – some recommend colonoscopy every 1–2 years after 8 years of disease

screening tools
• fecal occult blood test
  • low sensitivity and positive predictive value (PPV)
  • high rate of false positives (FP)
  • still recommended annually by the World Health Organization (WHO)
• sigmoidoscopy
  • can identify ~ 50% of lesions
  • usually followed up with a colonoscopy if lesions are detected
• double contrast barium enema
  • detects up to 50% of large (> 1cm) adenomas, 39% of polyps
  • abnormalities are followed up with colonoscopy
• colonoscopy
  • can remove lesions during procedure
  • can identify proximal lesions missed by sigmoidoscopy
• CEA: not good for screening but appropriate to monitor for recurrence (increases before clinical findings); therefore, obtain pre and post-operative levels
• annual digital rectal exam (DRE)

ANORECTUM

HEMORRHOIDS
etiology
• come from a plexus of dilated veins (cushion) arising from the superior or inferior hemorrhoidal veins (vascular and connective tissue complexes)
proposed causal factors
• increased intra-abdominal pressure
• chronic constipation
• pregnancy
• obesity
• portal hypertension
classification and management
• internal hemorrhoids = above dentate line (see Colour Atlas G18)
  • plexus of superior hemorrhoid veins --> portal circulation
  • engorged vascular cushions usually at 3, 7, 11 o’clock positions --> when patient in lithotomy position
  • painless rectal bleeding, anemia, prolapse, mucus discharge, pruritis, burning pain
• 1st degree: bleed but do not prolapse through the anus
  • treatment: high fibre bulk diet, sitz baths, steroid cream, rubber band ligation, sclerotherapy, photocoagulation
• 2nd degree: bleed but prolapse with straining, spontaneous reduction
  • treatment: rubber band ligation, photocoagulation
• 3rd degree: bleed and prolapse requiring manual reduction
  • treatment: same as 2nd degree, may require closed hemorroidectomy
• 4th degree: permanently prolapsed, cannot be manually reduced, bleeding
  • treatment: closed hemorroidectomy
external hemorrhoids = below dentate line
- plexus of inferior hemorrhoid veins → systemic circulation
- not to be confused with a perianal skin tag = residual excess skin after thrombosis of prior external hemorrhoid
- dilated venules usually mildly symptomatic unless thrombosed, in which case they are very painful
- usually present with pain after bowel movement
- treatment
  - medical therapy: dietary fiber, stool softeners, avoid prolonged straining
  - thrombosed hemorrhoids resolve within 2 weeks
  - hemorrhoidectomy when patient presents within the first 48 hours of thrombosis, otherwise treat conservatively

ANORECTUM . . . CONT.

ANAL FISSURES
tear of anal canal below dentate line (very sensitive squamous epithelium)
90% posterior midline, 10% anterior midline
if off midline: IBD, STDs, TB, leukemia or anal carcinoma
repetitive injury cycle after first tear
spasm occurs preventing edges from healing and leads to further tearing
ischemia may ensue and contribute to chronicity

Etiology
- large, hard stools and irritant diarrheal stools
- tightening of anal canal secondary to nervousness/pain
- others: habitual use of carthartics, childbirth

Acute Fissure
- very painful bright red bleeding especially after bowel movement
- treatment is conservative: stool softeners, sitz baths

Chronic Fissure
- triad: fissure, sentinel skin tags, hypertrophied papillae
- treatment = surgery
  - objective is to relieve sphincter spasm → increases blood flow and promotes healing
  - lateral subcutaneous internal sphincterotomy at 3 o’clock position
  - anal dilation (4 fingers)
- alternative treatment: topical nitro – increases local blood flow, promoting healing
- botulinum toxin – inhibits release of acetylcholine (ACh), stops sphincter spasm

ANORECTAL ABSCESS
infection in one (or more) of the anal spaces
usually a bacterial infection of a blocked anal gland at the dentate line
E. Coli, Proteus, Streplococci, Staphylococci, Bacteriodes, anaerobes
constant pain, may not be associated with bowel movement
abscess can spread vertically downward (perianal), vertically upward (supralevator) or horizontally (ischiorectal)
treatment: incision and drainage are curative in 50% of cases, 50% develop anorectal fistulas
- may also require antibiotics

Perianal Abscess
- travels distally in the intersphincteric groove
- unremmiting pain, indurated swelling

Ischiorectal Abscess
- penetrate through the external anal sphincter
- in fatty fossa, can spread readily: necrotizing fasciitis, Fournier’s gangrene
- pain, fever and leukocytosis prior to red, fluctuant mass

Intersphincteric
- between the internal and external sphincters
- fluctuant mass palpated in DRE

Supralevator Abscess
- difficult to diagnose, rectal mass and swelling detectable with exam under anesthesia

FISTULA IN ANO
a connection between two epithelial lined surfaces, one must be the rectum or anus
an inflammatory tract with internal os at dentate line, external os on skin
same perirectal process as anal abscess therefore usually associated with abscess
other causes: post-op, trauma, arising from anal fissure, malignancy, radiation proctitis
intermittent or constant purulent discharge from para-anal opening, pain palpable cord-like tract
treatment
• identify internal opening
• Goodsall’s rule (see Figure 9) – a fistula with an external opening anterior to the transverse anal line will have its internal opening at relatively the same position (e.g. external opening at 2 o’clock = internal opening at 2 o’clock) whereas all external openings posterior to the line will tend to have their internal openings in the midline
• fistulous tract identification (probing or fistulography) under anesthesia
• unroof tract from external to internal opening, allow drainage
• seton (thick suture) can be placed through tract
  1) promotes drainage
  2) promotes fibrosis and decreases incidence of incontinence
  3) delineates anatomy
• post-op: sitz baths, irrigation + packing to ensure healing proceeds inside to outside complications
  • recurrence, fecal incontinence

![Figure 9. Goodsall’s Rule](Illustration by M. Gail Rudakewich)

PILOMODIAL DISEASE
acute abscess or chronic draining sinus in sacrococcygeal area
develops secondary to obstruction of the hair follicles in this area ——>
leads to formation of cysts, sinuses or abscesses
usually asymptomatic until acutely infected
treatment
• acute abscess - incision and drainage
• chronic disease - pilonidal cystotomy or excision of sinus tract and cyst
  +/- marsupialization

RECTAL PROLAPSE
protrusion of full thickness of rectum through anus
initially reduces spontaneously until continuously prolapsed (must be differentiated from hemorrhoidal prolapse)
increased incidence in gynecological surgeries, chronic neurologic/psychiatric disorders affecting motility results in fecal and flatus incontinence secondary to dilated and weakened sphincter
occurs in extremes of age
• < 5 years old spontaneously resolve with conservative treatment (stool softeners)
• > 40 years old usually require surgical treatment: anchoring rectum to sacrum (e.g. Ripstein procedure), excision of redundant rectum followed by colon anastomosis to lower rectum

ANAL NEOPLASMS
epidermoid carcinoma of anal canal (above dentate line)
• most common tumour of anal canal (75%)
• squamous cell or transitional cell
• anus is prone to human papilloma virus (HPV) infection, therefore at risk for anal squamous intraepithelial lesions (ASIL), high grade squamous intraepithelial lesion (HSIL) and low grade squamous intraepithelial lesion (LSIL) terminology used
• presents with rectal pain, bleeding, mass
• treatment of choice is chemotherapy, radiation +/- surgery with 80% 5 year survival
• malignant melanoma of anal margin
• 3rd most common site after skin, eyes
• aggressive, distant metastases are common at time of diagnosis
• early radical surgery is treatment of choice
• < 15% 5 year survival
LIVER CYSTS
normally asymptomatic
if large —> upper abdominal discomfort/mass

Parasitic Liver Cysts
Cystic Echinococcosis
• infection with parasite Echinococcus granulosus
• endemic Southern Europe, Middle East, Australia, South America
• transmitted by fecal/oral route in infected carnivores
• asymptomatic mass (most often) or chronic pain, hepatomegaly
• rupture can cause biliary colic, jaundice or anaphylactic reaction
• diagnosis
  • detection of anti-Echinococcus Ab (IgG) using ELISA or IHA
  • presence of mass, often calcified, on U/S or CT
  • DO NOT do needle biopsy – seeding of abdominal cavity or anaphylaxis
• treatment
  • medical – albendazole (anti-helmintic) – cure in up to 30%
  • surgical – conservative – open endocystectomy +/- omentoplasty
    radical – partial hepatectomy or total pericystectomy

Non-Parasitic Liver Cysts
simple cyst – no clinical significance
multicystic (50% have polycystic kidney) – hepatocellular function is usually well preserved
choledochal cyst
• congenital malformations of pancreaticobiliary tree
• 4 types with the extreme form called Caroli’s disease
  (multiple cystic dilations in intrahepatic ducts)
• clinical features - recurrent abdominal pain, intermittent jaundice, RUQ mass
• diagnosis - U/S, transhepatic cholangiography, LFTs
• treatment is surgical (extent of resection depends on type of cyst) -
  liver transplant indicated if cyst involvement of intrahepatic bile ducts (Caroli’s disease)
• complications of chronic disease are biliary cirrhosis, portal hypertension, bile duct carcinoma
neoplastic
• cystadenoma: premalignant, usually require resection
• cystadenocarcinoma

LIVER ABSCESSES
Bacterial Liver Abscess
most common hepatic abscess in Western world
usually secondary to suppurrative process in abdomen
• cholangitis, appendicitis, diverticulitis, generalized sepsis, also seeding from endocarditis
organism related to primary source
• abdominal - Gram –ve rods (E. coli), anaerobes (Bacteroides), Enterococcus
• extra-abdominal - Gram +ve organisms (e.g. from bacterial endocarditis, pneumonitis)
usually present with fever, malaise, chills, anorexia, weight loss, abdominal pain or nausea with right upper quadrant (RUQ) tenderness, hepatomegaly, jaundice, and pleural dullness to percussion
lab - leukocytosis, anemia, elevated LFTs
diagnosis
• U/S, CXR (R basilar atelectasis/effusion), CT, serum antibody titre,
  percutaneous aspiration and drainage
• more common in right lobe
treatment
• treat underlying cause
• surgical drainage and IV antibiotics
overall mortality 15% - higher rate if delay in diagnosis, multiple abscesses, malnutrition

Amoebic Abscess
infection caused by protozoan Entamoeba histolytica
transmission usually occurs by food borne exposure
common in South and Central America
associated with fever, leukocytosis, diarrhea, RUQ pain, hepatomegaly
often a single large cavity in the right lobe (90%), found on U/S or CT
treatment: parenteral antibiotics (metronidazole) + amebicide (i.e. iodoquinol), aspiration of abscess if large; surgical drainage indicated if complications arise (rupture)
NEOPLASMS

Benign Liver Neoplasms

hemangioma (cavernous)
- most common benign hepatic tumour; results from malformation of angioblastic fetal tissue
- female:male = 6:1, steroid therapy, estrogen therapy and pregnancy may promote tumour growth
- most are small and do not cause symptoms, larger tumours can produce significant pain or compress nearby structures, or shock if ruptures
- usually no treatment, unless tumour bleeds or is symptomatic (excision by lobectomy or enucleation)
- arteriography is diagnostic, but red blood cell scan as useful and cheaper
- do not biopsy → massive hemorrhage

adenoma
- benign glandular epithelial tumour
- young women (30 – 50 years old) on oral contraceptive (OCP) for many years
- 25% present with RUQ pain or mass
- malignant potential
- diagnosis: mass on U/S or CT
- treatment
  - stop OCP or anabolic steroids
  - excise especially if large (>5 cm) due to increased risk of malignancy and spontaneous rupture/hemorrhage

central nodular hyperplasia
- thought to be due to local ischemia and tissue regeneration
- female: male = 2:1 (in age 40 on average)
- rarely grow or bleed, no malignant potential
- "central stellate scar" on CT scan, Tc99 scan is helpful
- treatment: resect only if symptomatic

Malignant Liver Neoplasms

primary
- usually hepatocellular adenocarcinoma, others include cholangiocarcinoma, angiosarcoma, hepatoblastoma, hemangioendothelioma
- uncommon in North America, but 20-25% of all carcinomas in the Orient and Africa
- male:female═2:1
- risk factors
  - chronic hepatitis B and C infections
  - cirrhosis (especially macronodular)
  - OCP’s - 3x increased risk
  - steroids
  - smoking, alcohol
  - chemical carcinogens (aflatoxin, vinyl chloride - associated with angiosarcoma)
  - parasite infection (Clonorchis sinensis associated with cholangiocarcinoma)
  - hemochromatosis, (α1-antitrypsin deficiency)
- signs and symptoms
  - RUQ discomfort, right shoulder pain
  - jaundice, weakness, weight loss, fever
  - hepatomegaly, bruise, rub
  - 10-15% ascites with blood (sudden intra-abdominal hemorrhage)
  - paraneoplastic syndromes – i.e. Cushing’s syndrome
- diagnosis
  - elevated alkaline phosphatase (ALKP), bilirubin, and alpha-feto-protein (AFP) (80% of patients)
  - imaging: U/S (best), liver scan, CT, MRI, angiography
  - biopsy
- treatment
  - cirrhosis relative contraindication to tumour resection due to decreased hepatic reserve/regenerative capacity
  - surgery - 10% of patients have resectable tumours
  - liver transplant (not if Hep B) and no extrahepatic tumour
  - non-surgical - chemotherapy – adriamycin has shown promise, ethanol injection, hepatic artery ligation, arterial embolization, poor response to radiation
- prognosis
  - 70% have mets to nodes and lung
  - 5 year survival of all patients - 5%
  - 3 month survival if no treatment
  - 5 year survival of patients undergoing complete resection - 11-40%

secondary
- most common hepatic malignancy
- bronchogenic (most common), GI, pancreas, breast, ovary, uterus, kidney, gallbladder, prostate
- treatment
  - hepatic resection if control of primary is possible, no extrahepatic mets and < 4 lesions (prognosis same if mets are multilobar compared with all in one lobe)
  - possibly chemotherapy
  - 5 year overall survival 20-50% with resection of colorectal mets (overall survival with colorectal mestastases to liver approximately 6-7 months)
PORTAL HYPERTENSION (see Gastroenterology Chapter)

Table 12. Child's Classification for Determining Operative Risk for Shunting Procedure in Portal Hypertension

<table>
<thead>
<tr>
<th></th>
<th>A</th>
<th>B</th>
<th>C</th>
</tr>
</thead>
<tbody>
<tr>
<td>Serum bilirubin (mg/dL)</td>
<td>&lt; 2</td>
<td>2-3</td>
<td>&gt; 3</td>
</tr>
<tr>
<td>Serum albumin (g/dL)</td>
<td>&gt; 3.5</td>
<td>3-3.5</td>
<td>&lt; 3</td>
</tr>
<tr>
<td>Presence of ascites</td>
<td>absent</td>
<td>controllable</td>
<td>refractory</td>
</tr>
<tr>
<td>Encephalopathy</td>
<td>absent</td>
<td>minimal</td>
<td>severe</td>
</tr>
<tr>
<td>Malnutrition</td>
<td>absent</td>
<td>mild</td>
<td>severe</td>
</tr>
<tr>
<td>Operative mortality</td>
<td>2%</td>
<td>10%</td>
<td>50%</td>
</tr>
</tbody>
</table>

Management of Bleeding Varices in Portal Hypertension

- acute bleeding
  - volume resuscitation (predominantly blood products)
  - somatostatin (octreotide)
  - endoscopic therapy – sclerotherapy or banding (90% effective)
  - balloon tamponade (Blakemore tube)
    - 12-24 hours: 75% effective initially (20-50% rebleed rate)
    - risk of aspiration, ulceration, asphyxiation, or rupture; therefore oro/nasotracheal intubation indicated
  - transjugular intrahepatic porto-systemic shunt (TIPS)
    - prevention of recurrence (70% after initial bleed)
  - endoscopic therapy – sclerotherapy or banding
  - surgical shunts (see Surgical Shunt section)
  - TIPS

Shunting Procedures to Decrease Portal Venous Pressure

- portal decompression
  - operative mortality: Child's A 0-5%, B 5-15%, C 20-50%
  - portocaval
    - eliminates portal venous flow - direct anastomosis between inferior vena cava (IVC) and portal vein
    - used in acute bleeding/ascites
    - very effective (> 90%) in controlling bleed
    - risk of hepatic encephalopathy high (11-38%); hepatic failure (13-18%) due to low portal flow from shunt
  - distal spleno-renal (Warren)
    - anastomosis of splenic vein to left renal vein
    - procedure of choice for elective shunt surgery
    - not used in patients with ascites
    - decreased rate of hepatic encephalopathy and failure as portal flow and liver detoxification partially intact
  - transjugular intravascular portasystemic shunt (TIPS)
    - new technique performed by radiologists
    - creates a shunt between portal and hepatic vein via a catheter placed in the liver
    - can be used to stop acute bleeding or prevent rebleeding
    - shunt usually remains open up to one year

Ascites

- management
  - portocaval shunt (side to side)
  - peritoneovenous shunt: drainage of intraperitoneal fluid to vascular compartment (i.e. Leveen shunt)
  - indications: failure of medical treatment, encephalopathy, azotemia

Hypersplenism

- treated conservatively
  - splenectomy or Warren shunt if severe or development of splenic vein thrombosis
LIVER TRANSPLANTATION

indications
- end-stage liver disease with life expectancy of less than one year
  and for whom no other therapy is appropriate
- progressive jaundice, refractory ascites, spontaneous hepatic encephalopathy, recurrent sepsis,
  recurrent variceal hemorrhage, coagulopathy, severe fatigue

Candidacy for Transplantation
parenchymal disease
- post-necrotic cirrhosis (chronic active hepatitis)
- alcoholic cirrhosis
- acute liver failure
- Budd-Chiari syndrome
- congenital hepatic fibrosis
- cystic fibrosis (CF)
cholestatic disease
- biliary atresia
- primary biliary cirrhosis (PBC)
- sclerosing cholangitis
inborn errors of metabolism
- alpha-1-anti-trypsin deficiency
- Wilson's disease
tumours
- primary malignant
- benign

Post-op Complications
primary non-function – urgent re-transplantation is indicated
acute and chronic rejection
hepatic artery thrombosis
portal vein thrombosis
biliary complications – fever, increasing bilirubin and ALP
venal caval obstruction
recurrence of hepatitis B

Survival
- patient survival at one year – 81%
- graft survival at one year – 76%, 70% at three years

BILIARY TRACT

CHOLELITHIASIS

Pathogenesis
imbalance of cholesterol and its solubilizing agents, bile salts and lecithin concentrations
if hepatic cholesterol secretion is excessive then bile salts and lecithin are “overloaded”,
supersaturated cholesterol precipitates and can form gallstones

Types of Stones
cholesterol (80%) = mixed (> 70% cholesterol by weight), radiolucent
- risk factors
  - female, fat, fertile, forties
  - North American First Nations peoples have highest incidence
  - diabetes mellitus (DM), pancreatitis
  - malabsorption, terminal ileal resection or disease
    (e.g. inflammatory bowel disease)
pigment stones (20%), may be radio-opaque
  - smooth green/black to brown; composed of unconjugated bilirubin, calcium, bile acids
  - black pigment stones
    - associated with cirrhosis, chronic hemolytic states
  - calcium bilirubinate stones
    - associated with bile stasis, (biliary strictures, dilatation and biliary infection (Clonorchis sinensis))

Natural History
- 80% are asymptomatic
- 18% develop symptoms over 15 years
BILIARY TRACT... CONT.

Clinical Presentation (in severity of increasing order)
- asymptomatic stones
  - most asymptomatic gallstones do NOT require treatment
  - consider operating if calcified "porcelain" gallbladder, sickle cell disease (15-20% associated cancer), DM, history of biliary pancreatitis
- biliary colic
- cholecystitis - acute and chronic
- complications of cholecystitis
- choledocholithiasis (CBD stones)
- cholangitis
- biliary induced pancreatitis
- biliary induced ileus

BILIARY COLIC (or CHRONIC CHOLECYSTITIS)
- many patients with acute cholecystitis have a history of episodic biliary colic
- mechanism: gallstone temporarily impacted in cystic duct, no infection
- signs and symptoms
  - steady pain (not colic) in epigastrium or RUQ for minutes to hours
  - frequently occurs at night or after fatty meal
  - can radiate to right shoulder or scapula
  - associated nausea/vomiting, burping, flatus, heartburn
  - no peritoneal findings
  - no systemic signs
- differential diagnosis - pancreatitis, peptic ulcer disease (PUD), hiatus hernia with reflux, gastritis, angina pectoris, irritable bowel syndrome (IBS) (see Acute Abdomen section)
- diagnostic investigation
  - normal blood work
  - U/S shows gallstones in gallbladder
- treatment
  - elective cholecystectomy (95% success)

ACUTE CHOLECYSTITIS
- mechanism
  - inflammation of gallbladder resulting from sustained obstruction of cystic duct by gallstone (80%)
  - no cholelithiasis in 20% (calculous - see below)
- signs and symptoms
  - often have history of biliary colic
  - severe constant epigastric or RUQ pain
  - anorexia, nausea and vomiting are common
  - systemic signs – low grade fever (<38.5 °C), tachycardia
  - focal peritoneal findings - Murphy's sign (sudden cessation of inspiration with deep RUQ palpation)
  - palpable gallbladder in one third of patients
- differential diagnosis - perforated or penetrating peptic ulcer, MI, pancreatitis, hiatus hernia, right lower lobe pneumonia, appendicitis, hepatitis, herpes zoster
- diagnostic investigation
  - elevated WBC, left shift
  - mildly elevated bilirubin, ALP
  - sometimes slight elevation AST, ALT
  - U/S shows distended, edematous gallbladder, pericholecystic fluid, large stone stuck in gallbladder neck, sonographic Murphy's sign (maximum tenderness elicited by probe over site of gallbladder)
- complications
  - hydrops: mucus accumulation in gallbladder; may lead to necrosis
  - gangrene and perforation: may cause localized abscess or generalized peritonitis (can occur 3 days after onset)
  - empyema of gallbladder (suppurative cholangitis) – gallbladder contains frank pus
  - cholecystoenteric fistula from repeated attacks of cholecystitis
  - gallstone ileus (see below)
  - choledocholithiasis - 15% of patients with gallstones
  - emphysematous gallbladder
- treatment
  - admit, hydrate, NPO, NG tube, analgesics once diagnosis is made, antibiotics if high risk (elderly, immunosuppressed)
  - E. coli, Klebsiella, Enterobacteriaceae and Enterobacter account for > 80% of infections, 1st and 2nd generation cephalosporins are first choice antibiotic coverage
  - lack of improvement with conservative treatment —> operate within 24-48 hours (cholecystectomy)
  - earlier O.R. if high risk (DM, steroids) or severe disease
  - cholecystostomy tube if general anesthetic contraindicated

COMPLICATIONS OF CHOLECYSTECTOMY
- general
  - anesthetic risk
  - post-op complications (see Surgical Complications section)
- specific
  - bile duct injury (0.2-1%)
  - correct with Roux-en-Y choledochojejunostomy (see Figure 12)
ACALCULOUS CHOLECYSTITIS
acute or chronic cholecystitis in the absence of stones (5%)
typically due to gallbladder stasis
usually diabetic, immunosuppressed, post-op or in intensive care unit (ICU)
etiology
• dehydration, prolonged fasting, TPN
• systemic disease (e.g. MOSF)
• generalized sepsis, trauma
• kinking or fibrosis of the gallbladder
• thrombosis of the cystic artery
• spincter spasm with obstruction of the biliary and pancreatic ducts
• collagen vascular disease, DM, immunosuppressed
treatment
• cholecystectomy or cholecystostomy

CHOLEDOCHOLITHIASIS
stones in common bile duct (CBD)
signs and symptoms
• 50% asymptomatic
• often have history of biliary colic
• tenderness in RUQ or epigastrium
• pale stool, dark urine
• intermittent chills, fever or fluctuating jaundice
• complications include biliary colic, cholangitis, pancreatitis, late benign biliary stricture, and biliary cirrhosis
diagnostic investigations
• increased bilirubin (> 10), ALP, 5′-NTD
• leukocytosis often > 20 x 10^9/L
• U/S – intra-extra-hepatic duct dilatation, does not detect CBD stones
• endoscopic retrograde cholangiopancreatography (ERCP) (if no previous cholecystectomy)
• otherwise percutaneous transhepatic cholangiography (PTC) appropriate
• intra-operative cholangiography
treatment
• antibiotics, NG tube (with abdo distention or vomiting), IV hydration
• if no improvement in 2-4 days then ERCP/PTC+sphincterotomy or surgery with CBD exploration and laparoscopic cholecystectomy

ACUTE CHOLANGITIS
mechanism: obstruction of CBD leading to biliary stasis, bacterial overgrowth, suppuration, and biliary sepsis - life threatening
etiology
• choledocholithiasis (60%)
• post-operative stricture
• pancreatic or biliary neoplasms
organisms: E. coli, Klebsiella, Pseudomonas, Enterococci, B. fragilis, Proteus
signs and symptoms
• Charcot’s triad (50-70% of cases): fever, jaundice, RUQ pain
• Reynold’s pentad: Charcot’s triad + mental confusion + hypotension leading to renal failure
diagnostic investigations
• elevated WBC
• elevated liver enzymes (ALP mild increase AST,ALT) and conjugated bilirubin
• U/S shows stones in gallbladder +/- stones seen in bile ducts +/- dilated extrahepatic or intrahepatic bile ducts
treatment
• immediate goal is to decompress the biliary tree
• initially hydration, electrolyte correction, broad-spectrum antibiotics
• urgent ERCP - diagnostic and therapeutic with papillotomy to remove stones
• if ERCP unavailable or unsuccessful, then PTC
• if ERCP, PTC unavailable, surgery to decompress CBD --> T-tube
prognosis
• suppurative cholangitis – mortality rate = 50%
BILIARY TRACT . . . CONT.

GALLSTONE PANCREATITIS
mechanism: gallstone impacted in common pancreatic duct
signs and symptoms- epigastric, back pain
diagnostic investigation
• high amylase, lipase, high liver enzymes
• most cases mild Ranson’s criteria
• U/S may show multiple stones (may have passed spontaneously), edematous pancreas
• CT if severe to evaluate for complications
treatment
• supportive
• cholecystectomy during same admission after acute attack subsided (23-60% recurrence if no O.R.)

GALLSTONE ILEUS
mechanism - cholecystoenteric fistula (usually duodenal) with large gallstone
impacting most commonly at the ileocecal valve
not an ileus, but a true partial or complete small bowel obstruction
clinical presentation
• crampy abdominal pain, nausea, vomiting
diagnostic investigation
• 3 views abdomen shows dilated small intestine, gallstone in RLQ and air in biliary tree (15%)
• upper GI series if unclear
treatment
• hydrate, operate to remove stone; (enterotomy) usually don’t have to remove gallbladder
(50% cholecystectomy)
• fistula usually closes spontaneously
• mortality 10-15%

CARCINOMA OF THE BILE DUCT
majority adenocarcinoma
2% of cancer deaths
associations
• age 50-70 years
• increased incidence in patients with diffuse gallbladder wall calcification, cholecystoenteric fistula and adenoma
• female/male = 2:1
clinical presentation
• usually found incidentally at time of elective cholecystectomy
• local: RUQ pain, palpable painless mass (if tumour in CBD),
• systemic: unremitting jaundice, pruritus, weight loss, anorexia
diagnostic investigations
• Klatskin tumour (at common hepatic duct bifurcation) causes increased ALP, bilirubin, but normal AST
• U/S and CT (dilated bile ducts), ERCP and PTC (depict tumour)
treatment
• in situ lesions require cholecystectomy only
• for advanced tumours, cholecystectomy + wedge resection of liver and regional lymphadenectomy
• +/- stents for palliation
• radiation or Whipple’s if tumour at low end of CBD
prognosis
• spread: growth into portal vein or hepatic artery, liver, hilar nodes
• 10-15% 5 year survival
• death results from progressive biliary cirrhosis, persistent intrahepatic infection and abscess formation, or sepsis

DIAGNOSTIC EVALUATION OF BILIARY TREE
U/S is diagnostic procedure of choice
oral cholecystography
• opaque drug taken night before, look for filling defect (stones)
• failure of gallbladder to opacify indicative of complete obstruction by stone or unable to concentrate because of inflammation
HIDA scan
• radioisotope technetium excreted in high concentrations in bile
• highly suggestive of acute cholecystitis when gallbladder not visualized due to cystic duct obstruction 4 hours after injection
• reliable with a bilirubin up to 20
ERCP (endoscopic retrograde cholangiopancreatography)
• visualization of upper GI tract, ampullary region, biliary and pancreatic ducts
• preferred method to demonstrate CBD stones and periampullary region
• complications – traumatic pancreatitis (1-2%), pancreatic or biliary sepsis
PTC (percutaneous transhepatic cholangiography)
• injection of contrast via needle passed through hepatic parenchyma
• useful for proximal bile duct lesions or when ERCP fails
• antibiotic premedication always,
• contraindications – coagulopathy, ascites, peri- or intrahepatic sepsis, disease of right lower lung or pleura
• complications – bile peritonitis, bilothorax, pneumothorax, sepsis, hemobilia
IOC (Intra-operative Cholangiography)

- indications – define anatomy, obstructive jaundice, history of biliary pancreatitis, small stones in gall bladder with a wide cystic duct (>15 mm), single faceted stone in gall bladder, bilirubin > 8 mg/dl

**JAUNDICE**

![Jaundice Diagram](image)

**PANCREAS**

**ACUTE PANCREATITIS** (see Gastroenterology Chapter)
- usually no surgical management in uncomplicated acute pancreatitis
- surgical indications in acute pancreatitis
  - secondary pancreatic infections - abscess, infected pseudocysts/necrosis
  - gallstone-associated pancreatitis
  - uncertainty of clinical diagnosis
  - worsening clinical condition despite optimal supportive care
- complications
  - pseudocyst – most common complication
    - collection of pancreatic secretions in a cyst lacking true epithelium
    - risk of rupture, hemorrhage and infection (rare)
    - 2-3 weeks post-attack: persistent pain, fever, ileus, mass, nausea/vomiting, early satiety, persistent elevation of amylase
    - endocrine and exocrine abnormalities
    - diagnosis - clinical, U/S, CT
    - treatment - 40% resolve spontaneously within 6-12 weeks, supportive management for 6-8 weeks until thick, fibrous wall has formed, then internal (preferred) or external drainage (latter if infected or sick patient)
    - biopsy pseudocyst wall to rule out malignancy (cystadenocarcinoma)
• abscess (5%)
  • 1-4 weeks post-attack: fever, toxic, abdominal pain, distention
  • diagnosis: increased amylase, increased AST/ALT (50%), elevated WBC, CT (fluid and gas)
  • high mortality - requires extensive surgical debridement and broad-spectrum antibiotics
• ascites
  • secondary to pseudocyst disruption (common) or direct pancreatic duct disruption
  • diagnose by paracentesis: high amylase, high protein
  • treatment: NPO, TPN 2-3 weeks, somatostatin
  • ERCP if not resolved to determine anatomy; then surgical internal drainage or distal resection
• necrosis
  • diagnosis by CT or elevated serum acute phase reactants
  • treatment: if <30% will spontaneously resolve, if >30% or infected - debridement
• hemorrhage
  • due to erosion of arterial pseudoaneurysm secondary to pseudocyst, abscess, or necrotizing pancreatitis
  • clinical presentation: increased abdominal mass, abdominal pain, hypotension, falling hematocrit
  • diagnosis: angiography
  • treatment: immediate surgery
• sepsis
• MOSF

prognosis of all complications
• 80% improve rapidly
• 20% have at least one complication from which 1/3 die

CHRONIC PANCREATITIS (see Gastroenterology Chapter) (see Colour Atlas G7)

surgical treatment
• indications for surgical treatment: debilitating abdominal pain, CBD obstruction, duodenal obstruction, persistent pseudocyst, pancreatic fistula, pancreatic ascites, variceal hemorrhage secondary to splenic vein obstruction, rule out pancreatic cancer
• CT and ERCP are mandatory prior to consideration of surgical management
• pancreatic duct drainage
  • Puestow (most common) and Duval procedure
  • 80-90% have pain relief, but 5 years post-op only 50-60% remain pain-free
• pancreatic resection - Whipple (pancreaticoduodenectomy) 80% have pain relief
• pancreatectomy - use when no dilated ducts, amount of resection depends on disease focus (i.e. limited vs. subtotal vs. total pancreatectomy)

PANCREATIC CANCER

epidemiology
• fifth most common cause of cancer death
• African descent at increased risk
• male:female = 1.7:1
• age (average 50-70)

risk factors
• increased age
• smoking - 2-5x increased risk, most clearly established risk factor
• high fat/low fibre diets
• chronic pancreatitis
• diabetes
• heavy alcohol use
• chemical: betanaphthylamine, benzidine

clinical presentation is related to location of tumour
• head of the pancreas (70%)
  • weight loss, obstructive jaundice, abdominal pain – dull ache in midepigastrium, progressive, often worse at night, may radiate to back
  • painless jaundice occurs more often with ampullary or primary bile duct tumours, and is not common in pancreatic cancers
  • palpable tumour mass = incurable
• carcinoma of body or tail of pancreas (30%)
  • tends to present later and usually inoperable
  • < 10% jaundiced
  • sudden onset diabetes mellitus
  • weight loss, vague midepigastric pain
• surgical dictum: vague abdominal pain with weight loss +/- jaundice in a patient > 50 years old is pancreatic cancer until proven otherwise

diagnosis
• serum chemistry non-specific: elevated ALP and bilirubin (>18)
• evidence of obstruction: U/S, CT – evaluation of metastasis
pathology
- ductal adenocarcinoma - most common type (75-80%)
- giant cell carcinoma (4%)
- adenosquamous carcinoma (3%)
- other: mucinous, cystadenocarcinoma, acinar cell carcinoma

spread
- early to local lymph nodes and liver

treatment
- operable (i.e. no metastases outside abdomen, liver, or peritoneal structures, and no involvement of hepatic artery, superior mesenteric artery, portal vein at body of pancreas)
  - 20% of head of pancreas cancers can be resected
  - Whipple's procedure (pancreatoduodenectomy) for cure - 5% mortality (see Figure 14)
  - distal pancreatectomy +/- splenectomy, lymphadenectomy if carcinoma of midbody and tail of pancreas
- inoperable (i.e. involves liver, vasculature or regional nodes)
  - most body and tail cancers not resectable
  - relieve biliary/duodenal obstruction with endoscopic stenting or double bypass procedure: cholecchoenterostomy, gastroenterostomy
- palliative surgery
  - goal is to alleviate pain, and biliary and duodenal obstruction
  - combination chemotherapy/radiotherapy for palliation, increased medical survival post-surgery

prognosis
- average survival - 7 months
- 5 year survival is 10%
- following Whipple's procedure, mean survival - 18 months
- most important prognostic indicator is lymph node status

Figure 14. Whipple Procedure

Illustration by Myra Rudakewich

SPLEEN

HYPERSPLENISM (see Hematology Chapter)
exaggeration of normal splenic functions such as removal and destruction of aged/defective RBCs, sequestration of normal cells and production of immunoglobulins

etiology
- primary hypersplenism
  - rare
  - diagnosis of exclusion
- secondary hypersplenism
  - congestion (most common) secondary to portal hypertension
  - neoplasia - lymphoma, leukemia, myeloid metaplasia
  - infections - Epstein Barr virus (EBV), TB
  - inflammatory diseases - sarcoid, rheumatoid arthritis (RA)
  - hematologic - spherocytosis, G6PD deficiency
  - storage diseases - Gaucher's disease, amyloid

clinical presentation
- +/- LUQ fullness, discomfort, spontaneous rupture
- anemia, leukopenia, thrombocytopenia

diagnosis
- CBC, differential and smear, hemoglobin (Hb) electrophoresis
- bone marrow biopsy
- splenic function tests: radiolabeled RBC/platelets (measure rate of disappearance)

treatment
- splenectomy
  - reduces number of transfusions, number of infections, prevents hemorrhage, and decreases pain
Spleen...cont.

Splenectomy
indications
• always
  • primary splenic tumour (rare)
  • hereditary spherocytosis
• usually
  • primary hypersplenism
  • chronic immune thrombocytopenia purpura
  • splenic vein thrombosis causing esophageal varices
  • splenic abscess
• sometimes
  • splenic injury (most common reason for splenectomy)

complications
• short term
  • atelectasis of the left lower lung
  • injury to surrounding structures e.g. gastric wall, tail of pancreas
  • post-op hemorrhage
  • post-op thrombocytosis, leukocytosis
  • subphrenic abscess
• long term
  • post-splenectomy sepsis (encapsulated organisms)
  • 4% of splenectomized patients
  • 50% fatality

prophylaxis
• vaccinations: pneumococcal, *Haemophilus influenzae*
• penicillin for children < 18 years old

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Splenectomy
management – hemostatic control, splenorrhaphy, partial splenectomy or total splenectomy as indicated in stable patients with lower grade injury - extended bedrest with serial hematocrit levels and close monitoring

Breast

Evaluation of a breast mass

History
• mass – when and how first noticed, tenderness, change in size over time and with menstruation
• nipple discharge
• gynecologic history
  • past medical history – benign breast disease, breast cancer, radiation therapy to breast of axilla
  • past surgical history – breast biopsy, lumpectomy, mastectomy, hysterectomy, oophorectomy
  • family history – especially in first degree relatives
  • constitutional features – anorexia, weight loss, respiratory symptoms, chest pain, bony pain

Physical exam – inspection, palpation
• carcinoma is usually firm, nontender, poorly circumscribed and immobile, and other signs of malignancy
• skin changes: edema, dimpling, retraction, redness, ulceration
• nipple: bloody discharge, crusting, ulceration, inversion
• prominent veins, palpable axillary/supraclavicular lymph nodes, arm edema

Radiographic Studies
• indications for mammography
• screening – every 1-2 years for women ages 50-69, women age 40-49 with average risk of developing breast cancer no evidence to include or exclude (Canadian Task Force on Preventive Health Care)
• metastatic adenocarcinoma of unknown primary
• nipple discharge without palpable mass
• mammogram findings indicative of malignancy
  • stellate appearance and spiculated border - pathognomonic of breast cancer
  • microcalcifications, ill-defined lesion border
  • lobulation, architectural distortion
  • increased vascularity, interval mammographic changes
  • normal mammogram does not rule out suspicion of cancer, based on clinical findings
  • U/S – differentiate between cystic and solid mass

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Procedures
- needle aspiration – for palpable cystic lesions, send fluid for cytology if serosanguinous or bloody
- fine needle aspiration (FNA) – for palpable solid masses
- U/S guided/mammotome core biopsy
- excisional biopsy – definitive method for tissue diagnosis

FIBROCYSTIC DISEASE
benign breast condition consisting of fibrous and cystic changes in breast
age 30-50 years
clinical features – breast pain, swelling with focal areas of nodularity or cysts,
frequently bilateral, varies with menstrual cycle
treatment – if no dominant mass, observe to ensure no mass dominates
  - for a dominant mass, FNA
  - if > 40 years, mammography every 3 years
  - avoid xanthine-containing products (coffee, tea, chocolate, cola drinks) and nicotine
  - for severe symptoms – danazol (2-3 months), or tamoxifen (4-6 weeks)

FIBROADENOMA
most common benign breast tumour in women under age 30
no malignant potential
clinical features – smooth, rubbery, discrete, well circumscribed nodule, non-tender,
mobile, hormonally dependant
management – usually excised to confirm diagnosis

FAT NECROSIS
due to trauma (although positive history in only 50%)
clinical features – firm, ill-defined mass with skin or nipple retraction, +/- tenderness
management – will regress spontaneously but complete excisional biopsy the safest approach to rule out carcinoma

PAPILLOMA
solitary intraductal benign polyp
most common cause of bloody nipple discharge
management – excision of involved duct

DIFFERENTIAL DIAGNOSIS OF NIPPLE DISCHARGE
- bloody – papilloma, papillary/intraductal carcinoma, Paget’s, fibrocystic change
- serous – duct hyperplasia, pregnancy, OCP, menses, cancer
- green/brown – mamillary duct ectasia, fibrocystic change
- purulent – superficial or central abscess
- milky – postlactation, OCP, prolactinoma
investigate with galactogram to identify lesion in ducts

MASTITIS
nursing mothers: sporadic or recurrent
etiologic agent: S. aureus, S. epidermidis
unilateral localized pain, tenderness and erythema
sporadic: acinar and duct systems uninvolved, therefore, infant should continue being nursed
recurrent: due to regurgitation of milk back into ducts, therefore, discontinue nursing and suppress lactation
management – application of heat or ice packs, antibiotic therapy (cloxacillin 250-500 mg QID),
if fluctuant then incise and drain
must rule out inflammatory carcinoma if suspicious

MALE BREAST LUMPS
gynecomastia
- breast hypertrophy in males
etiology
  - physiologic – exposure to maternal estrogens in newborns, in men > 50, due
to male menopause, with relative estrogen increase – breast tissue is
  enlarged, firm, tender, will regress spontaneously in 6-12 months
  - drug-induced – estrogens, digoxins, thiazide, phenytoin, theophylline,
diazepam, some antihypertensives, tricyclics, antineoplastic drugs – discontinue offending drug
  - pathologic – cirrhosis, renal failure, malnutrition, hyperthyroidism, adrenal dysfunction,
testicular tumours, hypogonadism
  - any dominant mass should be biopsied to rule out carcinoma
breast carcinoma
  - usually > 50 years
  - hard, painless lump +/- nipple retraction, discharge, ulceration
  - often metastatic at time of diagnosis, therefore, poor prognosis
BREAST CANCER

epidemiology
• most common cancer in women (excluding skin)
• second leading cause of cancer mortality in women
• most common cause of death in 5th decade
• lifetime risk of 1/9

risk factors
• age - 80% > 40
• sex - 99% female
• 1st degree relative with breast cancer
• risk increased further if relative was premenopausal
• geographic - highest national mortality in England and Wales, lowest in Japan
• nulliparity
• late age at first pregnancy
• menarche < 12; menopause > 55
• obesity
• excessive alcohol intake, high fat diet
• certain forms of fibrocystic change
• prior history of breast cancer
• history of low-dose irradiation
• prior breast biopsy regardless of pathology
• OCP/estrogen replacement may increase risk

staging
• clinical: assess tumour size, nodal involvement, and metastasis
  • tumour size by palpation, mammogram
  • nodal involvement by palpation
  • metastasis by physical exam, CXR, LFTs, bone scan
• pathological
  • histology
  • axillary dissection should be performed for accurate staging and to reduce risk of axillary recurrence
  • estrogen/progesterone receptor testing
    • other prognostic indicators - nuclear and histologic grade, presence or absence of estrogen and progesterone receptors,
    DNA content, and proliferative fraction (S phase)

<table>
<thead>
<tr>
<th>Stage</th>
<th>Tumour</th>
<th>Nodes (regional)</th>
<th>Metastasis</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>in situ</td>
<td>none</td>
<td>none</td>
</tr>
<tr>
<td>I</td>
<td>&lt; 2 cm</td>
<td>none</td>
<td>none</td>
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<tr>
<td>II</td>
<td>&lt; 2 cm</td>
<td>movable ipsilateral</td>
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<tr>
<td>or 2-5 cm</td>
<td>none or movable ipsilateral</td>
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<tr>
<td>or &gt; 5 cm</td>
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<tr>
<td>III</td>
<td>any size</td>
<td>fixed ipsilateral or internal mammary</td>
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</tr>
<tr>
<td>or skin/chest</td>
<td>any</td>
<td>none</td>
<td>none</td>
</tr>
<tr>
<td>any wall invasion</td>
<td>any</td>
<td>none</td>
<td>none</td>
</tr>
<tr>
<td>any tumour</td>
<td>any</td>
<td>none</td>
<td>distant</td>
</tr>
</tbody>
</table>

Illustration by Mary Sims

Table 13. Staging of Breast Cancer (American Joint Committee)
Pathology

non-invasive

- ductal carcinoma in situ (DCIS)
  - proliferation of malignant epithelial cells completely contained within breast ducts
  - more common than lobular carcinoma in situ
  - 80% are non-palpable and detected by screening mammogram
  - risk of development of infiltrating ductal carcinoma in same breast is 25-30%; considered a pre-malignant lesion

lobular carcinoma in situ (LCIS)

- proliferation of malignant epithelial cells completely contained within breast lobule
- no palpable mass, no mammographic findings, usually found on biopsy for another abnormality
- risk marker for future infiltrating ductal carcinoma in either breast (20 to 30% twenty year risk)

invasive

- infiltrating ductal carcinoma (most common - 80%)
  - originates from ductal epithelium and infiltrates supporting stroma
  - characteristics - hard, scirrhous, infiltrating tentacles, gritty on cross-section
- invasive lobular carcinoma (8-10%)
  - originates from lobular epithelium
  - more apt to be bilateral, better prognosis
  - does not form microcalcifications
- Paget’s disease (1-3%)
  - ductal carcinoma that invades nipple with scaling, eczematoid lesion
- inflammatory carcinoma (1-4%)
  - ductal carcinoma that involves dermal lymphatics
  - most aggressive form of breast cancer
  - presents with erythema, skin edema, warm swollen tender breast, +/- lump
  - peau d’orange indicates advanced disease (IIib-IV)
  - also papillary, medullary, colloid, tubular cancers

sarcomas of breast (rare)

- most common = giant benign variant of fibroadenoma (cystosarcoma phyllodes) - 1 in 10 malignant
- staging more important than pathology in determining prognosis

Primary Treatment of Breast Cancer

total mastectomy – removes breast tissue, nipple-areolar complex and skin
modified radical mastectomy (MRM) – removes breast tissue, pectorali fascia, nipple-areolar complex, skin and axillary lymph nodes

Stage 0

- DCIS – total ipsilateral mastectomy vs. wide local excision (WLE) plus radiation therapy (XRT), axillary node dissection is not required for DCIS
- LCIS – close observation vs. bilateral total mastectomy, axillary node dissection is not required
- Paget’s disease – total mastectomy vs. MRM

Stages I, II - surgery for cure

- MRM vs. WLE with axillary node dissection plus XRT
- adjuvant chemotherapy in node-positive patients and high risk node-negative patient
- see Figure 16

Stages III, IV - operate for local control

- includes surgery, radiation and systemic therapy
- individualized, but mastectomy is most common procedure
- even with aggressive therapy most patients die as a result of distant metastasis

induction chemotherapy

- tumours > 5 cm
- inflammatory carcinomas
- chest wall or skin extension
BREAST... CONT.

Stage I or II
Mastectomy (MRM)/axillary node dissection
or
Segmental Mastectomy (lumpectomy)
+ axillary node dissection
+ radiation therapy

- Premenopausal
  - ER +ve
    - Nodes -ve
      - Consider Tamoxifen
    - Nodes +ve
      - Combination Chemotherapy
  - ER -ve
    - (?)

- Postmenopausal
  - ER +ve
    - Nodes -ve
      - Consider Tamoxifen
    - Nodes +ve
      - Combination Chemotherapy
  - ER -ve
    - Nodes -ve
    - Nodes +ve

NB. controversy related to adjuvant chemotherapy for premenopausal node –ve

Figure 16. NIH Recommendations

Adjuvant Therapy – Combination Chemotherapy
indications – node-positive patients, high risk node-negative patients, and palliation for metastatic disease,
ER negative patients
  - sub-groups of stage I at high risk of recurrence
    - lymphatic invasion
    - high-grade tumours
    - high S-phase fraction
    - aneuploid DNA

consider patient’s ability to tolerate side effects
CMF (cyclophosphamide, methotrexate, 5-fluorouracil) x 6 months

Adjuvant Therapy - Hormonal
indications – ER positive, pre- or post-menopausal, node-positive or high risk node-negative patients.
adjuvant or palliative therapy
Tamoxifen (anti-estrogen) – is agent of choice, continue for 5 years
alternatives to tamoxifen
  - previous clinical response to one hormonal treatment predicts response to another,
thus secondary hormonal therapies are instituted
    - progestins - megestrol acetate (Megace)
    - aromatase inhibitors - induce medical adrenalectomy
e.g. amino-glutethamide + hydrocortisone
    - estrogens - diethylstilbestrol
    - androgens - fluoxymesterone
    - oophorectomy - premenopausal patients with metastatic disease, no benefit over tamoxifen

Adjuvant Therapy - Radiation
with breast-conserving surgery
adjuvant radiation to breast decreases local recurrence, increases disease free survival
(no change in overall survival)

Post-Surgical Management
follow-up of post-mastectomy patient
  - history and physical every 4-6 months
  - yearly mammogram of remaining breast
follow-up of segmental mastectomy patient
  - history and physical every 4-6 months
  - mammograms every 6 months x 2 years, then yearly thereafter
when clinically indicated
  - chest x-ray
  - bone scan
  - LFTs
  - CT of abdomen
  - CT of brain
BREAST... CONT.

Local/Regional Recurrence
recurrence in treated breast or ipsilateral axilla; 10% develop contralateral malignancy
50% have metastatic disease - need metastatic workup; occurs most frequently in first 3 years
treatment is palliative for this group

Metastatic Disease
lung 65%
bone 56%
liver 56%

Prognosis
all patients: 63% 5 year survival, 46% 10 year survival
if disease localized to breast: 75-90% clinical cure rate
if localized and receptor-positive: 90% 5-year survival
if positive axillary nodes: 40-50% 5-year survival

THYROID

(see Endocrinology/Otolaryngology Chapters)

HERNIA

a fascial defect; protrusion of a viscus into an area in which it is not normally contained
incidence
• male:female = 9:1
• lifetime risk of developing hernia
• males 5%
• females 1%
• most common surgical disease of males
general types
• complete – hernia sac and contents protrude through defect
• incomplete – incomplete protrusion through the defect
• internal hernia - sac is within abdominal cavity
• external hernia - sac protrudes completely through abdominal wall
• strangulated hernia - vascular supply of protruded viscus is compromised (ischemia)
• incarcerated hernia - irreducible hernia, not necessarily strangulated
• Richter's hernia - contents of the sac consist of only one side of intestinal wall (usually antimesenteric)
  • danger: self-reducing gangrenous bowel
• sliding hernia - part of wall of hernia formed by protruding viscus
  (usually cecum, sigmoid colon, bladder)

Locations and Anatomy
borders of Hasselbach's triangle - lateral edge of rectus sheath, inguinal (Poupart's) ligament,
inferior epigastric vessels
inguinal
• indirect
  • males > females (most common hernia in men and women)
  • etiology
  • persistent processus vaginalis (congenital) in 20% of adults
  • anatomy
  • originates in deep inguinal ring
  • lateral to inferior epigastric artery
  • often descends into scrotal sac
  • complications
  • incarceration, strangulation
• direct (1% of all men)
  • etiology
  • acquired weakness in floor of Hesselbach's triangle (transversalis fascia)
  • due to wear/tear, combined with increased intra-abdominal pressure
  • anatomy
  • through Hasselbach's triangle
  • medial to inferior epigastric artery
  • usually does not descend into scrotal sac
  • complications
  • incarceration, strangulation
• pantaloon
  • combined direct and indirect hernias
  • peritoneum draped over inferior epigastric vessels
femoral
  • epidemiology
    • affects mostly females (pregnancy, exertion)
  • anatomy
    • into femoral canal, below inguinal ligament but may override it
    • located medial to femoral vein within femoral canal
  • complications
    • tendency to incarcerate (1/3)/strangulate since it has a narrow neck

contents of spermatic cord
  • vas deferens, testicular artery/veins
  • genital branch of genital femoral nerve
  • lymphatics, cremaster muscle

other
  • incisional: ventral hernias - hernia at site of wound closure (may be secondary to wound infection)
  • umbilical: passes through umbilical ring (congenital, ascites, pregnancy, obesity)
  • epigastric: defect in linea alba above umbilicus
  • obturator: through obturator foramen
  • spigelian: ventral hernia through defect in linea semilunaris
  • lumbar: defect in posterior abdominal wall; superior - Grynfeltt's, inferior - Petit's
  • Littre's: hernia involving Meckel’s diverticulum

clinical presentation
  • contributing factors
    • obesity, chronic cough, pregnancy, constipation, straining on urination, ascites,
      activities which increase intra-abdominal pressure, congenital abnormality (patent processus)
    • previous hernia repair
  • groin mass of variable size
  • develops insidiously in most cases
  • occasionally precipitated by single forceful muscular event
  • associated discomfort
    • worse at end of day
    • relieved at night when patient reclines and hernia reduces
    • relieved with manual reduction
  • +/- obstruction
    • +/- local tenderness
  • must examine patient in both supine and standing positions
  • hernial sac and contents enlarge and transmit palpable impulse when patient coughs or strains
  • may auscultate bowel sounds
  • unable to “get above” groin mass with palpation
  • mass does not transilluminate
  • strangulation results in
    • intense pain followed by tenderness
    • intestinal obstruction
      • gangrenous bowel
      • sepsis
  • a surgical emergency
    • small, new hernias more likely to strangulate
  • do not attempt to manually reduce hernia if sepsis present or contents of hernial sac
    thought to be gangrenous

treatment
  • surgical: goals are to prevent strangulation, eviscerations and for cosmetics
  • indirect hernias - principle of repair is high ligation of sac and tightening of the internal ring
  • direct hernias - principle of repair is to rebuild Hesselbach's triangle: need good fascia or a prosthesis
  • femoral hernias - principle of repair is to remove sac of fat and close the femoral canal with sutures

postoperative complications
  • scrotal hematoma
  • deep bleeding - may enter retroperitoneal space and not be initially apparent
  • difficulty voiding
  • painful scrotal swelling from compromised venous return of testes
  • neuroma/neuritis
  • stenosis/occlusion of femoral vein when treating femoral hernias causing acute leg swelling

prognosis (inguinal hernia repair)
  • indirect: < 1% risk of recurrence
  • direct: 3-4% risk of recurrence
Trimodal trauma death
- immediate (seconds) = 55%
- early (1-2 hours) = 35%
- late (days) = 10%

Primary survey (see Emergency Medicine Chapter)

A = airway (including c-spine control)
- goal: secure airway and protect immobile spinal cord (cervical collar)
- means: chin lift, jaw thrust, oral/nasal airway, endotracheal intubation, cricothyroidotomy

B = breathing
- goals
  - secure oxygenation and ventilation
  - treat life-threatening thoracic injuries
  - means: inspect, auscultate, percuss, palpate
  - rule out: airway obstruction, tension pneumothorax, open pneumothorax, flail chest, cardiac tamponade, massive hemothorax

C = circulation
- goals
  - secure adequate tissue perfusion
  - treat external bleeding
  - means: HR, BP, peripheral perfusion, urine output (U/O), mental status, capillary refill, skin colour
    - 2 large bore (14 to 16 guage) IV’s in upper extremities (Ringer’s Lactate = 2L)
    - gastric decompression with NG tube
    - bladder decompression with Foley catheter
    - child: 20 cc/kg bolus Ringer’s lactate
    - consider PRBC’s if no BP response to fluid

D = disability
- goal
  - determine neurologic injury
  - means: pupils, mental status (GCS), motor/sensory exam

E = exposure/environment
- goals
  - complete disrobing for inspection and palpation
  - warm environment for patient

Secondary survey
- AMPLE history
- complete physical exam (including all orifices)
- examine patient’s back (log-roll)
- full abdominal exam
- rule out: hemotympanum, ototorhea, Battle’s sign, periorbital ecchymoses, rhinorrea, hyphema, nasal septum hematoma, mandibular fracture, thoracic trauma, broken ribs/sternum, subcutaneous emphysema, pelvic fractures
- rectal exam (tone, blood, prostate position)
- extremity exam (neurovascular, fractures)
- treat pain (e.g. Fentanyl)

Laboratory
- CBC, Diff., amylase, LFTs, lactate, PTT, INR, type and cross, urine analysis

Radiology
- lateral c-spine
- AP chest
- AP pelvis
  - rule out: c-spine injury, thoracic vessel injury
  - focussed abdominal sonogram for trauma (FAST) ultrasound (or diagnostic peritoneal lavage (DPL))
  - rule out: free fluid
  - complete film series as necessary (including extremities)
  - CT head/abdomen as required
  - thoracic aortagram as required

if continued bleeding/perforated viscus/extensive organ damage --> consider O.R.
PREOPERATIVE PREPARATION

PREOPERATIVE

informed consent
consults - anesthesia, medicine, cardiology, etc.
blood components: group and screen or crossmatch depending on procedure
diet - NPO after midnight
AAT, vital signs routine
IV - balanced crystalloid at maintenance rate (4:2:1 rule)
  • Ringer's lactate or normal saline
investigations
  • CBC, lytes, BUN, creatinine, urine analysis
  • INR/PT, PTT with history of bleeding disorder
  • ABGs if predisposed to respiratory insufficiency
  • CXR (PA and lateral) if > 35 years old or previously abnormal within past 6 months
  • ECG > 35 years old or as indicated by past cardiac history
  • patient's regular meds including prednisone - consider pre-op boost
  • prophylactic antibiotics (on call to OR) (e.g. cefazolin) if
    • clean/contaminated cases (i.e. GI/GU/respiratory tracts are entered)
    • contaminated cases - trauma
    • insertion of foreign material (e.g. vascular grafts)
    • high risk patients (e.g. prosthetic heart valves, rheumatic heart disease)
    • bowel prep (decreases bacterial population e.g. Ancef, Cipro, Flagyl)
drains
  • nasogastric (NG) tube
    • indications: gastric decompression, analysis of gastric contents, irrigation/dilution of gastric contents, feeding (only if necessary — due to risk of aspiration, naso-jejunal tube preferable)
    • contraindications: absolute - obstruction of nasal passages due to trauma, suspected basilar skull fracture, relative - maxillofacial fractures; for these may use oral-gastric tube
  • Foley catheter
    • indications: to accurately monitor urine output, decompression of bladder, relieve obstruction
    • contraindications: suspected disruption of the urethra, difficult insertion of catheter

SURGICAL COMPLICATIONS

WOUND COMPLICATIONS

Wound Infection
risk of infection depends on type of procedure
  • clean (elective, nontraumatic without inflammation) - < 1.5%
  • clean-contaminated (GI, biliary, urinary) - < 3%
  • contaminated (surgery on unprepped bowel, emergency surgery for GI bleeds/perforation/trauma with acute inflammation) - 5%
  • dirty (penetrating trauma, pus present) - 33%
most common etiologic agents = S. aureus (20%), E. coli, enterococcus (10% each)
predisposing factors
  • patient characteristics: age, diabetes, steroids, immunosuppression, obesity, burn, malnutrition, patient with other infections, traumatic wound, radiation, chemotherapy
  • other factors: prolonged preoperative hospitalization, duration of surgery (> 2 hrs), reduced blood flow, break in sterile technique, use of drains, multiple antibiotics, hematoma, seroma, foreign bodies (drains, sutures, grafts)
clinical presentation
  • typically fever post operative day (POD) # 3-6
  • pain, wound erythema, induration, frank pus or purulosanguinous discharge, warmth
treatment
  • re-open affected part of incision, culture wound, pack, heal by secondary intention
  • antibiotics only if cellulitis or immunodeficiency present
prophylaxis
  • consider IV antibiotics
  • debridement of necrotic and non-viable tissue
complications
  • fistula, sinus tracts, sepsis, abscess, suppressed wound healing, superinfection
  • note: Streptococcus and clostridium wound infections may present with fever within the first 24 hrs. post-op

Wound Hemorrhage/Hematoma
secondary to inadequate surgical control of hemostasis
risks: anticoagulant therapy, myeloproliferative disorders (e.g. polycythemia vera)
symptoms: pain, swelling, discoloration of wound edges, leakage

MCCQE 2006 Review Notes
General Surgery – GS45
SURGICAL COMPLICATIONS. CONT.

Wound Dehiscence
- Definition: disruption of fascial layer, abdominal contents contained by skin
- Evisceration: disruption of all abdominal wall layers and extrusion of abdominal contents (mortality of 15%)
- Incidence: 0.3-5% of abdominal incisions
- Typically POD 5-8
- Most common presenting sign is sero-sanguinous drainage from wound
- Predisposing factors
  - Local: poor closure, increased intra-abdominal pressure (e.g., chronic obstructive pulmonary disease (COPD)), ileus, bowel obstruction, poor wound healing (hemorrhage, infection)
  - Systemic: hypoproteinemia, steroids, age, DM, immunosuppression, sepsis, jaundice
- Treatment: operative closure
  - Evisceration is a surgical emergency
  - Mild dehiscence may be treated expectantly with delayed repair of the resulting hernia

URINARY AND RENAL COMPLICATIONS

Urinary Retention
- May occur after any operation with general anesthesia (GA) or spinal anesthesia
- More likely in older males with history of prostatism
- Treatment: bladder catheterization

Acute Renal Failure (see Nephrology Chapter)
- Defined as urine output < 25 cc/hr, increasing Cr, increasing BUN
- High associated mortality > 50%
- Classified according to primary cause e.g., pre-renal, renal, post-renal
- Treatment: according to underlying cause
- Decreased renal perfusion treated with fluid boluses
- Consider central venous pressure (CVP) line or Swan-Ganz catheter if patient does not respond to fluid bolus

RESPIRATORY COMPLICATIONS

Atelectasis
- Comprises 90% of post-op pulmonary complications
- Clinical manifestations usually in first 24 hours post-op
  - Low fever, tachycardia, crackles, decreased breath sounds, bronchial breathing, cyanosis, tachypnea, CXR (increased density)
- Risk factors
  - COPD
  - Smoking
  - Abdominal or thoracic surgery
  - Oversedation
  - Significant post-op pain
- Pre-operative prophylaxis
  - Quit smoking
  - Deep abdominal breathing and coughing
- Post-operative prophylaxis
  - Incentive spirometry
  - Minimize use of depressant drugs
  - Good pain control
  - Frequent changes in position (postural drainage)
  - Deep breathing and coughing
  - Early ambulation

Chest physiotherapy

Aspiration Pneumonitis
- Aspiration of gastric contents can be lethal
- Major determinant of degree of injury is gastric pH
- Occurs most often at time of anesthetic induction and at extubation
- Risk factors
  - General anesthetic
  - Decreased level of consciousness
  - Dysphagia
  - Non-functioning nasogastric tube
- Clinical manifestations
  - Respiratory failure
  - Increased sputum
  - Fever
  - Cough
  - Decreased level of consciousness
  - Tachycardia, cyanosis
  - Infiltrate on CXR
SURGICAL COMPLICATIONS. CONT.

treatment
- immediate removal of debris and fluid from airway
- consider endotracheal intubation and flexible bronchoscopic aspiration
- IV antibiotics to cover oral aerobes and anaerobes

Pulmonary Embolus
- blood clot from the venous system that embolizes to the pulmonary arterial system
- risk factors
  - operations, immobility, vessel injury, hypercoagulable states (low protein C, S)
  - CHF, obesity, OCP, age, pregnancy, age, polycythemia vera
- clinical manifestations
  - dyspnea, tachypnea, pleuritic chest pain
  - hypotension, fever, hemoptysis, right ventricle strain
  - ABG shows hypoxia and low pCO2
  - ECG shows S1Q3T3, right bundle branch block (RBBB) and right axis deviation (50% of cases)
  - CXR findings
- treatment
  - IV heparin (PTT = 2.0)
  - long term coumadin (INR = 2-3) for 3 months
  - greenfield filter if unable to anticoagulate
  - embolectomy if patient unstable
  - prophylaxis
    - compression stockings, ambulation if possible
    - subcutaneous heparin (5000 units Q12H beginning pre-op)

Pulmonary Edema
- occurs during or immediately after operation
- results from circulatory overload
  - overzealous volume replacement
  - left ventricular failure
  - shift of fluid from peripheral to pulmonary vascular bed
  - negative airway pressure
  - alveolar injury due to toxins
- treatment
  - O2
  - remove obstructing fluid
  - correct circulatory overload
  - diuretics, positive end expiratory pressure (PEEP) in intubated patient

Respiratory Failure
- clinical manifestations - dyspnea, cyanosis, evidence of obstructive lung disease, pulmonary edema, unexplained decrease in PaO2
- earliest manifestations - tachypnea and hypoxemia (pO2 < 60, RR > 25)
  - NB: hypoxemia may initially present with confusion/delirium
- treatment
  - O2 by mask
  - pulmonary toilet (i.e. clear secretions from airway)
  - bronchodilators
  - treatment of acute respiratory insufficiency – intubation and ventilation
- if these measures fail to keep PaO2 > 60, consider acute respiratory distress syndrome (ARDS)
- control of post-operative pain can decrease pulmonary complications
  - problematic with thoracic and upper abdominal operations

CARDIAC COMPLICATIONS
- abnormal ECGs common in post-operative period (compare to pre-op)
- common arrhythmia – supraventricular tachycardia (SVT)
- atrial fibrillation (secondary to fluid overload, pulmonary embolus (PE), MI, pain)

Myocardial Infarction (MI)
- surgery increases risk of MI
- majority of cases on operative day or within first 4 postoperative days
- incidence
  - 0.5% in previously asymptomatic men > 50 years old
  - 40-fold increase in men > 50 years old with previous MI
- clinical manifestations
  - often silent without chest pain
  - new onset congestive heart failure (CHF) (dyspnea), cardiac arrhythmias, hypotension
  - chest pain
- risk factors
  - pre-operative hypertension
  - pre-operative CHF
  - operations > 3 hours
  - intra-operative hypotension
  - angina pectoris
  - MI in 6 months preceding surgery
  - increased age
PARALYTIC ILEUS
normal bowel sounds disappear following abdominal surgery
also follows peritonitis, abdominal trauma, and immobilization
return of GI motility following abdominal surgery varies
  • small bowel motility returns by 24-48 hours
  • gastric motility returns by 48 hours
  • colonic motility - up to 3-5 days
due to normal paralysis of myenteric plexus (adynamic ileus)
two forms
  • intestinal ileus
  • gastric dilatation
must rule out secondary causes
  • hypokalemia
  • narcotics
  • intraperitoneal infection
symptoms
  • abdominal distension, nausea and vomiting
  • absent or tinkly bowel sounds
  • flatus and stool indicate a resolving ileus
treatment
  • NG tube, fluid resuscitation and time
  • for prolonged ileus, consider TPN

POST-OPERATIVE DELIRIUM
disturbance of sleep-wake cycle
disturbance of attention, distractibility, disorientation
fluctuating course throughout day
incidence: 40% (likely an underestimate)
derived unrecognized
no correlation with type of anesthetic agent
risk factors
  • > 50 years old or very young
  • pre-existing cognitive dysfunction
  • depression
  • peri-operative biochemical derangements
  • > 5 prescribed medications post-operatively
  • use of anticholinergic medications preoperatively
  • cardiopulmonary bypass
  • ICU setting
  • substance abuse
treatment
  • minimize non-essential medications if possible
  • hydrate, maintain electrolyte balance
  • treat underlying cause if possible
  • well-lit room, visual cues, exercise, family members present
  • benzodiazepines if necessary; try to avoid antipsychotics (e.g. Haldo)

POST-OPERATIVE FEVER
fever does not necessarily imply infection
  • timing of fever may help identify cause
  • "6 Ws" - CLINICAL PEARL
  • Wind (pulmonary)
  • Water (urine-UTI)
  • Wound
  • Walk (deep vein thrombosis (DVT) - pulmonary embolism (PE))
  • Wonder drugs (drug fever)
  • Wanes (rhymes with veins: IV sites)
0-48 hours
  • usually atelectasis
  • consider early wound infection (especially Clostridia, Group A Streptococcus)
  • leakage of bowel anastomosis (tachycardia, hypotension, oliguria, abdominal pain)
  • aspiration pneumonia
  • addisonian crisis
  • thyroid storm
  • transfusion reaction
POD# 3
  • after day 3 infections more likely
  • UTI - patient instrumented? e.g. Foley catheter
  • wound infection (usually POD 3-6)
  • IV site - especially IVs in place > 3 days
  • septic thrombophlebitis
  • intra-abdominal abscess (usually POD 5-10)
  • DVT (POD 7-10)
drug fever (POD 6-10)
also consider - cholecystitis, PE, sinusitis, prostatitis, peri-rectal abscess, drug fever, URTI, factitious fever
treatment
  • treat primary cause
  • acetaminophen, aspirin
INTRA-ABDOMINAL ABSCESS
A collection of pus walled-off from rest of peritoneal cavity by inflammatory adhesions and viscera; usually polymicrobial. Danger: may perforate secondarily —> diffuse bacterial peritonitis. Common sites: pelvis, Morrison’s pouch, subphrenic, paracolic gutters, lesser sac, periappendiceal. Clinical manifestations: persistent, spiking fever, dull pain, weight loss, leukocytosis or leukopenia. Impaired function of adjacent organs (e.g. ileus, or diarrhea with rectal abscess). Co-existing effusion (pleural effusion with subphrenic abscess).

Diagnosis: CBC, blood cultures. Usually by U/S or CT (if > POD# 7). DRE (pelvic abscess). Treatment: drainage (surgical or percutaneous). Antibiotics to cover aerobes and anaerobes (Clindamycin/Gentamicin, Flagyl/Gentamicin, 3rd generation cephalosporin).

MINIMALLY INVASIVE SURGERY
SURGICAL PROCEDURES

Appendectomy
objective: surgically remove inflamed or ruptured appendix
indications: RLQ pain, peritoneal signs on physical exam, fever and anorexia, N/V, leukocytosis
anatomic landmarks: incision over McBurney’s point, cecum, mesoappendix, appendiceal artery
complications: perforation of appendix, pelvic abscess, enterocutaneous fistula

Laparoscopic Cholecystectomy
objective: remove gall bladder with small surgical incisions to relieve symptoms/pathologic process
indications: acute or chronic cholecystitis, cholelithiasis, choledocholithiasis
anatomic landmarks: right subcostal port incisions, peri-umbilical incision, gall bladder, cystic duct and artery
complications: CBD injury, hollow viscus injury, post-op pancreatitis, bile peritonitis, subhepatic or subphrenic

Colectomy/Small Bowel Resection
objective: remove diseased segment or entire bowel
indications: malignancy, diverticular disease, ischemic bowel, IBD, volvulus, SBO, trauma
anatomic landmarks: midline abdominal incision, blood supply,
mesentery and regional lymph nodes to the resected segment
complications: anastomotic leak or stricture, recurrent disease, pelvic abscess, enterocutaneous fistula

Partial/Total Mastectomy
objective: remove diseased portion or entire breast +/- axillary lymph node sampling
indications: malignant disease, high-risk histopathology
anatomic landmarks: transverse incision over breast, clavicle (superior), latissimus dorsi (posterior),
clavicle (posterior), 6th rib (inferior), long thoracic and thoracodorsal nerves, axillary vein,
pectoralis muscle and fascia, axillary lymph nodes
complications: lymphedema or lymphadenitis, recurrent disease, hematoma or seroma, axillary vein injury,
long thoracic nerve injury, thoracodorsal nerve injury

Inguinal Hernia Repair
objective: repair tissue and fascial defect to relieve GI and cosmetic symptoms
indications: inguinal hernia, small bowel obstruction, incarceration, or strangulation
anatomic landmarks: inguinal incision, Scarpa’s fascia, external oblique muscle and fascia, internal oblique,
external ring, spermatic cord (male), round ligament (female), ilioinguinal nerve, inferior epigastric vessels,
Cooper’s ligament, transversalis fascia
complications: recurrence, spermatic cord injury, ilioinguinal nerve entrapment, testicular swelling

REFERENCES


