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Chapter 1

UPPER GIT
Perforated Peptic Ulcer

Overview

○ Epidemiology
  • The 15% death rate correlates with increased age, female sex, and gastric perforations
  • Severity of illness and occurrence of death are directly related to the interval between perforation and surgical closure

Clinical Presentation

○ Symptoms
  • Sudden, severe upper abdominal pain which typically occurs several hours after the last meal
  • There may be a history of previous dyspepsia, previous or current Treatment and Management for peptic ulcer or ingestion of NSAID
  • Rarely is heralded by nausea or vomiting
  • Shoulder pain. Back pain is uncommon

○ Signs
  • The patient appears severely distressed, lying quietly with the knees drawn up and breathing shallowly to minimize abdominal motion
  • Fever and hypotension (late sign)
  • The Epigastric tenderness may not be as marked as expected because the board-like rigidity
  • Tympanitic percussion over the liver and ileus
Differential Diagnosis

- Hepatobiliary: hepatitis, cholecystitis, pancreatitis, cholangitis
- Intestine: appendicitis, colitis, bowel perforation, ischemia
- Extraabdominal: inferior myocardial infarction, basal pneumonia

Work Up

- Laboratory
  - CBC: leukocytosis
  - Serum amylase mildly elevated
  - ABG: metabolic acidosis
- Imaging
  - Erect chest x-ray: air under diaphragm in 85%
  - CT scan abdomen with IV and PO contrast in doubtful cases

Treatment and Management

(See Flowchart 1)

- Medical therapy (See Table 1)
  - Non-operative management is appropriate only if there is clear evidence that the leak has sealed (by contrast study) in the absence of peritonitis
  - IV antibiotic and proton pump inhibitor
Surgical therapy
- Laparotomy and omental patch for perforated duodenal ulcer and prepyloric gastric ulcer
- Resection for gastric ulcer (most likely cancerous)

Red Flag

- Acute onset or chronic symptoms
- Shock and peritonitis
- Air under diaphragm but minimal symptoms and signs (sealed perforation)

Reference

Flowchart 1: Perforated Peptic Ulcer

Suspecting perforated peptic ulcer based on history and examination

Initial management
ABC, IV line, resuscitation
CBC, chemistry, coagulation
Liver function, amylase
 Erect chest x-ray

Red Flag Features:
Acute onset, peritonitis, shock

Non-sealed perforation
Resuscitation
Antibiotic
Cefuroxim and metronidazole
Pip/Taz for severe sepsis
Proton pump inhibitor IV
(Pantoprazole)
NPO and NGT

To OR
Laparotomy and omental patch
H. Pylori eradication

Sealed perforation
Conservative
Antibiotic
Cefuroxim and metronidazole
Pip/Taz for severe sepsis
Proton pump inhibitor IV
(Pantoprazole)
NPO, NGT and close monitor

Urgent CT abdomen with PO + IV contrast
Positive for contrast leak from perforation

Gastric Outlet Obstruction

Overview

○ Epidemiology
  • Occurs in 2% of patients with ulcer disease causing obstruction of pylorus or duodenum by scarring and inflammation
  • Cancer must be ruled out because most patients who present with symptoms of gastric outlet obstruction will have a pancreatic, gastric, or duodenal malignancy

Clinical Presentation

○ Symptoms
  • Nausea, nonbilious vomiting contains undigested food particles, early satiety, bloating, anorexia, epigastric pain and weight loss

○ Signs
  • Chronic dehydration and malnutrition
  • Tympanitic mass (dilated stomach in the epigastric area and/or left upper quadrant
  • Succussion splash in the epigastrium

Differential Diagnosis

○ Hepatobiliary: Pancreatitis, pancreatic cancer

○ Stomach: volvulus, obstructed hiatus hernia, foreign body
**Work Up**

- Laboratory: CBC, electrolyte panel, Liver function tests

- Imaging
  - Plain abdominal radiograph
  - Contrast upper GI studies (Gastrografin or barium)
  - CT scans with oral contrast

- Upper endoscopy with biopsy

**Treatment and Management**

(See flowchart 2)

- Medical therapy (See Table 1)
  - Resuscitation and correction of electrolyte imbalance
  - IV proton pump inhibitor
  - NPO and NG tube
  - H.pylori eradication

- Surgical therapy
  - Antrectomy vs. vagotomy vs. dilatation / stenting

**Red Flags**

- Weight loss and anorexia

- Unexplained anemia

- Palpable mass

**References**
1- Andres E Castellanos, MD; Chief Editor: John Geibel, MD, DSc, MA: Gastric Outlet Obstruction. eMedicine of Medscape


3- Gerard M. Doherty, MD, Lawrence W. Way, MD. CURRENT Work Up and Treatment and Management: Surgery, 13e, 2010

4- Maxine A. Papadakis, MD, Stephen J. McPhee, MD. Quick Medical Work Up and Treatment and Management, 2013
Flowchart 2: Peptic Ulcer and Gastric Outlet Obstruction

Suspecting peptic ulcer disease based on history and examination

Initial management
CBC, chemistry, coagulation liver function, amylase
Erect chest x-ray

Red Flag Features
Epigastric pain, severe nausea and non-bilious vomiting, weight loss, epigastric fullness

Simple, uncomplicated peptic ulcer
Proton pump inhibitor
Lifestyle modification
Gastroenterology referral

Urgent CT abdomen with PO + IV contrast

No
Gastric outlet obstruction

Yes
Resuscitation
Correction of electrolyte
NPO, NGT
Proton pump inhibitor IV
Pantoprazole
Endoscopy to rule out malignant cause
H.pylori eradication

Failed

Rule out malignancy
Surgery
Anrectomy vs vagotomy
Dilatation/stenting is an option
H.pylori eradication
Upper GI Bleeding

Overview

- Definition: bleeding proximal to the ligament of Treitz, which accounts for 75% of GI bleeding

- Etiology
  - Above the GE junction: epistaxis, esophageal varices (10-30%), esophagitis, esophageal cancer, Mallory-Weiss tear (10%)
  - Stomach: gastric ulcer (20%), gastritis (e.g. from alcohol or post-surgery) (20%), gastric cancer
  - Duodenum: ulcer in cap/bulb (25%), aortoenteric fistula post aortic graft
  - Coagulopathy: drugs, renal disease, liver disease
  - Vascular malformation: Dieulafoy’s lesion, AVM

Clinical Presentation

- In order of decreasing severity of the bleed: hematochezia followed by hematemesis, coffee ground emesis, melena and then occult blood in stool

Treatment and Management

(See flowchart 3)

- Initial management (See Table 2)
  - Resuscitation and monitoring (2 large bore IV lines, fluid, urinary catheter)
  - Send blood for CBC, cross and type, coagulation profile, electrolytes, liver and kidney functions
  - Keep NPO and insert NGT.
• IV Proton pump inhibitors.
  ▪ It decreases risk of rebleeding if endoscopic predictors of rebleeding are seen
  ▪ It is given to stabilize clot, not to accelerate ulcer healing
  ▪ If it is given before endoscopy, it decreases the need for endoscopic intervention
• In case of esophageal varices: IV octreotide (50 mcg loading dose followed by constant infusion of 50 mcg/hr and IV antibiotic (ciprofloxacin)
• Consider IV erythromycin (or metoclopramide) to accelerate gastric emptying prior to gastroscopy to improve visualization
  o Localization of the bleeder: esophagogastroduodenoscopy (EGD)
  o Definitive management
    • EGD: establish bleeding site+ treat lesion
    • Bleeding peptic ulcer: injection of epinephrine around bleeding point, thermal hemostasis (bipolar electrocoagulation or heater probe) and endoclips
    • Variceal bleeding: banding or glue injection, the medical therapy +/- TIPS.

Reference

Flowchart 3: Upper GI Bleeding

Suspecting Upper GI bleeding based on history and examination

Initial Management
- ABC +IV line resuscitation
- CBC, Chemistry, liver function, coagulation
- NGT for decompression

Medical Therapy
- Proton pump inhibitor IV
  - Pantoprazole infusion
- Suspect esophageal varices
- Octreotide IV
- Antibiotics: ciprofloxacin

Cause of bleeding
- Peptic ulcer
  - Yes
  - Upper GI endoscopy successful
  - Continue conservative therapy
  - Re-bleeding
    - Yes
    - OR
      - Duodenotomy and suture
    - No
      - Conservative therapy
  - No
    - Gastric / Esophageal varices
      - Yes
      - Sengstaken tube
      - Prepare for TIPS
      - banding/glue
      - Continue conservative therapy
  - No

Bowel Obstruction

Overview

- **Definition:** partial or complete blockage of the bowel resulting in failure of intestinal contents to pass through lumen

- **Pathogenesis**
  - Disruption of the normal flow of intestinal contents ~ proximal dilation + distal decompression
  - May take 12-24 h to decompress, therefore passage of feces and flatus may occur after the onset of obstruction
  - Bowel ischemia may occur if blood supply is strangulated or bowel wall tension lesion leads to venous congestion
  - Bowel wall edema and disruption of normal bowel absorptive function ~ increased intraluminal fluid ~ transudative fluid loss into peritoneal cavity, electrolyte disturbances

- **Etiology**
  - **Small bowel obstruction (SBO)**
    - Extramural: Adhesions (most common), hernia
    - Intraluminal: Intussusception, gallstones ileus, bezoar.
    - Intramural: Crohn’s disease, radiation stricture and tumors
  - **Large bowel obstruction (LBO)**
    - Intramural: Cancers (most common), diverticulitis, crohn’s stricture
    - Extramural: Volvulus and adhesions
  - **Pseudo-obstruction (functional):** paralytic ileus, electrolyte disturbance, drugs
Clinical Presentation

- Must differentiate between obstruction and ileus, and characterize obstruction as acute vs. chronic, partial vs. complete (constipation vs. obstipation), small vs. large bowel, strangulating vs. non-strangulating, and with vs. without perforation / ischemia.

- Nausea, vomiting, abdominal pain, abdominal Distention and constipation

- Complications (of total obstruction)
  - Hypovolemia (due to third spacing) and bacterial translocation
  - Strangulation (10% of bowel obstructions): a surgical emergency
    - Cramping pain turns to continuous ache, hematemesis and melena (if infarction)
    - Fever, tachycardia, peritoneal signs and early shock
    - Leukocytosis and metabolic acidosis
  - Perforation: secondary to ischemia and luminal distention

Differential Diagnosis

- Small bowel: mesenteric ischemia, bowel perforation, Crohn’s disease

- Large bowel: diverticulitis, colitis, appendicitis

- Hepatobiliary: cholecystitis, pancreatitis
Peptic ulcer disease (perforation)

Aortic aneurysm

**Work Up**

**Laboratory**
- May be normal early in disease course
- CBC: hematocrit (hemoconcentration)
- Electrolytes: to assess degree of dehydration
- Amylase elevated
- Metabolic alkalosis due to frequent emesis
- In case strangulation: leukocytosis with left shift, lactic acidosis and elevated LDH (late signs)

**Radiological**
- Upright CXR or left lateral decubitus (LLD) to rule out free air, usually seen under the right hemidiaphragm
- Abdominal x-rays to determine SBO vs. LBO vs. ileus
  - Erect (air fluid levels), Supine (dilated bowel).
  - In case of ischemic bowel: look for free air, pneumatosis, thickened bowel wall, air in portal vein, dilated small and large bowels, thickened or hose-like haustra (normally thin finger-like projections)

- CT scan
  - It provides information on level of obstruction, severity and cause
  - Important to r/o closed loop obstruction, especially in the elderly
Upper GI series/small bowel series for SBO (if no cause apparent, i.e. no hernias, no previous surgeries) can be used
• MRI in pregnant patients

Treatment and Management

(See flowchart 4)

o Initial management (See Table 1)
  • Resuscitation and electrolyte correction (with normal saline/Ringer’s first, then add potassium after fluid deficits are corrected)
  • NG tube to relieve vomiting, prevent aspiration and decompress small bowel by prevention of further distention with swallowed air
  • Foley catheter to monitor in/outs
  • Antibiotic in case of strangulation or perforation

o Definitive management
  • Conservative therapy Especially if adhesions are the cause
  • Indication for surgery: virgin abdomen, peritonitis, perforation and septic shock

Reference


Flowchart 4: Bowel Obstruction

Suspecting bowel obstruction based on history and examination

Initial management
CBC, chemistry, ABG, IV line
Erect chest x-ray
Abdominal x-ray (2 views)
Nasogastric tube
Foley catheter

Resuscitation
Preoperative antibiotic
Cefruxim + metronidazole
Pip/Taz for severe sepsis
Urgent OR

Red flag presentation
Virgin abdomen, shock peritonitis

Yes

Resuscitation
Preoperative antibiotic
Cefruxim + metronidazole
Pip/Taz for severe sepsis
Urgent OR

No

Fluid administration
Conservative therapy
Close monitoring
Obtain CT abdomen

Large bowel obstruction on CT

Volvulus
Sigmoidoscopy → failed → laparotomy and resection + ostomy

Malignant
Diverting ostomy vs. formal resection +/- ostomy

Adhesion
Conservation → failed laparotomy and adhesolysis +/- resection

Hernia
Groin exploration
Viable bowel → mesh repair.
Dead bowel → resect + hemiorrhaphy

Small bowel obstruction on CT

Acute Appendicitis

Overview

- **Pathophysiology**
  - Obstruction of appendiceal lumen from lymphoid hyperplasia in wall of appendix (children) or fecalith (adult)

Clinical Presentation

- **Symptoms**
  - Vague, often colicky, periumbilical or epigastric pain that shifts to right lower quadrant, with steady ache worsened by walking or coughing
  - In >95% of patients with acute appendicitis, anorexia is the first symptom, followed by abdominal pain, which is followed in turn by vomiting
  - Constipation

- **Signs**
  - Low-grade fever (<37.8 °C) in the absence of perforation
  - Localized tenderness with guarding in the right lower quadrant and rebound tenderness
  - Psoas sign (pain on passive extension of the right hip)
  - Obturator sign (pain with passive flexion and internal rotation of the right hip)
  - Atypical presentations due to anatomic variations in the position of the inflamed appendix
    - Tenderness may be most marked in the flank
    - Pain in the lower abdomen, often on the left; urge to urinate or defecate
Abdominal tenderness absent, but tenderness on pelvic or rectal examination

Complications
- Perforation, appendicular abscess or phlegmon (mass) and pylephlebitis (suppurative thrombophlebitis of the portal venous system)

Differential Diagnosis
- Mesenteric lymphadenitis, Gastroenteritis, Crohn’s disease
- Renal: ureteric stone
- In female: ruptured ovarian cyst, ovarian torsion, ectopic pregnancy
- Beware of “crossover” diseases like
  - Sigmoid colon flopped into RLQ mimicking appendicitis

Work Up
- Laboratory
  - CBC: Mild leukocytosis, (12,000 to 18,000 cells/mm³)
  - Urinalysis can be useful to rule out UTI
  - Pregnancy test if applicable
Imaging

- Ultrasound scan (pelvic) is indicated in young women of childbearing age if ovarian pathology is suspected
- CT scan is indicated: suspecting appendix mass or abscess, unclear Work Up and in elderly
- MRI in pregnant patient

Invasive

- Laparoscopy is a useful tool in case all above investigations failed

Elderly patients with appendicitis often pose a more difficult diagnostic problem because of the atypical presentation

Treatment and Management

(See flowchart 5)

- Preoperative antibiotics (See Table 1)
  - All what is needed in case of appendicular mass phlegmon (followed by interval appendectomy)
  - Antibiotic coverage is limited to 24 to 48 hours in cases of non-perforated appendicitis. and 7 to 10 days for perforated appendix

- Percutaneous drainage for appendicular abscess

- Appendectomy open or laparoscopic.
Red Flags

- Females patient with missed menses or vaginal bleed
- Elderly patient with atypical, recurrent symptoms and weight loss
- Palpable mass on examination

Reference


Flowchart 5: Acute Appendicitis

Suspecting appendicitis based on history and examination

Initial management
- ABC, IV line
- CBC, Chemistry

Red Flag Presentation
- Female, elderly, atypical or recurrent symptoms, long history, or/and palpable mass on exam

Preoperative antibiotics
- Cefuroxim + metronidazole
- Obtain consent
- Book for appendectomy (open vs. laparoscopy)

Further work up
- B-hCG (married female)
- Urine analysis
- Abdominal ultrasound
- CT abdomen (optional)

OR finding
- Perforated appendix
- Intra-abdominal sepsis (IAS)

No
- Continue same antibiotics for 24hrs

Yes
- *Continue antibiotics for 7-10 days for mild IAS
- * Pip/taz inmod to severe IAS

Phlegmon or abscess

No

Yes
- Manage according to the result of the above-mentioned tests
- * IV Cefuroxim and metronidazole
- * Percutaneous drainage for abscess
- * Consider interval appendectomy
Anal Fissure

Overview

- Definition: is a tear in the anoderm distal to the dentate line
- Incidence: Most commonly occur in the posterior midline; 10% occur anteriorly
- Etiology
  - It is related to trauma from either the passage of hard stool or prolonged diarrhea
  - A lateral location of a chronic anal fissure suggests: Crohn’s disease, Syphilis, Tuberculosis, HIV/AIDS or anal carcinoma

Clinical Presentation

- Symptoms
  - Severe, tearing pain during defecation
  - Sensation of intense and painful anal spasm lasting for several hours after a bowel movement
  - Constipation and hematochezia (less common). Fissures may present as painless non-healing wounds that bleed intermittently
- Signs
  - Acute fissure is a superficial tear of the distal anoderm
  - Chronic fissures: an ulcer with heaped-up edges. The white fibers of the internal anal sphincter visible at the base of the ulcer and associated external skin tag and/or a hypertrophied anal papilla internally
Differential Diagnosis

- Thrombosed hemorrhoid
- Perianal fistula or abscess

Work Up

- Work Up is confirmed by visual inspection of the anal verge while gently separating the buttocks
- Digital (by touching the anal sphincter, anal spasm will be appreciated) and anoscopy may cause severe pain and may not be possible
- Examination under anesthesia (EUA) if in doubt or there is suspicion of another cause for the perianal pain

Treatment and Management

(See flowchart 6)

- Medical therapy (See Table 4)
  - Life-style modification, stool softener and sitz bath
  - Local analgesia
  - Local nitroglycerin or calcium channel blocker (nifedipine - less side effects).

- Surgical therapy
  - Partial Lateral internal sphincterotomy for refractory and chronic fissure
**Red Flags**

- New onset in an elderly patient
- Unexplained anaemia, tenesmus or weight loss
- Family history of bowel cancer or inflammatory bowel disease
- Associated anal mass or enlarged groin mass
- Positive faecal occult blood test

**Reference**

1. Lisa Susan Poritz, MD; Chief Editor: John Geibel, MD, DSc. eMedicine of Medscape
2. Kelli M. Bullard Dunn and David A. Rothenberger. Schwartz’s Principles of Surgery, 9e , 2010
Fistula in Ano

Overview

- **Definition:** an abnormal tract or cavity with an external opening in the perianal skin which is communicating with the rectum or anal canal by an identifiable internal opening.

- **Pathophysiology**
  - History of previous anorectal abscess, which was drained spontaneously or surgically

Clinical Presentation

- **Symptoms:** perianal discharge, pain, swelling and/or bleeding

- **Signs**
  - Perianal exam: an external opening that appears as an open sinus or elevation of granulation tissue. Spontaneous discharge of pus or blood via the external opening may be apparent or expressible on digital rectal examination.
  - Digital rectal examination may reveal a fibrous tract or cord beneath the skin. It also helps to delineate any further acute inflammation that is not yet drained. Lateral or posterior induration suggests deep postanal or ischiorectal extension.
  - The sphincter tone and voluntary squeeze pressures should be assessed before any surgical therapy.
Classification

- Four main types
  - Intersphincteric: the track is in the intersphincteric plane. The external opening is usually in the perianal skin close to the anal verge.
  - Transsphincteric: The fistula starts in the intersphincteric plane or in the deep postanal space. The track traverses the external sphincter, with the external opening at the ischioanal fossa. Horseshoe fistula is also in this category.
  - Suprasphincteric: The fistula starts in the intersphincteric plane in the midanal canal and then passes upward to a point above the puborectal muscle. The fistula passes laterally over this muscle and downward between the puborectal muscle and the levator ani into the ischioanal fossa.
  - Extrasphincteric: The fistula passes from the perineal skin through the ischioanal fossa and the levator ani and finally penetrates the rectal wall.

Differential Diagnosis

- Perianal abscess
- Hemorrhoid
Work Up

- History and Physical Examination
- Anoscopy is usually required to identify the internal opening.
- MRI and Fistulogram in complex or recurrent types
- Proctoscopy or flexible sigmoidoscopy is performed to rule out other lesions and inflammatory bowel disease

Treatment and Management

(See flowchart 6)

- Abscess: incision and drainage (See Table 3)
- Simple, superficial: fistulotomy
- Crohn’s disease, anterior fistula in female, complex or high: seton, referral to colorectal surgeon for other options.

Red Flags

- History of Crohn’s disease or HIV
- Anterior location or multiple fistulae
- Complex, recurrent or high location
Hemorrhoids

Overview

- Definition: Hemorrhoids are cushions of submucosal tissue containing venules, arterioles and smooth-muscle fibers that are located in the anal canal

- Types
  - External hemorrhoids are located distal to the dentate line and are covered with anoderm
  - Internal hemorrhoids are located proximal to the dentate line and covered by insensate anorectal mucosa
  - Combined internal and external hemorrhoids

References

1. Juan L Poggio, MD, MS, FACS, FASCRS; Chief Editor: John Geibel, MD, DSc, MA : Fistula-in-Ano . eMedicine of Medscape


3. Mark L. Welton, MD, Carlos E. Pineda, MD, George J. Chang, MD, Andrew A. Shelton, MD. CURRENT Work Up & Treatment and Management: Surgery, 13e , 2010

Clinical Presentation

- **Symptoms**
  - Bright red bleeding per rectum and mucus discharge
  - When very large, a sense of rectal fullness or discomfort
  - Pain during bowel movements (when thrombosed)
  - Anal Itching
  - Thrombosed external hemorrhoid
    - Acute severe perianal pain
    - The pain usually peaks within 48–72 hours
    - It is a purple-black, edematous, tense subcutaneous perianal mass that is quite tender.
    - Ischemia and necrosis of the overlying skin, resulting in bleeding

- **Signs:** purple-colored swelling protruding through anus

- **Complications**
  - Incarceration, thrombosis and necrosis
  - Severe bleeding
  - Painful lumps in the anal area

Classification

- Internal hemorrhoids are graded according to the extent of prolapse
  - First-degree: bulge into the anal canal and may prolapse beyond the dentate line on straining
  - Second-degree: prolapse through the anus but reduce spontaneously
  - Third-degree: prolapse through the anal canal and require
manual reduction
- Fourth-degree hemorrhoids: prolapse but cannot be reduce.

**Differential Diagnosis**

- Perianal hematoma
- Perianal abscess
- Rectal prolapse
- Anal papilla

**Work Up**

- Rectal Examination: visual and digital
- Anaoscopy, proctoscopy
- Colonoscopy: to rule out colonic pathology especially > 40 years.

**Treatment and Management**

(See flowchart 6)

- 1st to 3rd degree (See Table 4)
  - Medical therapy: diet/lifestyle modification, flavonoid, sitz bath and stool softener
  - Surgical therapy: banding or sclerotherapy and hemorrhoidectomy
- 4th degree: hemorrhoidectomy
Thrombosed external hemorrhoid
  • Medical therapy and analgesia
  • Incision and evacuation of clot

**Red Flags**

- Weight loss, change in stool size and bowel habit
- Abdominal pain, anorexia or unexplained anemia
- Family History of bowel cancer or inflammatory bowel disease
- Elderly, HIV patients

**References**


2- Mark L. Welton, MD, Carlos E. Pineda, MD, George J. Chang, MD, Andrew A. Shelton, MD. CURRENT Work Up & Treatment and Management: Surgery, 13e, 2010

3- Maxine A. Papadakis, MD, Stephen J. McPhee, MD. Quick Medical Work Up & Treatment and Management, 2013

Flowchart 6: Anorectal Diseases

Suspecting anorectal disease based on history and examination

Anoscopy (If not painful)

- Hemorrhoid
  - 1st, 2nd and 3rd degree
  - Medical therapy: *Diet/lifestyle modification* *Flavonoid* *Sitz bath* *Stool softener*
  - Failed
    - Banding and sclerotherapy
    - Failed
      - Hemorrhoidectomy
  - 4th degree

- Fistula
  - 4th degree
  - Medical therapy: *Incision and drainage* *Cefruxime and metronidazole*
  - Fistulotomy
  - Failed
    - Seton
      - MRI
      - Colorectal referral

- Abscess
  - *Incision and drainage* *Cefruxime and metronidazole*

- Fissure
  - Chronic
    - Lateral partial internal sphincterotomy
  - Acute
    - Medical therapy: Stool softener Sitz bath Local topical nifedipine

Red flag presentation
Crohn’s, anterior type in female, complex, high

No
- No
- Yes

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Pilonidal Disease

Overview

- Definition
  - Acute/chronic recurring abscess or chronic draining sinus over the sacrococcygeal or perianal region

- Incidence: most commonly in people aged 15-30 years

- Etiology
  - Unknown. It is speculated that the cleft creates a suction that draws hair into the midline pits when a patient sits

Clinical Presentation

- Symptoms
  - A painful, fluctuant mass in the sacrococcygeal region with purulent discharge

- Signs
  - Abscess: red, warm, local tenderness and fluctuation with or without induration
  - Sinus: Midline natal cleft pit sinus
    - Chronic draining sinuses with multiple mature tracts with hairs protruding from the pitlike openings
Work Up

- It is a clinical Work Up best elicited by history and physical examination findings

Treatment and Management

- Abscess: incision and drainage with antibiotic therapy
- Sinus: excision (without closure or with modified closure of skin).

References


2-Maxine A. Papadakis, MD , Stephen J. McPhee, MD. Quick Medical Work Up & Treatment and Management , 2013

3-Mark L. Welton, MD, Carlos E. Pineda, MD, George J. Chang, MD, Andrew A. Shelton, MD. CURRENT Work Up & Treatment and Management: Surgery, 13e , 2010

Lower GI Bleeding

Overview

- Definition: bleeding distal to ligament of Treitz

- Etiology
  - Upper GI source
  - Anorectal disease: fissure and hemorrhoid
  - Diverticular disease
  - Vascular: angiodysplasia
  - Neoplasm: benign (polyp) or malignant
  - Inflammation: ulcerative colitis, infectious, radiation, ischemic

Clinical Presentation

- Hematochezia, anemia, occult blood in stool and rarely melena

Treatment and Management

(See flowchart 7)

- Initial management
  - Resuscitation and monitoring (2 large bore IV line, fluid, urinary catheter)
  - Send blood for CBC, cross and type, coagulation profile, electrolytes, liver and kidney function

- Localization
  - Insert NGT to rule out upper GI source if suspected.
• Proctoscopy
• Colonoscopy for mild to moderate bleed
• RBC scan for stable patient with massive bleeding
• Angiogram for unstable patient with massive bleeding

○ Definitive management
  • Colonoscopy and angiogram
  • Surgery rarely indicated if conservative treatment fails.

Reference


Flowchart 7: Lower GI Bleeding

Suspecting Lower GI bleeding based on history and examination

**Initial Management**
- ABC + IV line resuscitation
- CBC, Chemistry, liver function, coagulation
- NGT for rule out upper GI source
- Anoscopy to rule out anorectal source

- **Red flag presentation**
  - Massive bleeding

  - No
    - Minimal to moderate bleeding
    - Colonoscopy

  - Yes
    - Stable
      - No
        - Angiogram
      - Yes
        - RBC Scan
          - No
            - Failed
            - Enteroscopy failed
            - Surgery
Chapter 3

HEPATOBILIARY
Gall Stones

Overview

- Risk Factors
  - Cholesterol stones
    - Obesity, age <40
    - Estrogens: female, multiparity, oral contraceptive pills
    - Ethnicity: Caucasian more prevalent.
    - Terminal ileal resection or disease (e.g. Crohn’s disease)
    - Impaired gallbladder emptying: starvation, TPN, DM
    - Rapid weight loss: rapid cholesterol mobilization and biliary stasis
  - Pigment stones (contain calcium bilirubinate)
    - Cirrhosis
    - Chronic hemolysis
    - Biliary stasis (strictures, dilation, biliary infection)
    - Protective factors: statins, vitamin C, coffee

Clinical Presentation

- Asymptomatic (80%), biliary colic (10-25%), cholecystitis, choledocholithiasis (8-15%), cholangitis, gallstone pancreatitis and gallstone ileus

Work Up
o Ultrasound: diagnostic procedure of choice

o Other modalities of investigations in special cases e.g. CT, MRCP, ERCP, HIDA scan

Treatment and Management

o No need for Treatment and Management in most of the cases

o Indication of cholecystectomy if: increased risk of malignancy (choledochal cysts, Caroli’s disease, porcelain (calcified) gallbladder), sickle cell disease, pediatric patient, bariatric surgery, diabetes, immunosuppression

Reference


Acute Cholecystitis

Overview

- Pathogenesis
  - Inflammation of gallbladder resulting from sustained gallstone impaction in cystic duct or Hartmann’s pouch
  - No cholelithiasis in 5-10%

Clinical Presentation

- Symptoms
  - Often have history of biliary colic
  - Severe constant (hours to days) epigastric or RUQ pain, anorexia, nausea and vomiting,

- Signs
  - Fever: usually <38.5°C but it goes higher in the presence of complication
  - Focal peritoneal findings: Murphy’s sign, palpable, tender gallbladder (in 33%)

- Complications
  - Mucocele: long term cystic duct obstruction results in mucous accumulation in gallbladder.
  - Gangrene (20%) and perforation (2%): result in abscess formation or peritonitis
  - Empyema of gallbladder: suppurative cholecystitis, (pus inside gallbladder)
  - Cholecystoenteric fistula, from repeated attacks of
cholecystitis. It can lead to gallstone ileus

• Emphysematous cholecystitis: bacteria produce gas inside gallbladder lumen, wall or pericholecystic space (more in diabetic patient)

• Mirizzi syndrome: extra-luminal compression of CBD/CHD due to large stone in cystic duct, which can create a fistula between gallbladder and common bile duct.

Differential Diagnosis

- Hepatobiliary: hepatitis, cholangitis, pancreatitis
- Peptic ulcer disease
- Basal pneumonia
- Renal: pylonephritis
- Cardiac: angina or myocardial infarction.

Work Up

- Laboratory: leukocytosis and left shift, mildly elevated bilirubin, AST, ALT and ALP

- Abdominal ultrasound: 98% sensitive, consider HIDA scan if U/S equivocal
Treatment and Management

(See flowchart 8)

- Admission: hydrate, NPO, NG tube (if persistent vomiting from associated ileus) and analgesics

- Antibiotics: to cover E. coli, Klebsiella, Enterococcus and Clostridium (See Table 5)

- Cholecystectomy
  - Laparoscopic is standard of care.
  - Percutaneous cholecystostomy tube: for critically ill or inoperable patients.

Red Flags

- Asymmetric thickening of gall bladder wall on u/s (suspect malignancy).

- Diabetic or immunocompromized patient

- Male with chronic recurrent attackes

- Peritonitis or shock

Reference


Flowchart 8: Acute Cholecystitis

Suspecting acute cholecystitis based on history and examination

Initial management
ABC, IV line
CBC, LFT, Bilirubin, amylase
Abdominal ultrasound

Red Flag Presentation
Early presentation (within 72 hrs from onset), diabetic, peritonitis or/and shock

Yes

Preoperative antibiotics
Cefuroxim + metronidazole
Laparoscopic cholecystectomy
or cholecystostomy tube for poor surgical candidate

Conservative therapy
Cefuroxim + metronidazole
NPO
Monitor for deterioration
Clinical + laboratory

No

Operative finding
Gangrenous/perforated gallbladder, Intra-abdominal sepsis (IAS)

In case of deterioration
Yes

Continue same antibiotics for 24hrs

*Continue same antibiotics for 7-10 days for mild-moderate IAS
*Consider pip/taz in severe IAS

Stabilized

Internal cholecystectomy

Choledocholithiasis

Overview

- Definition: stones in common bile duct (CBD)

- Etiology: Primary vs. secondary stones
  - Primary: formed in bile duct, indicates bile duct pathology (e.g. benign biliary stricture, sclerosing cholangitis, choledochal cyst, cystic fibrosis)
  - Secondary: formed in gallbladder (most common)

Clinical Presentation

- Symptoms
  - 50% are asymptomatic. Often has history of biliary colic

- Signs
  - Tenderness in RUQ or epigastrium
  - Pale stool, dark urine and fluctuating jaundice

- Complications
  - Cholangitis (pain, jaundice and fever), pancreatitis, biliary stricture and biliary cirrhosis

Work Up

- Laboratory
  - CBC: usually normal; leukocytosis suggests cholangitis
  - LFT: increased AST, ALT early in disease. Increased bilirubin (direct > 50% of the total bilirubin, more sensitive),
ALP and GGT
• Amylase/lipase: to rule out biliary pancreatitis

Imaging
• Ultrasound: intra/extra-hepatic duct dilatation
• MRCP (90% sensitive, almost 100% specific, not therapeutic)
• ERCP or PTC: diagnostic and therapeutic

Treatment and Management
(See Flowchart 9)

Antibiotics (See Table 5)

ERCP for CBD stone extraction followed by laparoscopic cholecystectomy

Red Flags

Jaundice with RUQ pain and fever

Altered mental status or shock

Reference


Flowchart 9: Obstructive Jaundice

Suspecting obstructive jaundice based on history and examination

**Initial management**
- ABC, IV line
- CBC, chemistry, liver function, Bilirubin (total and direct), amylase, coagulation
- abdominal ultrasound

**Red flag presentation**

*Charcot’s triad* (fever, right upper quadrant pain and Jaundice)
*Reynold’s pentad* (Charcot’s + shock and change in mental status)

No
- Obstructive jaundice with NO evidence of cholangitis
  - **Prophylactic antibiotic**
  - Ceftrix and metronidazole
  - Endoscopic retrograde cholangiopancreatography (ERCP) with sphincterotomy and retrieval of stone

Yes
- Ascending cholangitis +/- septic shock
  - **Resuscitation**
  - Antibiotic: *pip-taz*
  - **Decompression**
  - Elective if respond to conservative therapy within 24hrs
  - Emergency when refractory
  - Methods:
    - ERCP → failed → PTC →
    - failed or not available →
    - CBD exploration + T-tube

Acute Pancreatitis

Overview

○ Definition
• Acute inflammatory non-infectious process, ranging from mild parenchymal edema to severe hemorrhagic pancreatitis, leading to necrosis

○ Etiology
• Gallstone (40-60%), alcohol (20-30%), idiopathic, trauma, medication, and iatrogenic (ERCP)

○ Pathophysiology
• Intracellular activation of trypsinogen to trypsin leads to activation of other proenzymes and cellular auto-digestion causing a systemic inflammatory response.

Clinical Presentation

○ Symptoms
• Pain (epigastric pain radiating to back), nausea and vomiting, ileus,
• Pain worse when supine, better when sitting forward
• Rarely may have coexistent cholangitis or pancreatic necrosis

○ Signs
• Fever, signs of shock, may present with jaundice
• Upper abdomen is tender, most often without guarding, rigidity, or rebound
• There may be distention and absent bowel sounds from
paralytic ileus

- Complications
  - Local
    - Pseudocyst: collection of pancreatic secretions >4 wks old surrounded by a defined wall of granulation tissue
    - Abscess/infection, necrosis
    - Splenic, mesenteric and portal vessel thrombosis or rupture
    - Pancreatic ascites and pancreatic pleural effusion
    - Diabetes mellitus
  - Systemic
    - ARDS, sepsis and multi-organ failure
    - Coagulopathy, DIC and hypocalcemia

**Differential Diagnosis**

- Hepatobiliary: hepatitis, cholecystitis, cholangitis
- Peptic ulcer disease
- Aortic dissection
- Beware of “crossover” diseases like
  - Inferior myocardial infarction (MI) causing epigastric pain
  - Right lower lobe pneumonia presenting with RUQ pain

**Work Up**

- Laboratory
  - CBC: Leukocytosis
• High amylase/lipase
• Elevated ALT (> 150 IU /L) and AST strongly suggest gallstone etiology of pancreatitis

○ Imaging
  • Abnormal x-ray: sentinel loop (dilated jejunum), colon cutoff
  • Ultrasound may show multiple stones (may have passed spontaneously), edematous pancreas
  • CT scan: peri-pancreatic inflammation, pseudocyst, phlegmon, abscess and pancreatic necrosis (non beneficial if done after initial symptoms 64,24-48 hours).

**Treatment and Management**

(See flowchart 10)

○ Ranson’s criteria for determining prognosis of acute pancreatitis

○ Simple pancreatitis: fluid resuscitation and supportive measures

○ Complicated pancreatitis: antibiotics (See Table 5) and drainage / debridement

○ Surgical debridement must be avoided early in the course of severe pancreatitis as it entails high mortality.
Red Flags

- Associated jaundice (may need urgent ERCP) ascites or shock
- Palpable abdominal mass (abscess or pseudocyst)

Reference

1- Gerard M. Doherty, MD, Lawrence W. Way, MD. CURRENT Work Up & Treatment and Management: Surgery, 13e , 2010


3- Maxine A. Papadakis, MD , Stephen J. McPhee, MD. Quick Medical Work Up & Treatment and Management , 2013


Flowchart 10: Acute Pancreatitis

Suspecting pancreatitis based on history and examination

Initial management
ABC, IV line, CBC, chemistry, amylase
Abdominal X-ray + ultrasound
CT abdomen (when suspect complication like abscess, necrosis, bleeding etc.)
ICU for severe pancreatitis

PANCREAS
Pain control: narcotic
Morphine, pethidine
Adequate resuscitation as early as possible (within 24hrs from onset of pain)
Nutritional support (Enteral preferable)
Cholecystectomy (when attack resolves) and
Cholangiogram (ERCP-when there is picture of cholangitis)
Reassessment of response
Electrolyte correction
Antibiotic
In infected necrosis or abscess
Stomach (NPO +NGT) decompression

Antibiotic
Meropenem
Percutaneous drainage
For abscess
Surgery
Necrosectomy: wait for 4 weeks for necrosis to demarcate

Yes
Complicated pancreatitis (infected necrosis, abscess)
No

Chapter 4

HERNIA
Overview

- **Definition:** protrusion of an abdominal content through an abnormal fascial defect into an area in which it is not normally contained.

- **Epidemiology**
  - More common in males than females for groin hernias.
  - Lifetime risk of developing a hernia: males 20-25%, females 2%.
  - 50% are indirect inguinal hernia, 25% are direct inguinal hernia, and 5% are femoral.

- **Risk Factors**
  - Activities which increase intra-abdominal pressure like obesity, chronic cough, pregnancy, constipation, straining on urination or defecation, ascites, and heavy lifting.
  - Congenital abnormality (e.g., patent processus vaginalis).
  - Previous hernia repair.

- **Classification**
  - Complete vs. incomplete: complete vs. partial protrusion through the defect.
  - Reducible vs. irreducible (incarcerated).
  - Richter’s hernia
    - Only part of the circumference of the bowel (usually the anti-mesenteric border) is incarcerated or strangulated so may not be obstructed.
    - A strangulated Richter’s hernia may self-reduce and thus be overlooked, leaving a gangrenous segment. This segment is at risk of perforation.
• Sliding hernia
  ▪ Part of wall of hernia is formed by protruding viscus (usually cecum, sigmoid colon or bladder)
• Anatomical classification
  ▪ Groin
• Indirect and direct inguinal, and femoral
• Pantaloon: combined direct and indirect hernias, peritoneum draped over inferior epigastric vessels
  ▪ Epigastric: defect in linea alba above umbilicus
  ▪ Incisional: ventral hernia at site of wound closure, may be secondary to wound infection
  ▪ Other: Littre’s (involving Meckel’s diverticulum), Amyand’s (containing ruptured appendix), lumbar and obturator hernia
  ▪ Parastomal, umbilical and Spigelian (ventral hernia through linea semilunaris)

Clinical Presentation

○ Symptoms
  • Mass of variable size, which gets worse at the end of the day and relieved with supine position or with reduction

○ Signs
  • Reducible mass with positive cough impulse

○ Complications:
  • Incarceration (irreducible)
  • Obstruction
  • Strangulation
Differential Diagnosis

- Inguinal hernia
  - Lymph node: lymphadenitis, lymphoma, metastatic cancer
  - Hydrocele, undescended testes
  - Vascular: pseudoaneurysm, saphena varix
  - Infection: psoas abscess, infected hematoma
  - Femoral hernia

- Umbilical hernia
  - Urachal cyst
  - Pilonidal disease.
  - Epigastric hernia
  - Primary of secondary abdominal wall neoplasm

- Incisional hernia
  - Diastasis recti, wound hematoma or seroma

Work Up

- Physical examination usually sufficient

- Ultrasound to confirm Work Up and rule out other causes

- CT scan: CT required for obturator hernias, internal abdominal hernias, Spigelian and femoral hernias in obese patients)
Treatment and Management
(See Flowchart 11)

- Prophylactic antibiotics (See Table 6)
- Elective repair: Simple, reducible
- Emergency: Incarcerated, obstructed and strangulated hernias.
- Open vs. laparoscopy

Red Flags

- Very painful, irreducible, bowel obstruction or sepsis
- History of previous cancer
- Pulsatile swelling,
- Very tender, negative cough impulse, overlying skin changes, abdominal distention

Reference


Suspecting inguinal hernia based on history and examination

Red Flag Presentation
Incarceration, bowel obstruction, strangulation, sepsis

No

Simple reducible

Elective repair vs watchful waiting (elderly, large defect)

Yes

Initial management
ABC, IV line, fluid
CBC, chemistry, ABG
Abdominal x-ray

Admit for OR
Antibiotic (cefuroxime and metronidazole)
Groin exploration
Relief obstruction
Give 100% O2 and cover bowel with warm saline and assess

Bowel viable, healthy

Return the bowel and perform tension-free repair with mesh

No

Resection and anatomical repair of hernia
Cellulitis and Erysipelas

Overview

○ Definition
  • Skin infections that develop as a result of bacterial entry though break in the skin barrier
  • Erysipelas involves the upper dermis and superficial lymphatics
  • Cellulitis involves the deeper dermis and subcutaneous fat

○ Incidence: about 200 cases per 100,000 patient-years

○ Etiology
  • Most common pathogens are beta-hemolytic streptococci, then Staphylococcus aureus
  • Gram negative bacilli and anaerobes in a minority of cases, especially in diabetic patients

Clinical Presentation

○ Symptoms: pain, swelling and fever

○ Signs
  • May present with fever
  • Areas of skin erythema, edema, and warmth with pain.
  • Erysipelas lesions are raised above the level of surrounding skin, and there is a clear line of demarcation between involved and uninvolved tissue.
  • While the edges in cellulitis are diffuse and vague
  • The lower extremities are the most common site of infection
for both erysipelas and cellulitis
• May present with lymphangitis and inflammation of regional lymph nodes

Treatment and Management

(See flowchart 12)

- Overview therapy
  • Elevation of the affected area facilitates gravity drainage of edema and inflammatory substances
  • Treatment and Management of underlying conditions (diabetes, any dermatological disease)
  • The skin should be sufficiently hydrated to avoid dryness
  • Analgesia and antipyretic: paracetamol 1 g PO q8h

- Antibiotics (See Table 7)
  • Cellulitis without associated purulent drainage or abscess
    - If mild and no need for hospital admission, Outpatient Treatment and Management recommendations:
      - Amoxicillin-clavulanate (augmentin) 625 mg PO BID for 10 days
      - If patient needs admission, Inpatient Treatment and Management recommendations:
  • Crystalline Penicillin 4 millions units q6h or Cefuroxime
    - Broad-spectrum antibiotic in diabetic patient
    - Cellulitis with purulent drainage and/or abscess
    - Admission to hospital
    - If an abscess is present, drainage will be required for Treatment
and Management

- Send swab for culture and sensitivity
- Augmentin 1.2 g IV q8h or cefuroxime and metronidazole, then shift to oral on discharge
- In severe or resistant cases: start Tazocin (Piperacillin-tazobactam) 4.5 g IV q8h for 1-3d; then change according to the result of the culture and sensitivity

Red Flags

- Diabetic patient, shock.
- Severe pain out of proportion to exam.
- Bruises, necrotic area and subcutaneous air

Reference


2-Larry M Baddour, Daniel J Sexton, MD, Sheldon L Kaplan, uptodate Literature review, 2013

Diabetic Septic Foot

Overview

- Diabetic foot infections are associated with significant morbidity and mortality
- Risk factors: neuropathy, angiopathy (peripheral vascular disease), and poor blood sugar control.
- Forms: Cellulitis, Deep-skin and soft-tissue infections, Acute osteomyelitis or Chronic osteomyelitis
- Etiology
  - Most diabetic foot infections are polymicrobial
  - The microbiology of diabetic foot wounds is variable depending on the extent of involvement
  - Superficial diabetic foot infections: due to aerobic gram-positive cocci (including S. aureus, S. pyogenes)
  - Ulcers that are deep, chronically infected are more likely to be polymicrobial. Such wounds may involve the above organisms in addition to enterococci, enterobacteriaceae, pseudomonas aeruginosa, and anaerobes
  - Wounds with extensive inflammation, necrosis, pus drainage, or gangrene with signs of systemic toxicity should be presumed to have anaerobic organisms in addition to the above pathogens. Potential pathogens include anaerobic streptococci, bacteroides species, and clostridium species
Work Up

- Laboratory
  - CBC: leukocytosis
  - Chemistry: renal function may impaired due to sepsis

- Foot x-rays: osteomyelitis, destructed bone, atherosclerotic vessels, gas in subcutaneous tissue in cases of gas gangrene

Treatment and Management

(See Flowchart 12)

Overview

- Careful wound management
- Nutrition support, blood sugar control
- Fluid and electrolyte management
- Appropriate antibiotic
- Surgical debridement

- Antibiotics therapy (See Table 7)
  - Empiric antibiotic should be started then modified according to culture
  - Mild to moderate infection
    - One of the following antibiotics for 10-14 days: Augmentin (monotherapy).
    - Cefuroxime or Ciprofloxacin or ceftriaxone with metronidazole is an alternative
    - Vancomycin for MRSA
  - Moderate to severe infection: Tazocin or meropenem
Surgical debridement is necessary for eradication and control of infections. Revascularization (via angioplasty or bypass grafting) and/or amputation may be necessary.

Wound management:
- Debridement of callus and necrotic tissue.
- Frequent dressing with gauze and other materials that include gels, hydrocolloids, honey, and antiseptics containing iodine or silver salts.
- Wound swab for culture and sensitivity.

Red Flags

- Surgery
- Pain out of proportion to physical examination
- Sepsis and shock
- Very high WBC or leucopenia

Reference


Surgical Site Infection

Overview

Epidemiology

- Surgical site infection (SSI) is the common cause of nosocomial infection in surgical patients
- SSI is associated with significant morbidity and mortality, prolonged hospital stay, and increased cost

Definition

- Infection related to an operative procedure that occurs at or near the surgical incision within 30 days of the procedure, or within one year if prosthetic material is implanted at surgery

Classification of SSI

- Incisional: divided into
  - Superficial: involve only the skin or subcutaneous tissue outer to the fascia
  - Deep: involve deep soft tissue of an incision inner to the fascia
- Organ or space infection: may involve any part of the operative field (other than the incision) that was manipulated during the operative procedure

Overview classification of wound

- Clean, clean-contaminated, contaminated and dirty wounds
- SSI rates according to wound class were
  - Clean is 1 to 2 %, clean-contaminated is 2 to 7 %, contaminated is 6 to 15 % and dirty wound can reach up to 40 %
Clinical Presentation

- **Symptoms**
  - Pain, swelling and fever. It can present with discharge

- **Signs**
  - Fever and signs of infection (warm, red, tender, swollen area)

Differential Diagnosis

- Wound dehiscence, enterocutaneous fistula, seroma, hematoma

Work Up

- Clinically and by imaging (ultrasound and/or CT scan)

Treatment and Management

(See Flowchart 12)

- Antibiotics (See Table 7)
- Open the wound and drain the pus for incisional SSI
- Percutaneous drainage for deep space infection
Reference


Flowchart 12: Skin and Soft Tissue Infection

Suspecting Skin/soft tissue infection based on history and examination

Initial management
ABC, IV line
CBC, chemistry, coagulation
X-ray

Red Flag Presentation
Diabetic, signs of necrotizing fascitis or/and shock

Yes
Broad-spectrum antibiotics
Ceftriaxone + metronidazole
Or Pip/Taz
Procedure
Urgent debridement for necrotic tissue
Drainage of abscess

No
Broad-spectrum antibiotics
Cefuroxime + metronidazole

Surgical site infection (SSI)
Superficial → Open the wound
Deep → Percutaneous drainage

Abscess
Incision and drainage
Overview

○ Epidemiology
  ● Fifth leading cause of death overall; leading cause of death for ages 1–44 years
  ● 3 peaks of death
    ▪ 1st peak (within minutes)
      ● Account for 50% of trauma-related deaths. It is due to great vessels/heart laceration or brain stem/spinal cord injury
    ▪ 2nd peak (within hours)
      ● Account for 30% of deaths. It is due to bleeding and CNS injury
      ● There hours are called the “GOLDEN HOURS” because they from a window for the trauma team to save the patient
    ▪ 3rd peak (from 1 day to weeks)
      ● Account for 20% of deaths, which is due to Infection and multi-organ failure

Assessment of Trauma Patient

○ Primary survey
  ● ABCDE
    ▪ Airway and alignment of C-spine
    ▪ Indication for intubation (summarized in ABCDE)

○ Airway protection

○ Breathing insufficiency

○ Control \( \text{PaO}_2 \) and \( \text{PaCO}_2 \)
o Disaster prevention (for anticipated airway obstruction)

o Event (for transportation to another hospital)
  ▪ Breathing
    • Look, listen and measure O₂ saturation
  ▪ Circulation (Pulse, blood pressure) and control the bleeding
  • Source of bleeding is summarized in “FAST”

o Floor (external bleed)

o Abdomen and pelvis

o Skeleton or Soft tissue

o Thorax
  ▪ Disability
    • Includes: Glasgow Coma Scale (GCS: 3-15), pupil reflex, AVPU and lateralization


  ▪ Exposure
    • Do not forget the back (Log rolling), avoid hypothermia
    • Adjuncts to primary survey
      ▪ 2 large bore IV line
      ▪ Tube in each orifice: gastric tube and Foley’s catheter
      ▪ Labs: CBC, chemistry, ABG and coagulation (not always needed)
• Chest and pelvic x-ray
• Focused Assessment with Sonography for Trauma (FAST)
• Cardiac monitor and ECG

• Resuscitation
  • Resuscitation with fluid (1 liter of ringer lactate) followed by blood as soon it become available
  • Correct 4 Hs
  • Hypotension
  • Hypothermia
  • Hypocoagulation (coagulopathy)
  • Hydrogen excess (acidosis)
    • Monitor response: vital signs, urine output (0.5 ml/Kg/hr) and may need invasive monitoring

○ Secondary survey and adjuncts
  • AMPLE history
    • Allergy
    • Medication
    • Past history
    • Last meal
    • Event
  • Head to toe examination
  • Specific imaging
    • Specific x-ray
    • Whole-body CT scan
  • It is the imaging of choice in trauma
    • Angiogram
- Indicated when there is leak of contrast (blush) on CT angiogram
- It has no rule in stopping venous bleed

- Reassessment
  - Always reassess
  - Monitor response: vital signs, urine output (0.5 ml/Kg/hr) and may need invasive monitoring

- Definitive Care: e.g. angiogram, surgery, transfer, etc.

- Tertiary survey (after 24hrs from admission)
  - Review all studies, repeat head-to-foot examination, review all laboratory test results for any missed injuries

References


Flowchart 13: Polytrauma-Blunt

Suspecting polytrauma based on history from paramedic/relative

Primary Survey
A: airway and alignment of C-spine
B: breathing
C: circulation and control bleeding
D: disability (GCS, pupil reflex, lateralization)
E: exposure and avoid hypothermia

Adjunct to primary survey
IV line and laboratory tests
Nasogastric/orogastric tube
Foley catheter (if no contraindication)
FAST
X-ray: chest and pelvis +/- ECG

Identify the cause!
5 possible areas:
Scalp: by examination
Chest: by chest x-ray
Abdomen: by repeating FAST
Pelvis: pelvis x-ray
Extremity and spine: by examination and x-ray

Severe mechanism
Rollover, ejection, fatality in the same car, motorcycle, pedestrian injury, fall from more than double patient’s height
Difficult to assess and examine

If stabilized
Whole-body CT Scan

If stabilized Whole-body CT Scan
No
Yes
FAST positive

Unstable Hemodynamic

Resuscitate and reassess

Resuscitate and reassess

OR

Yes

No

AMPLE history
Secondary survey
Adjuncts
Specific x-ray
CT Scan
<table>
<thead>
<tr>
<th>Medication</th>
<th>Parenteral therapy and dose</th>
<th>Proton pump inhibitors</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Omeprazole</em></td>
<td>40mg every 24 hours</td>
<td>not more than 20mg daily</td>
</tr>
<tr>
<td><em>Esomeprazole</em></td>
<td>major peptic ulcer bleeding (unexplained)</td>
<td>initial intravenous infusion of 80mg, then by continuous intravenous infusion 40mg/hour for 72 hours</td>
</tr>
<tr>
<td><em>Lansoprazole</em></td>
<td>30mg every 12-24 hours</td>
<td>use half normal dose in moderate to severe liver disease</td>
</tr>
<tr>
<td><em>Pantoprazole</em></td>
<td>40mg every 12-24 hours or 80mg IV then infusion infusion</td>
<td></td>
</tr>
<tr>
<td><em>Ranitidine</em></td>
<td>50mg every 6-8 hours</td>
<td>use half normal dose if sCr is less than 1.10 mg/dl</td>
</tr>
</tbody>
</table>

**Note:**

- Omeprazole and Lansoprazole reduce antral and effect of Clopidogrel.
- Lansoprazole and Pantoprazole possibly reduce ameliorative effect of Clopidogrel.
<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Suggested regimen</th>
<th>Alternative</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cholecystitis</td>
<td>Ceftriaxone 2g IV every 8h</td>
<td>Piperacillin-tazobactam 4.5g IV every 8h</td>
</tr>
<tr>
<td>Cholangitis</td>
<td>Metronidazole 500mg IV every 8h</td>
<td>Piperacillin-tazobactam 4.5g IV every 8h</td>
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<tr>
<td>Appendicitis</td>
<td>Ceftriaxone 2g IV every 8h</td>
<td>Metronidazole 500mg IV every 8h</td>
</tr>
<tr>
<td>Peritonitis</td>
<td>Ceftriaxone 2g IV every 8h</td>
<td>Metronidazole 500mg IV every 8h</td>
</tr>
</tbody>
</table>

**Treatment duration: 5 days**

Perform a CT scan with IV contrast at days 4-7 and begin Mepenox IV as needed.
<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Microorganism (usual)</th>
<th>Suggested regimens</th>
<th>Primary</th>
<th>Alternative</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hernia</td>
<td></td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>

- Hernia repair with or without mesh, open or laparoscope
- Ceftazidime, Meropenem
- Not routinely recommended but in immunocompromised or taking corticosteroids
- Vancomycin, Teicoplanin
- Ceftriaxone, Piperacillin/Tazobactam
### Table 7

<table>
<thead>
<tr>
<th>Compartment</th>
<th>Primary</th>
<th>Suggested Regimens</th>
</tr>
</thead>
<tbody>
<tr>
<td>Skin and soft tissue</td>
<td>Low, moderate bleeding</td>
<td></td>
</tr>
<tr>
<td></td>
<td>High bleeding</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Massive bleeding</td>
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</tbody>
</table>

<table>
<thead>
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</tbody>
</table>

### Compartment

- Low, moderate bleeding: 
  - [Details provided in text]

- High bleeding: 
  - [Details provided in text]

- Massive bleeding: 
  - [Details provided in text]

<table>
<thead>
<tr>
<th>Compartment</th>
<th>Primary</th>
<th>Suggested Regimens</th>
</tr>
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<tbody>
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</tbody>
</table>

### Compartment

- Low, moderate bleeding: 
  - [Details provided in text]

- High bleeding: 
  - [Details provided in text]

- Massive bleeding: 
  - [Details provided in text]
### Polytrauma

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dose</th>
<th>Regimen</th>
</tr>
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<tbody>
<tr>
<td>Propofol</td>
<td>2 mg/kg</td>
<td>IV every 40-60 sec</td>
</tr>
<tr>
<td>Midazolam</td>
<td>50 mg</td>
<td>IV every 24 hours</td>
</tr>
<tr>
<td>Morphine</td>
<td>0.2-0.3 mg/kg</td>
<td>IV every 4-6 hours</td>
</tr>
<tr>
<td>Fentanyl</td>
<td>1-2 mcg/kg</td>
<td>IV every 3-6 hours</td>
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</table>

**Note:** Always monitor for adverse effects and adjust doses as necessary.
<table>
<thead>
<tr>
<th>Low molecular weight heparins</th>
<th>Moderate risk</th>
<th>High risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Enoxaparin (Clexane)</td>
<td>20mg (2000 units) subcutaneous injection 24 hours before surgery, then 20mg (2000 units) subcutaneous injection every 24 hours</td>
<td>40mg (4000 units) every 24 hours</td>
</tr>
<tr>
<td>Dabigatran etexilate (Pradaxa)</td>
<td>30mg oral twice daily for at least 24 hours</td>
<td>3000 units given subcutaneously</td>
</tr>
</tbody>
</table>

**Deep-vein thrombosis prophylaxis (DVT)**

**Heparin 5000 units**

Loading dose: 5000 units given subcutaneously. This should be started within two hours of operation.

Then, every 8 or 12 hours.

**Note:**

- Heparin should be started on admission and an alternate anticoagulant such as aspirin is prescribed. Platelet count return to normal range in those who require warfarin.
- Continuous infusion of unfractionated heparin is effective if the subcutaneous route is not feasible. However, this increases the risk of major bleeding and also requires hemostatic monitoring.
<table>
<thead>
<tr>
<th>(A)</th>
<th>(B)</th>
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<tbody>
<tr>
<td>atracurium besylate</td>
<td>bacillus calmette-gue rin</td>
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<tr>
<td>abacvir sulfate + lamivudine + zid-ovudine</td>
<td>bacitrin zinc + polymixin b sulphate</td>
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<tr>
<td>acetazolam ide</td>
<td>adalimumab</td>
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<tr>
<td>acetylcholine chloride</td>
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<td>acyclovir</td>
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<td>alprostadil (prostaglandin e1) pediatric</td>
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<td>alteplase</td>
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<td>estradiol valerate</td>
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<td>etanercept</td>
<td>haloperidol</td>
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<td>ethambutol hcl</td>
<td>heparinecalcium for subcutaneous injection</td>
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<td>ethanolamine oleate</td>
<td>(heparine sodium (bovine serum heparin)</td>
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<tr>
<td>ethinyl estradiol</td>
<td>(hepatitis b vaccine (child)</td>
</tr>
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<td>ethionamide</td>
<td>homatropine</td>
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<tr>
<td>Drug Name</td>
<td>Description</td>
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<tr>
<td>-------------------------</td>
<td>-----------------------------------------------------------------------------</td>
</tr>
<tr>
<td>ethosuximide</td>
<td>human chorionic gonadotrophin</td>
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<tr>
<td>etomidate</td>
<td>human fibrinogen</td>
</tr>
<tr>
<td>etoposide</td>
<td>(human isophane insulin (nph</td>
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<tr>
<td>etravirine</td>
<td>human menopausal gonadotrophins, follicle</td>
</tr>
<tr>
<td>(F) stimulating hormone</td>
<td>+ luteinizing hormone</td>
</tr>
<tr>
<td>factor ix fraction</td>
<td>for injection, which is sterile and free of hepatitis, hiv and any other infectious disease agent</td>
</tr>
<tr>
<td>factor viii</td>
<td>(human soluble insulin (regular</td>
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<tr>
<td>fat emulsion</td>
<td>hyaluronidase</td>
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<tr>
<td>(felodipine retard</td>
<td>hydralazine hceimesilate</td>
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<td>fentanyl citrate</td>
<td>hydrochlorothiazide</td>
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<tr>
<td>ferrous salt</td>
<td>hydrocortisone</td>
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<tr>
<td>ferrous sulphate or</td>
<td>hydroxurea</td>
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<tr>
<td>fumarate + folic acid</td>
<td>filgrastim g-csf</td>
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<td>filgrastim g-csf</td>
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<td>fludarabine phoaphate</td>
<td>hyocine butylbromide</td>
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<td>fludrocortisones acetate</td>
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<td>flumazenil</td>
<td>ibuprofen</td>
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<td>fluorescein</td>
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<td>Drug Name</td>
<td>Description</td>
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<tr>
<td>fluoxetine</td>
<td>imidazole derivative</td>
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<td>flupenthixol</td>
<td>imipenem + cilastatin</td>
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<tr>
<td>fluphenazine decanoate</td>
<td>imipramine hcl</td>
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<td>(indapamide (sustaind release</td>
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<tr>
<td>fluticasone</td>
<td>indinavir</td>
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<td>fluvoxamine malate</td>
<td>indomethacin</td>
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<td>follitropin</td>
<td>infliximab</td>
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<td>formoterol + budesonide turbuhaler</td>
<td>influenza virus vaccine</td>
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<td>foscarnet</td>
<td>injectable polio vaccines (ipv) (salk (vaccine</td>
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<tr>
<td>fosinopril</td>
<td>insulin aspart</td>
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<td>insulin glargine</td>
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<td>(G)</td>
<td>insulin lispro</td>
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<td>ganciclovir</td>
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<td>gemcitabine</td>
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<td>isoniazid</td>
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<td>meloxicam</td>
<td>isoprenaline hcl (isoproterenol hcl)</td>
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<td>isosorbide dinitrate</td>
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<td>isosorbide dinitrate</td>
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<td>Drug Name</td>
<td>Medication</td>
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<td>meningococcal polysaccharide sero group (a,c,y,w-135)</td>
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<tr>
<td>mercaptopurine</td>
<td>itraconazole</td>
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<td>ivabradine</td>
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<td>metformin hcl</td>
<td>kanamycin</td>
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<tr>
<td>methadone hcl</td>
<td>kaolin + pectin</td>
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<tr>
<td>methotrexate</td>
<td>ketamine hcl</td>
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<tr>
<td>methoxsalen + ammidine</td>
<td>ketoconazole</td>
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<td>methoxy polyethylene glycol-epoetin beta</td>
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<td>methyldopa</td>
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<td>methylerrgonovine maleate</td>
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<td>lamotrigine</td>
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<td>latanoprost</td>
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<td>l-carnitine</td>
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<td>leflunomide</td>
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<td>lenalidomide</td>
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<td>letrozole</td>
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<td>Leucovorin calcium</td>
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<td>leuprolid depo acetate</td>
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<td>Drug Name</td>
<td>Substance</td>
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<tr>
<td>minocycline hcl</td>
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<tr>
<td>mirtazapine</td>
<td>levofoaxacin</td>
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<tr>
<td>misoprostol</td>
<td>levothyroxine sodium</td>
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<tr>
<td>mitomycin</td>
<td>lidocaine + fluorescein sodium</td>
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<tr>
<td>mitoxantrone hydrochloride</td>
<td>Lidocaine hcl</td>
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<tr>
<td>mixed gas gangrene antitoxin</td>
<td>linezolid</td>
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<tr>
<td>moclopemide</td>
<td>liquid paraffin</td>
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<tr>
<td>mometasone furoate</td>
<td>lisinopril</td>
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<tr>
<td>montelukast sodium</td>
<td>lithium carbonate</td>
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<tr>
<td>orphine sulphate</td>
<td>lomustine</td>
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<tr>
<td>moxifloxacin hydrochloride</td>
<td>Loperamide hcl</td>
</tr>
<tr>
<td>ultienzyme (pancreatic enzymes:pro-</td>
<td>lopinavir + ritonavir</td>
</tr>
<tr>
<td>tease200-600u;lipase5,000-10,000u and</td>
<td></td>
</tr>
<tr>
<td>amylase5,000-10,000u)/capsule or enteric</td>
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<tr>
<td>coated tablet</td>
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<tr>
<td>multivitamins</td>
<td>lorazepam</td>
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<td>mupirocin</td>
<td>losartan potassium</td>
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<td>muromonab-cd3</td>
<td>lubricant</td>
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<td>mycophenolate mofetil</td>
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<td>(N)</td>
<td>magnesium oxide</td>
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<tr>
<td>nafarelin</td>
<td>mannitol</td>
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<tr>
<td>nalbuphine hcl</td>
<td>maprotilline hcl</td>
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<td>naloxone hcl</td>
<td>measles vaccine</td>
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<tr>
<td>naphazoline</td>
<td>mebendazole</td>
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<tr>
<td>Naproxene</td>
<td>mebeverine hcl</td>
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<tr>
<td>Natalizumab</td>
<td>mechlorethamine hcl</td>
</tr>
<tr>
<td>natamycin</td>
<td>meclozine + vitamine B6</td>
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<tr>
<td>Drug Name</td>
<td>Drug Name</td>
</tr>
<tr>
<td>---------------------------------</td>
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<tr>
<td>phenylephrine hcl</td>
<td>nateglinide</td>
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<td>phenytoin sodium</td>
<td>nelfinavir</td>
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<td>phosphate enema</td>
<td>neomycin sulphate</td>
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<tr>
<td>phosphate salt</td>
<td>neostigmine methylsulphate</td>
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<td>phytomenadione</td>
<td>niclosamide</td>
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<tr>
<td>pilocarpine</td>
<td>nicotine (24-hour effect dose)</td>
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<tr>
<td>pioglitazone</td>
<td>nifedipine retard (modified release)</td>
</tr>
<tr>
<td>piperacillin + tazobactam</td>
<td>nilotinib</td>
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<tr>
<td>plasma protein solution</td>
<td>nimodipine</td>
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<tr>
<td>pneumococcal polyvalent (23 valent) vaccine</td>
<td>nitrazepam</td>
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<tr>
<td>poliomyelitis vaccine live oral: (sabin strain)</td>
<td>nitrofurantoin</td>
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<td>polyacrylic acid</td>
<td>nitroglycerin</td>
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<tr>
<td>polyethylene glycol,3350-13.125g oral powder, sodium bicarbonate 178.5mg,sodium chloride350mg, potassium chloride 46.6mg/sachet</td>
<td>isosorbide dinitrate</td>
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<td>polymyxin b sulphate + neomycin sulphate + hydrocortisone</td>
<td>non sedating antihistamine tablet (cetirizine or noratadine)</td>
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<tr>
<td>polystyrene sulphate resins (calcium)</td>
<td>noradrenalin acid tartrate</td>
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<td>pramipexole</td>
<td>norfloxacin</td>
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<td>pravastatin</td>
<td>nystatin</td>
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<td>praziquantel</td>
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<tr>
<td>prazosin hcl</td>
<td>octreotide</td>
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<tr>
<td>prednisolone</td>
<td>ofloxacin</td>
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<tr>
<td>Drug Name</td>
<td>Description</td>
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<tr>
<td>-----------------------------------------------</td>
<td>--------------------------------------------</td>
</tr>
<tr>
<td>pregabalin</td>
<td>oily phenol injection</td>
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<tr>
<td>Prilocaine + felypressin</td>
<td>olanzapine</td>
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<tr>
<td>Primaquine phosphate</td>
<td>olopatadine hcl</td>
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<tr>
<td>Primidone</td>
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<tr>
<td>Procainamide hcl</td>
<td>ondansetron</td>
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<tr>
<td>Procarbazine</td>
<td>orienograftim (g-csf)</td>
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<tr>
<td>Procyclidine hydrochloride</td>
<td>oxaliplatin</td>
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<td>Progesterone</td>
<td>oxybuprocaine</td>
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<td>Proguanil hcl</td>
<td>oxybutynin hcl xl</td>
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<td>Promethazine hcl</td>
<td>oxymetazoline</td>
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<td>proparacaine</td>
<td>oxytocin</td>
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<td>propylthiouracil</td>
<td>paclitaxel</td>
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<tr>
<td>Propranolol hcl</td>
<td>paliperidone</td>
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<tr>
<td>Protamine sulfate</td>
<td>palivizumab</td>
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<tr>
<td>prothionamide</td>
<td>pancuronium bromide</td>
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<td>Protirelin (thyrotrophin-releasing hormone,thr)</td>
<td>pantoprazole sodium sesquihydrate</td>
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<td>Pseudoephedrine hcl 30mg + anti-histamine</td>
<td>papaverin</td>
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<td>Pumactant phospholipid</td>
<td>para-amino salicylate sodium</td>
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<td>Pura aluminum hydroxide</td>
<td>paracetamol</td>
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<td>Pyrazinamide</td>
<td>pegaspargase</td>
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<td>Pyrethrins</td>
<td>pegylated interferon alpha 2a</td>
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<td>Pyridostigmine</td>
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<tr>
<td>Pyridoxine hcl (vitamine b6)</td>
<td>penicillamine</td>
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<tr>
<td>Drug Name</td>
<td>Description</td>
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<tr>
<td>Pyrimethamine</td>
<td>penicillin benzathine (penicillin g)</td>
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<tr>
<td>Prilocaine + felypressin</td>
<td>pentamidine isethionate</td>
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<td>primaquine phosphate</td>
<td>pentavalent vacc.(hbv+hib+dtp)</td>
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<td>(Q)</td>
<td>pentoxifylline</td>
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<td>quetiapine</td>
<td>perindopril</td>
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<td>quinidine sulfate</td>
<td>permethrin</td>
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<td>quinie sulphate</td>
<td>phenobarbital (phenobarbitone)</td>
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<tr>
<td>(R)</td>
<td>phenoxy methyl penicillin (penicillin v potassium)</td>
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<tr>
<td>rabies immunoglobulin for i.m</td>
<td>phentolamine mesylate</td>
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<tr>
<td>injection</td>
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<tr>
<td>stibogluconate sodium (organic</td>
<td>rabies virus vaccine</td>
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<tr>
<td>pentavalent antimony)</td>
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<td>ranitidine</td>
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<td>recombinant factor via</td>
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<td>sulfadoxin500mg + pyrimethamine25mg</td>
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<td>rifampicin</td>
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<td>sumatriptan succinate</td>
<td>riluzole</td>
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<td>Drug Name</td>
<td>Description</td>
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<tr>
<td>(T) ringer’s lactate solution</td>
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<td>rivaroxaban</td>
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<td>tenofovir disoproxil fumurate</td>
<td>ropivacaine hcl</td>
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<td>rose bengal</td>
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<td>teriparatide</td>
<td>rosvastatin</td>
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<td>salbutamol</td>
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<td>tetanus immunoglobulin for i.m injection</td>
<td>salmeterol + fluticasone propionate</td>
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<td>scorpion anti – venin</td>
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<td>sevoflurance</td>
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<td>thioguanine</td>
<td>simethicone</td>
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<td>simvastatin</td>
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<td>tigecycline</td>
<td>sirolimus</td>
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<td>timolol</td>
<td>sitagliptin phosphate</td>
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<td>tinzaparin sodium</td>
<td>snake anti-venin</td>
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<tr>
<td>Drug Name</td>
<td>Component</td>
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<td>----------------------------------------------</td>
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<td>tiotropium</td>
<td>sodium acetate</td>
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<tr>
<td>tirofiban hydrochloride</td>
<td>sodium aurothiomalate</td>
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<tr>
<td>tobramycin + dexamethasone</td>
<td>sodium bicarbonate</td>
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<tr>
<td>tobramycin sulfate</td>
<td>sodium chloride</td>
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<tr>
<td>tolterodine tartrate</td>
<td>sodium cromoglycate</td>
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<tr>
<td>topiramate</td>
<td>sodium hyaluronate</td>
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<tr>
<td>trace elements additive (pediatric dose)</td>
<td>sodium hyaluronate intra-articular</td>
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<tr>
<td>tramadol hcl</td>
<td>sodium nitroprussude</td>
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<td>tranexamic acid</td>
<td>sodium phosphate</td>
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<td>trastuzumab</td>
<td>sodium valproate</td>
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<td>trazodone</td>
<td>somatropin (human growth hormone)</td>
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<td>sorafenib</td>
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<td>sotalol hydrochloride</td>
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<td>vinblastine sulfate</td>
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<td>Drug Name</td>
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<td>urofollitrophine f.s.h wax removal</td>
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<td>zidovudine + lamivudine</td>
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<td>zinc sulfate</td>
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<td>vitamine B complex</td>
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</tbody>
</table>
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Illustration

Flowchart by Hassan Adnan Bukhari

Medication Table by Faisal Ahmed Al-Wdani
Acknowledgement

Great appreciation for Dr. Ghiath Al Sayed, Consultant General Surgery, King Fahad medical city, for reviewing and editing this book.