INTRA-ABDOMINAL INFECTIONS - II

PERITONITIS AND ABSCESES
I. PERITONITIS
II. INTRAPERITONEAL ABSCESS
III. VISCERAL ABSCESS

I. PERITONITIS - Inflammation of the peritoneum.
A. Most cases are endogenous in origin from organisms forming normal flora of mucous membranes lining viscera of abdominal cavity: upper GI tract, lower small bowel, large intestine, and vagina. Intact mucosa blocks invasion of organisms. Intestinal normal flora has 400 to 500 different microorganisms.
B. Infection usually polymicrobial with an average of 4 organisms (range 1 - 12).
C. Organisms: 

*Escherichia coli*

1. Virulence factors:
   a. Adherence - 20 different adherence factors have been described from *E. coli.*
   b. Endotoxin - lipopolysaccharide of outer membrane. Lipid A is the toxic portion. Released when bacteria lyse. Lysis occurs as a result of:
      1. complement complex action on membrane
      2. ingestion and killing phagocytes
      3. killing by antibiotics
Toxicity of lipid A is primarily from its ability to activate complement and to stimulate the release of bioactive proteins such as cytokines.

Both complement and the cytokines are normal protective substances against infection. However, these compounds become toxic to the patient when they are produced in too high a concentration:

- LPS activated complement ➔ tissue inflammation
- LPS activated cytokines ➔ septic shock (collapse of the circulatory system and result in multiple organ system failure).

LPS ➔ LPS-LPS-binding protein ➔ CD14 receptors on monocytes and macrophages and other receptors on other cells such as endothelial cells.

At least three types of events are triggered by interaction of LPS with patients’ cells:
- production of cytokines
- activation of complement cascade
- multiple organ system failure
2. Etiology/Pathogenesis
   a. Escape from lumen of gastrointestinal tract which leads to infections such as peritonitis or localized in abscess.
   b. Polymicrobial - generally several organisms involved in infection.

3. Clinical Identification of Organism:
   a. Gram negative bacillus, oxidase negative, growth on MacConkey Agar, lactose positive.
   b. Facultative growth
   c. Identification to species based on pattern of physiological reactions (phenotype).
   d. Identification of serotypes within species based on antigenic classification:
      - O antigen - Region 1 of LPS contains polysaccharide antigens
      - H antigen - protein antigens of flagella
      - K - extracellular polysaccharide antigens

Additional Facultative Gram Negative Bacilli from GI Tract:
Enterobacteriaceae:
   - Klebsiella pneumoniae
   - Klebsiella oxytoca
   - Enterobacter cloacae
   - Enterobacter aerogenes
   - Serratia marcescens
   - Proteus mirabilis
   - Citrobacter freundii
   - Citrobacter koseri

Non-fermentors:
   - Acinetobacter
   - Achromobacter
   - Alcaligenes
   - Moraxella
**Pseudomonas aeruginosaa**

1. Virulence factors:

<table>
<thead>
<tr>
<th>VIRULENCE FACTOR</th>
<th>MECHANISM OF ACTION</th>
</tr>
</thead>
<tbody>
<tr>
<td>Exotoxin A</td>
<td>Increases tissue destruction by inhibition of protein synthesis (similar to diphtheria toxin)</td>
</tr>
<tr>
<td>Exotoxin S</td>
<td>Inhibits protein synthesis and phagocytic activity</td>
</tr>
<tr>
<td>Elastase</td>
<td>Disrupts immunoglobulins and complement, enhances tissue invasion; damages blood vessels</td>
</tr>
<tr>
<td>Phospholipase C</td>
<td>Disrupts cell membranes and pulmonary surfactant</td>
</tr>
<tr>
<td>Aliginate</td>
<td>Enhances adherence, protects from phagocytosis, contributes to aminoglycoside resistance</td>
</tr>
<tr>
<td>Leukocidin</td>
<td>Inhibits neutrophils and lymphocytes</td>
</tr>
<tr>
<td>Pyocyanin</td>
<td>Causes oxidative tissue damage, suppresses other bacteria, disrupts cilia activity</td>
</tr>
<tr>
<td>Pili</td>
<td>Enhance adherence to host epithelial cells</td>
</tr>
<tr>
<td>Resistance</td>
<td>Resistant to many antibiotics via change or loss of porins; contain a wide variety of beta-lactamases including carbapenemases; aminoglycoside hydrolyzing enzymes; efflux; etc.</td>
</tr>
<tr>
<td>Endotoxin</td>
<td>LPS; may cause septic shock</td>
</tr>
</tbody>
</table>

2. Etiology/pathogenesis

- a. Opportunistic pathogen – contaminated water
- b. Nosocomial infections
- c. Ubiquitous - particularly water
- d. Ecthyma gangrenosum: begins as local cutaneous edema, and progressing to erythema hemorrhagic bullae, and finally necrosis with blue to black eschars surrounded by erythematous margins

3. Clinical Identification of Organism:

- a. Gram negative bacillus, oxidase positive, growth on MacConkey agar.
- b. Blue green pigment - pyocyanin (blue) and pyoverdin (yellow).

Other Pseudomonadaceae of Clinical Importance:

- a. *Pseudomonas*
- b. *Burkholderia*
- c. *Comamonas*
- d. *Acidovorax*
- e. *Brevundimonas*
- f. *Stenotrophomonas*
Many species but *Candida albicans* the most frequent

1. **Virulence factors:**
   a. Adherence by interaction of glycoproteins on the yeast’s surface and patient’s epithelial cells.
   b. Germ tube or pseudohyphae believed to play a role in penetration of epithelial cells.
   c. Proteinases, phosphatases, and phospholipases aid penetration.
   d. Inflammatory reaction results with predominance of neutrophils.
   e. Organisms of relatively low virulence but if patient is compromised, yeast infections occur.
   f. Predisposing conditions include the following:
      (1) skin barrier damaged
      (2) mucosal barriers damaged
      (3) hormonal or nutritional imbalance
      (4) decreased numbers of phagocytic cells
      (5) intrinsic defects in function of phagocytic cells
      (6) cell mediated immunity problems
      (7) CAPD (chronic or continuous ambulatory peritoneal dialysis)

2. **Etiology/Pathogenesis:**
   a. Normal flora of intestinal tract
   b. Esophagitis, enteric, peritonitis and perianal
   c. *Candida* peritonitis due to peritoneal dialysis
   d. *Candida* ulceration - gastric, small or large bowel.
   e. Bowel involvement may be characterized by ulceration, superficial erosions, pseudomembrane formation, penetrating ulcers and perforation.

3. **Clinical Identification of Organism:**
   a. Unicellular, eukaryotic, budding cells - stain Gram positive
   b. Multiple by production of blastoconidia (buds).
   c. *C. albicans* is germ tube positive.

**Other yeasts of clinical importance:**

<table>
<thead>
<tr>
<th>Yeast Name</th>
<th>Germ Tube</th>
<th>Pseudohyphae</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blastoschizomyces</td>
<td><img src="image1.png" alt="Germ tube" /></td>
<td><img src="image2.png" alt="Pseudohyphae" /></td>
</tr>
<tr>
<td><em>Candida tropicalis</em></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Candida parapsilosis</em></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Candida glabrata</em></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Geotrichum</em></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Bacteroides fragilis

1. Virulence
   a. Capsule - polysaccharide
      Induces abscess formation
   b. Collagenase
   c. Hyaluronidase
   d. Fibrinolysin
   e. IgA, IgM, and IgG proteases

2. Etiology/pathogenesis
   a. Obligate anaerobic
   b. Normal flora in bowel (10^{11} CFU per gram of stool).
   c. Survives and grows in areas of low oxidation-reduction potential (E_h)
   d. Resistant to all aminoglycosides - does not have oxygen dependent transport mechanism across bacterial cell membrane
   e. Escaped from intestines. Usually several organisms which function in synergistic pathogenicity. Facultative organisms cause the initial infection and anaerobes, especially B. fragilis, cause intra-abdominal abscesses.
   f. Facultative organisms consume oxygen ---> reduced E_h and Bacteroides and/or other anaerobes grow.

3. Clinical Identification of Organism:
   a. Gram negative bacillus
   b. No growth in air (grows without oxygen)
   c. Bile salt resistant - B. fragilis group.

Other Anaerobic Gram Negative Bacilli:

   B. fragilis group - B. fragilis, B. ovatus, B. distasonis, B. vulgatus, B. thetaiotaomicron
   Prevotella melaninogenica
   Fusobacterium nucleatum
   Fusobacterium necrophorum

Anaerobic Gram Positive Bacilli:

   Clostridium
   Actinomyces

Anaerobic Gram Positive Cocci

   Peptostreptococcus
A. Pancreatic abscess

1. Virulence
   a. Reflux of contaminated bile
   b. Occasionally hematogenous – usually monomicrobial infection
   c. Occurs in 1 to 9% of patients after acute pancreatitis
   d. Can occur occasionally by penetration from a peptic ulcer

2. Etiology/Pathogenesis
   a. Polymicrobial (30-50%)
   b. *Escherichia coli*
   c. Other Enterobacteriaceae
   d. *Enterococcus*
   e. *Staphylococcus aureus*
   f. *Bacteroides*
   g. *Prevotella*
   h. *Fusobacterium*
   j. *Peptostreptococcus*

B. Hepatic abscess

1. Virulence:
   a. Usually associated with:
      cholecystitis     liver transplantation
      appendicitis     chronic granulomatous disease
      diverticulitis   inflammatory bowel disease
      peritonitis

2. Etiology:
   a. *Entamoeba histolytica* (3 to 9% of those with amebic dysentery)
   b. Most polymicrobial
   c. *E. coli*, *Klebsiella*, and other Enterobacteriaceae
   d. *Bacteroides*
   e. *Prevotella*
   f. *Fusobacterium*
   g. *Actinomyces*
   h. *Candida*
   i. *Staphylococcus aureus* (usually hematogenous)
   j. *Streptococcus pyogenes*    **“”**
   k. *Streptococcus* - viridans group
The specific types of microorganisms that cause hepatic abscess vary with the underlying disease. *S. aureus* and *S. pyogenes* abscesses usually result from bacteremia; *Candida* abscesses are usually found in neutropenic patients.

3. Clinical Identification:
   a. Blood cultures (50% positive)
   b. Ultrasonography guided diagnostic fine needle aspirate: aerobic and anaerobic cultures

4. Pathogenesis:
   The source of infection in the liver may be:
   a. Biliary
   b. Portal, in which a pathologic process such as appendicitis or inflammatory bowel disease is in the bed of the portal venous circulation and associated with pyelophlebitis (acute suppurative thrombophlebitis in the portal venous system)
   b. Infection in a contiguous structure (ex. gallbladder)
   c. Infection anywhere in the body via the hepatic artery
   d. Infection secondary to a penetrating wound or trauma to liver

C. Splenic abscess:
   1. Virulence
      a. Uncommon
      b. Multiple small abscesses - complication of hematogenous dissemination.
      c. Infection maybe from contiguous organs
   2. Etiology/Pathogenesis
      a. *Staphylococcus aureus* (sometimes secondary to endocarditis)
      b. *Streptococcus*
      c. Enterobacteriaceae (especially *Salmonella*)
      d. *Bacteroides*
      e. *Prevotella*
      f. *Candida* (increasing in incidence)
   3. Clinical Diagnosis
      Blood cultures positive in 70% if multiple abscesses

D. Appendicitis:
   a. Manifests as right lower quadrant abdominal pain
   b. Inflamed appendix – location variation results in variations in location of pain
   c. Persistent obstruction of the appendiceal lumen leads to gangrene and rupturing the pus filled appendix.
   d. If appendix ruptures and not walled off, peritonitis occurs
e. Colonic microflora are involved: anaerobes and Enterobacteriaceae
f. Mesenteric lymphadenitis is often confused with appendicitis (Yersinia)

E. Diverticulitis
Herniation of mucosa and submucosa can rupture and lead to peritonitis