Treatment of Surgical Infections

We Need to Know:
  • how infections arise
  • what normally prevents infection from occurring
  • treatment priorities
Life-Threatening Surgical Infections

Two mains types:

Intra-abdominal Infections

Skin and Soft Tissue Infections
Balance of Factors Normally Prevents Infection

- Bacterial Factors
- Environmental Factors
- Host Defenses
Some Virulence Factors of *Staphylococcus aureus*

**Cell Surface Components**
- opsonization
- phagocytosis (slime)
- peptidoglycan + teichoic acid (endotoxin-like properties)
- protein A
- capsule

**Extracellular Products**
- coagulase
- catalase
- TSST-1
- a toxin
- β toxin
- bacteriocins

**Phenotypic and Genetic Variability**
- changing antigenic structure
- antibiotic resistance (e.g. MRSA, VRSA)
Virulence Factors: Polysaccharide Capsule of *B. fragilis*

Encapsulated = Abscess

Unencapsulated
Microbial Synergy

1 + 1 =
Microbial Synergy

1 + 1 = 3
Microbial Synergy in “Mixed Infections”

- E. coli $2 \times 10^8$
- E. coli $2 \times 10^8$ plus B. fragilis $2 \times 10^9$
Host Defenses
Phagocytosis of Invading Microbes

From: James A Sullivan, http://comet.net/quill
Neutrophils
Oxidative Killing Mechanisms for Destruction of Microbes

Note: System REQUIRES molecular Oxygen

\[ \text{O}_2 + \text{Cl}^- \rightarrow \text{HOCl} \]

\[ \text{H}_2\text{O}_2 \rightarrow \cdot\text{OH} \]

\[ \text{O}_2^- \rightarrow \cdot\text{OH} \]

\[ \text{H}_2\text{O}_2 \rightarrow \cdot\text{OH} \]

\[ \text{H}_2\text{O} \rightarrow \cdot\text{OH} \]

\[ \text{H}_2\text{O}_2 + \text{H}_2\text{O} \rightarrow \cdot\text{OH} \]

\[ \text{O}_2^- + \text{H}_2\text{O}_2 \rightarrow \cdot\text{OH} \]

\[ \text{O}_2^- + \text{Fe}^{2+} \rightarrow \cdot\text{OH} \]

\[ \text{O}_2^- + \text{Fe}^{3+} \rightarrow \cdot\text{OH} \]

\[ \text{H}^+ + \text{H}_2\text{O}_2 \rightarrow \cdot\text{OH} \]

\[ \text{H}_2\text{O} + \text{H}_2\text{O} \rightarrow \cdot\text{OH} \]

\[ \text{H}_2\text{O} + \text{H}_2\text{O} \rightarrow \cdot\text{OH} \]
Simplified External View of Human
Cross Sectional View of Human

- Conjunctiva
- Mouth
- Urinogenital Tract
- Respiratory Tract
- Vagina
- Alimentary Tract
- Skin
- Anus
Formation of an Abscess
“Removes” Bacteria from Host
Intra-abdominal Surgical Infections
Defect in GI Tract

Serosa
Muscularis
Mucosa

Serosa
Muscularis
Mucosa
Pathogenesis of Intraabdominal Sepsis

- Bacterial Inoculation → Localized Inflammation ("Decisive Period")
- Localized Inflammation → Diffuse Inflammation (Peritonitis)
- Diffuse Inflammation (Peritonitis) → Death
- Localized Inflammation (Abscess) → Time
Bacteriology of Intra-abdominal Infection

- Vast majority of bacteria in colon are anaerobic species
  - Contribute little to clinical intraabdominal infection
- *Bacteria isolated in clinical infections make up* < 0.1 % of *normal colonic flora*
- Most common anaerobic pathogen, *Bacteroides fragilis*, accounts for only 1% of colonic flora
Causative Bacteria in Peritonitis or Intraabdominal Abscess

**Aerobic Species**
- *Escherichia coli*
- *Enterococcus faecalis*
- *Proteus* species
- *Klebsiella* species
- Other streptococci
- Other aerobes
- *Enterobacter* species
- *Pseudomonas aeruginosa*
- *Staphylococcus aureus*

**Anaerobic Species**
- *Bacteroides fragilis*
- Other *Bacteroides* species
- Clostridia
- Other anaerobes
- Peptostreptococci
- Fusobacteria
- Peptococci
- Propionibacteria
- Veillonella
Virulence Factors: Polysaccharide Capsule of *B. fragilis*

Encapsulated = Abscess

Unencapsulated
Role of Aerobes and Anaerobes in a Rat Model of Intra-abdominal Sepsis

Inoculum into Peritoneal Cavity

% of subjects

% Abscess % Mortality

E. coli
B. fragilis
E. coli + B. fragilis
Adjuvant Substances

• Inoculum of bacteria for infection *much less* if adjuvant substances
  – increase bacterial virulence
  – interfere with host defenses

• Most important adjuvant is **blood**

• Others include **bile salts**, **gastric mucin**, **pancreatic secretions**, **urine** and **chyle**
Classification of Peritonitis

**Primary**
- Types of Patients: Immuno-compromised, Pts with Cirrhosis, Children
- Source of Bacteria: Exogenous
- # Bacterial Species: Single
- "Surgery" Required: Seldom

**Secondary**
- Types of Patients: Relatively "Normal" Patients
- Source of Bacteria: Endogenous
- # Bacterial Species: Multiple
- "Surgery" Required: Usually

**“Tertiary”**
- Types of Patients: Compromised ICU Patients, MODS / MSOF
- Source of Bacteria: Endogenous
- # Bacterial Species: Multiple
- "Surgery" Required: Varies
<table>
<thead>
<tr>
<th>Mode</th>
<th>Early</th>
<th>Late</th>
</tr>
</thead>
<tbody>
<tr>
<td>Physical Exam</td>
<td>++++</td>
<td>++</td>
</tr>
<tr>
<td>CT scan</td>
<td>++</td>
<td>+++++</td>
</tr>
<tr>
<td>Laparoscopy</td>
<td>++</td>
<td>+ / ++</td>
</tr>
<tr>
<td>DPL</td>
<td>+ / -</td>
<td>++</td>
</tr>
<tr>
<td>Ultrasound</td>
<td>+</td>
<td>++</td>
</tr>
<tr>
<td>Celiotomy</td>
<td>++++</td>
<td>+</td>
</tr>
</tbody>
</table>
Timing of Surgical Intervention

Dramatically influences result/ outcome.

Example:
- little morbidity if gastric/ proximal small bowel perforations is closed ≤ 24°.
- colon perforation closed ≤ 12°.

? Development of sepsis/ organ failure.
Timing may determine what is being treated!!
- e.g. peritonitis vs. abscess
**Principles of Abdominal Source Control**

**Prompt** control ongoing contamination.
Remove (all) necrotic tissue.
- complete debridement before decision about reconstruction

**Insure excellent blood supply.**
- debridement of non-viable tissue
- knowledge of blood supply

**No tension** on repair.
Lembert ("sero-muscular") Suture to Close Defect
Source Control in the Proximal GI Tract: Influence of Anatomic Location
Variables in Operative Source Control: Acute Colonic Diverticulitis without Abscess

• Operative Treatment
  – 1. Segmental Resection with Colostomy / Hartman Pouch
  – 2. Segmental Resection, 1° Anastamosis
  – 3. Segmental Resection, 1° Anastamosis, Loop Ileostomy
  – 4. Segmental Resection, 1° Anastamosis with Exteriorization
  – 5. Subtotal Colectomy, Ileostomy
  – 6. Other

• Preoperative Bowel Preparation
  – 1. None
  – 2. Antibiotic

• Peritoneal Lavage
  – 1. None
  – 2. Antibiotic
  – 3. Other

• Wound Management
  – 1. Close
  – 2. Open
Goals of Antimicrobial Therapy for Intraabdominal Infections

- Hasten elimination of infecting microorganisms
- Shorten clinical manifestations of infection
- Minimize risk of recurrent infection
- Begun when diagnosis is suspected
- Anticipate pathogens most likely to be encountered at site of infection
**Recommended Antimicrobial Regimens for IAIs—The SIS**

**Single-Agent Regimens**
- Ampicillin/sulbactam
- Cefotetan
- Ertapenem
- Imipenem/cilastatin*
- Meropenem*
- Piperacillin/tazobactam*
- Ticarcillin/clavulanic acid
- Cefoxitin

**Combination Regimens**
- Aminoglycoside + antianaerobe (clindamycin or metronidazole)*
- Aztreonam + clindamycin*
- Cefuroxime + metronidazole
- Ciprofloxacin + metronidazole*
- 3\(^{rd}/4^{th}\) generation cephalosporin + antianaerobe*

*Also recommended for higher-risk patients with IAI.

### Recommended Antimicrobial Regimens for cIAIs—IDSA

<table>
<thead>
<tr>
<th>Type of Therapy</th>
<th>Agent(s) Recommended for Mild to Moderate Infections</th>
<th>Agents Recommended for High-Severity Infections</th>
</tr>
</thead>
<tbody>
<tr>
<td>Single-agent regimens</td>
<td></td>
<td></td>
</tr>
<tr>
<td>β-lactam/β-lactamase inhibitor combinations</td>
<td>Ampicillin/sulbactam*, ticarcillin/clavulanic acid</td>
<td>Piperacillin/tazobactam</td>
</tr>
<tr>
<td>Carbapenems</td>
<td>Ertapenem</td>
<td>Imipenem/cilastatin, meropenem</td>
</tr>
<tr>
<td>Combination regimens</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cephalosporin-based</td>
<td>Cefazolin or cefuroxime plus metronidazole</td>
<td>Third/fourth-generation cephalosporin (cefotaxime, ceftriaxone, ceftizoxime, ceftazidime, cefepime) plus metronidazole</td>
</tr>
<tr>
<td>Fluoroquinolone-based</td>
<td>Ciprofloxacin, levofloxacin, moxifloxacin, or gatifloxacin each in combination with metronidazole†</td>
<td>Ciprofloxacin in combination with metronidazole</td>
</tr>
<tr>
<td>Monobactam-based</td>
<td></td>
<td>Aztreonam plus metronidazole</td>
</tr>
</tbody>
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Intra-abdominal Sepsis: Treatment

Correct the primary pathology!!!
  ? exploratory surgery
  resect, patch, repair, debride, or drain underlying cause

Aggressive resuscitation and monitoring
  enormous "third space" fluid losses

Important role for appropriate antibiotics
  knowledge of pathogens based on origin of bacterial inoculation
Surgical Skin and Soft Tissue Infections
Classification of SSTIs

- **Uncomplicated**
  - Cellulitis
  - Impetiginous lesions
  - Furuncles
  - Simple abscesses
  - Can be treated by surgical incision and drainage alone

- **Complicated**
  - Deep soft tissue infections
  - May require surgical intervention
    - Infected ulcers
    - Infected burns
    - Major abscesses
  - Significant underlying disease state, which complicates response to treatment

Necrotizing Soft Tissue Infections

- Extensive tissue destruction
- High mortality rate
- Mixed aerobic and anaerobic
  - gram-negative and gram-positive bacteria
- Recognize early and treat promptly
  - Surgical Rx: debride all necrotic tissue
  - May require amputation
  - Worry about reconstruction later
- Hyperbaric O₂ controversial
External Appearances Can Be Deceiving!
Extensive Necrotizing Soft Tissue Infection
Microbiology of Necrotizing Soft Tissue Infections

Polymicrobial  most common
- *Staphylococcus aureus*
- Anaerobic streptococci
- Aerobic gram-negative bacilli
- *Bacteroides fragilis* -- unusual

Monomicrobial
- *Streptococcus pyogenes*
- *Streptococcus viridans*
- *Clostridium* sp.

Rare monomicrobial
- *Aspergillus*
- *Vibrio vulnificans*
Necrotizing Soft Tissue Infections: Streptococcal

Streptococcal soft-tissue infection
-20% of all infections
-Epidemic in southwest U.S.
-Toxic shock-like syndrome
Streptococcal Fasciitis with Myonecrosis
Necrotizing Soft Tissue Infections: Clostridial

Clostridial infections
- “Anaerobic cellulitis”
- Myonecrosis
Occult malignant disease
Relatively rare!
Clostridial Myonecrosis
36 Hours After Untreated Stab Wound
Necrotizing Soft Tissue Infection: Mixed

- Mixed infections ~ 80%
  - Perineum (Fournier-type)
  - Extremity
  - Abdominal wall
  - Any surgical wound
  - Head/neck---Unusual!
  - Chest wall---RARE!
Necrotizing Fasciitis of Perineum (Fournier-type)

- Massive scrotal edema
  - Patchy skin necrosis
- Extension into buttocks
Soft Tissue Gas on Radiographs: “Fournier” Gangrene
Necrotizing Infection of Extremity
Treatment of Severe Soft Tissue Infections

- Aggressive resuscitation
- Debridement in the operating room
- Broad-spectrum antibiotics
- Pack wound open (saline gauze)
- Return to OR *daily* until wound is under control
- Early (enteral) nutrition

*Treatment is the same for all severe soft tissue infections!*
## COMMON ANTIBIOTIC CHOICES FOR NECROTIZING SOFT TISSUE INFECTION

<table>
<thead>
<tr>
<th>Mixed infection (80%)</th>
<th>Monomicrobial (20%)</th>
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<tr>
<td>Carbapenem or Piperacillin/tazobactam + Aminoglycoside</td>
<td>High dose clindamycin or High-dose penicillin</td>
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</table>

There are several other alternatives

Review antibiotics after 48 hours
Summary: Skin and Soft Tissue Infection

- Soft tissue infections are commonly seen by general surgeons.
- Wide spectrum of disease.
- Severe soft tissue infections seldom respond to antibiotics alone.
- Aggressive debridement and a “high index of suspicion” are critical for resolution.
- Usually polymicrobial.
- Initial antibiotic choice based on anticipated pathogens.
Summary: Intra-abdominal Infection (IAI)

• IAI seldom respond to antibiotics alone.
• Source control is critical for resolution.
• Usually polymicrobial.
• Antibiotic choice based on anticipated pathogens.
• Antibiotics can have deleterious effects!
The End