



الكورس الاول --- اسس العناية المركزة

PREVENTIVE PRACTICES IN ICU

Sawa University

College of health and medical techniques

Department of Anesthesia

..... Stage

جامعة ساوة الاهلية

كلية التقنيات الصحية والطبية

قسم تقنيات التخدير

.....المرحلة 3....

....محاضرة رقم 5...

Lecture No. 5

الجانب العملي

استاذ المادة : د. نور عماد موسى
اختصاص تخدير وعناية مركزة



1. ALIMENTARY PROPHYLAXIS IN INTENSIVE CARE

Oral cavity & bowel as sources of infection in critically ill patients

- Microbial invasion of body: skin v/s GIT
- Skin: multi layered keratinised surface
- GIT: single cell layer mucosa □ 0.1 mm thick.
- Microflora outnumbers that in skin
- No of bacteria in 1 gram of stool (10 to 100 billion) > the no of people on Earth (6.5 billion)
- Real threat of microbial infection comes from GIT

Microbial density in the Alimentary tract

Segment	Population Density*
Oral cavity	10^5 – 10^6
Stomach	$<10^3$
Distal small bowel	10^7 – 10^9
Rectum	10^{10} – 10^{12}

*Colony-forming units (CFUs) per gram or mL of luminal contents.

From Simon GL, Gorbach SL. Intestinal microflora. Med Clin North Am 1982;66:557.

Microbes in GIT

- Moist environment in mouth & GIT - ideal for microbial proliferation.
- 400 to 500 different species of bacteria and fungi in the adult GIT.
- Levels of Protective Mechanisms from microbes in the GIT
 - 1.) Lumen – Acid - anti septic
 - 2.) Bowel wall – Physical barrier of Mucosa
 - 3.) Extraluminal - Reticuloendothelial system (2/3rd of it is located intra abdominally □ result of frequent microbial invasion across bowel wall.)

Predisposing Conditions for sepsis from GIT

1.) Translocation: Movement of organisms across the bowel wall into the systemic circulation.

3 conditions that promote it:

- a.) microbial overgrowth in the bowel lumen
- b.) disruption of the mucosal barrier
- c.) defective clearance by the lymphatic system.

2.) Reduced gastric acidity:

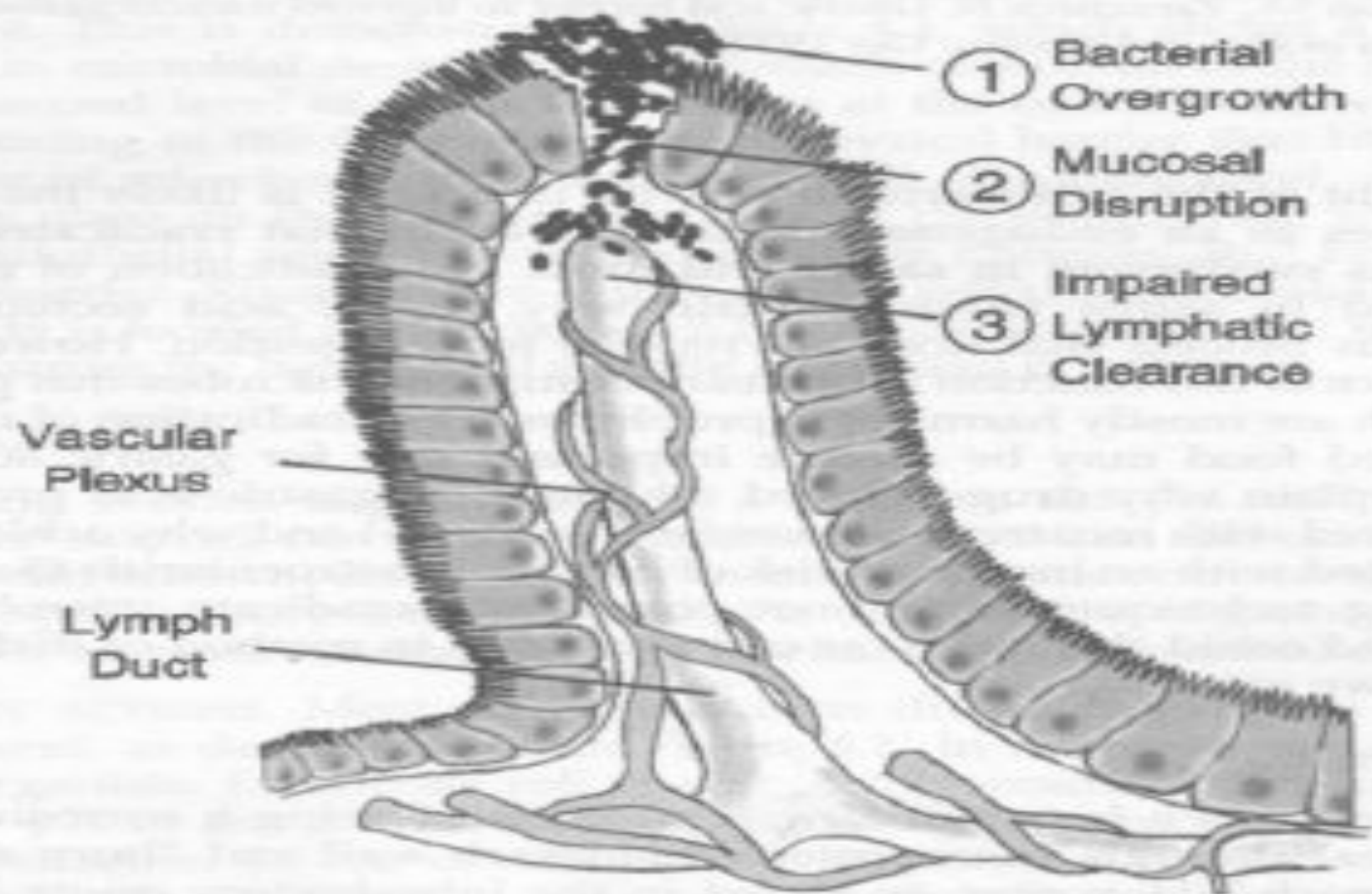


FIGURE 4.2 The triple threat of translocation. This diagram of an intestinal microvillus shows three conditions that predispose to bloodstream invasion by enteric microorganisms.

Pathogenesis:

Reduced gastric acidity: □ loss of antiseptic action □ bacterial overgrowth in stomach □ infections :

- Gastroenteritis
 - Pneumonia from aspiration of infectious gastric contents.
 - Septicemia from bacterial translocation across the bowel wall.
- Reason to avoid the use of drugs that inhibit gastric acid secretion.

Pathogenesis.

- **Mucosal lining**: normally shed and replaced every 2 - 3 days.
- When nutrient blood flow is inadequate to support the replacement process, the surface of the bowel becomes denuded, □ superficial erosions.
- Actions of gastric acid may aggravate this , but the **principal cause of stress-related mucosal injury is impaired blood flow**, not gastric acidity.

Clinical Consequences

- ▣ Erosions can be demonstrated in 75% to 100% of patients within 24 hours of admission to the ICU.

Effect:

- 1.) **Clinically silent** : majority
- 2.) **Microbial translocation**
- 3.) **Clinically apparent** GI bleeding in as many as 25% (*Chest 2001*)
- 4.) **Clinically significant bleeding** (i.e., significant drop in blood pressure or requires transfusion) occurs in only 1% - 5% .

High Risk Conditions for clinically significant bleeding

- 1. Mechanical ventilation for longer than 48 hours
- 2. Coagulopathy (i.e., platelets <50,000, INR >1.5, or PTT >2 X control)
- 3. Hypotension
- 4. Severe sepsis
- 5. Multisystem trauma
- 6. Severe head injury
- 7. Burns involving >30% of body surface area
- 8. Renal failure or hepatic failure

PREVENTIVE MANAGEMENT :

1. Enteral Nutrition:

- It exerts a trophic effect on the bowel mucosa that helps to maintain the structural and functional integrity of the bowel mucosa
- Can be considered adequate prophylaxis for stress-related gastric hemorrhage unless there is some other condition that raises special concern for GI bleeding, such as a coagulopathy, a prior history of bleeding from gastritis or PUD, or active PUD.

PREVENTIVE MANAGEMENT

2 .Pharmacologic Treatment :

1. Cytoprotection : uses an agent that provides local protection to the gastric mucosa.
 - ▣ **Sucralfate** : Aluminum salt of sucrose sulfate forms a protective covering on the gastric mucosa.

Alimentary prophylaxis

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2. Gastric acid reduction:

- **Histamine type-2 Receptor Antagonists** : Famotidine and ranitidine
- **Proton Pump Inhibitors**
 - Omeprazole
 - Lansoprazole
 - Rabeprazole
 - Pantoprazole
 - Esomeprazole

3.DECONTAMINATION OF THE ALIMENTARY TRACT

1.)Oral Decontamination

The aspiration of mouth secretions into the upper airways □ inciting event in most cases of hospital-acquired pneumonia.

- Aspiration of one micro liter of saliva will introduce about one million microbes into the airways. Successful regime in ICU

Non-absorbable antibiotics applied locally in the mouth.

- *Preparation:* mixture of 2% gentamicin, 2% colistin, and 2% vancomycin as a paste.
- *Regimen:* Apply paste to the buccal mucosa with a gloved finger q6h until the patient is extubated.
- Eradicates most aerobic bacteria and *Candida* species from the mouth in about one week.

3.DECONTAMINATION OF THE ALIMENTARY TRACT

- ❑ 2.) Selective digestive decontamination
- ❑ More extensive version of oral decontamination covering entire alimentary tract.
- ❑ Is selective as it does not eliminate normal flora inhabitants of the bowel which are important in preventing colonization with opportunistic pathogens.
- ❑ A successful SDD regimen
 - Oral cavity: A paste of 2% polymyxin, 2% tobramycin, and 2% amphotericin is applied to the inside of the mouth with a gloved finger q6H.
 - GI tract: A 10 mL solution containing 100 mg polymyxin E, 80 mg tobramycin, and 500 mg amphotericin is given via a NGT q6H.
 - Systemic: *i.v* cefuroxime, 1.5 grams q8H for the first 4 days of therapy

THANK YOU