Surgical infections and choice of antibiotics

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Definition

- Infections that occur as a result of surgical procedures.
- These infections may involve the surgical site or extend to other parts of the body.
- They are typically caused by bacteria and less commonly by viruses and fungi.

Pathogenesis

- The magnitude of response and eventual outcome after surgery is generally related to several factor:
 - The initial number of microbes.
 - The rate of microbial proliferation in relation to containment and killing by host defenses.
 - Microbial virulence.
 - The potency of host defenses.

Host defenses

- The mammalian host possesses several layers of endogenous defense mechanisms that serve to prevent microbial invasion, These defenses are integrated and redundant so that the various components function as a complex, highly regulated system that is extremely effective in coping with microbial invaders.
 - Entry of microbes into the mammalian host is precluded by a number of barriers that possess either an epithelial (integument) or mucosal (respiratory, gut, and urogenital) surface.
 - the skin harbors its own resident microflora that may block the attachment and invasion of noncommensal microbes (colonization resistance).
 - Host barrier cells may secrete substances that limit microbial proliferation or prevent invasion.
- Diseases of the skin (e.g., eczema and dermatitis) are associated with overgrowth of skin commensal organisms, and barrier breaches invariably lead to the introduction of these microbes.

Surgical site infections (SSIs)

- Surgical site infections: are infections of the tissues, organs, or spaces exposed by surgeons during performance of an invasive procedure.
- SSI occurs at the site of surgery within 30 days following surgery or up to 90 days following surgery where an implant is involved (e.g. an artificial heart valve or joint).

- The development of SSIs is related to three factors:
- (a) the degree of microbial contamination of the wound during surgery
- (b) the duration of the procedure
- (c) host factors such as diabetes, malnutrition, obesity, immune suppression; and a number of other underlying disease states.

Risk factors for development of SSIs

Risk factors for development of surgical site infections

Patient factors Older age Immunosuppression Obesity Diabetes mellitus

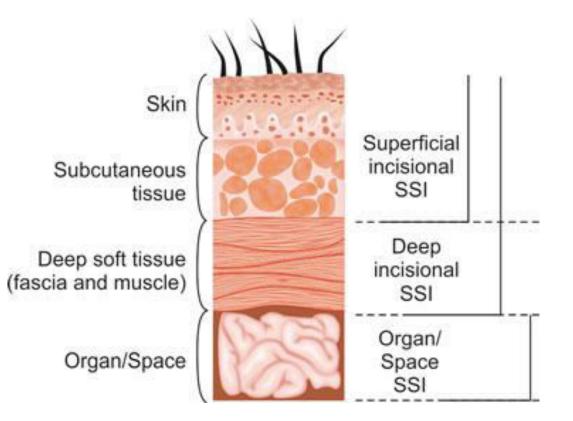
Chronic inflammatory process Malnutrition Smoking Renal failure Peripheral vascular disease Anemia Radiation Chronic skin disease Carrier state (e.g., chronic *Staphylococcus* carriage) Recent operation

Local factors

Open compared to laparoscopic surgery Poor skin preparation Contamination of instruments Inadequate antibiotic prophylaxis Prolonged procedure Local tissue necrosis Blood transfusion Hypoxia, hypothermia

Microbial factors

Prolonged hospitalization (leading to nosocomial organisms) Toxin secretion Resistance to clearance (e.g., capsule formation) CLASSIFICATION OF SURGICAL SITE INFECTIONS (SSIs) SSIs are divided into incisional SSI which includes those involving only skin and subcutaneous tissues (superficial incisional SSI) and those involving deeper softer tissues of the incision (deep incisional SSI), and organ/space SSI.



Superficial incisional SSI	Deep incisional SSI		
Occurs within 30 d	Occurs within 30 d OR within 90 days if an implant is present		
Only skin and subcutaneous tissue	Deep soft tissues (fascial and muscle layers)		
Patient has at least 1 of the following: a. Purulent drainage from incision b. Organisms identified from wound c. Superficial incision that is deliberately opened by surgeon d. Diagnosis of SSI by surgeon	Patient has at least 1 of the following: a. Purulent drainage from deep incision b. An incision that spontaneously dehisces or is deliberately opened or aspirated by surgeon, with or without culture c. Abscess or other evidence of infection that is detected on gross anatomic or histopathologic examination, or imaging		
And patient has at least 1 of the following: pain or tenderness, localized swelling, erythema or heat	And patient has at least 1 of the following: fever (temperature > 38°C), localized pain or tenderness		

SSI = surgical site infection.

- Organ/space SSI:
- Infection occurs within 30 days after operation or within 90 days if an implant is present.
- Infection involves anatomic structures which was opened or manipulated during an operation.
- Patient at least has one of the following:
 - Purulent discharge from a drain placed through a stab wound into the organ/space
 - Organisms isolated from the organ/space by wound culture
 - Abscess or other evidence of infection involving the organ/space is identified by direct examination, reoperation, histopathology or radiological examination.
 - Diagnosed by SSI by surgeon.

Classification of surgical wounds

Wound class	Definition	Examples of typical procedures	Wound infection rate (%)	Usual organisms
Clean	Nontraumatic, elective	Wide local excision of	2	Staphylococcus aureus
	surgery; no entry of GI, biliary, tracheo bronchial, respiratory, or GU tracts	breast mass		
Clean- contaminated	Respiratory, genitourinary, GI tract entered but minimal contamination	Gastrectomy, hysterectomy	<10	Related to the viscus entered

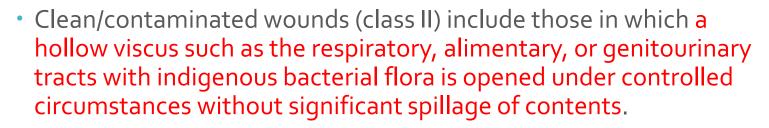
Contaminated	Open, fresh, traumatic wounds; uncontrolled spillage from an unprepared hollow viscus; minor break in sterile technique	Ruptured appendix; resection of unprepared bowel	20	Depends on underlying disease
Dirty	Open, traumatic, dirty wounds; traumatic perforated viscus; pus in the operative field	Intestinal fistula resection	28-70	Depends on underlying disease

Classification of surgical wounds

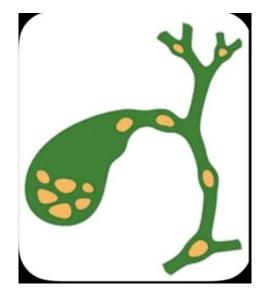
- Clean wounds (class I) include those in which no infection is present; only skin microflora potentially contaminate the wound, and no hollow viscus that contains microbes is entered.
- Examples: excision of ganglion cyst, laparoscopic hernial repair, mastectomy, thyroidectomy, splenectomy, ovarian resection, skin melanoma.







- While elective colorectal cases have classically been included as class II cases, a number of studies in the last decade have documented higher SSI rates (9–25%).
- Examples: cholecystectomy, colorectal surgery, hysterectomy, bowel resection, Gastric surgery.



Contaminated wounds (class III) include open accidental wounds encountered early after injury, those with extensive introduction of bacteria into a normally sterile area of the body due to major breaks in sterile technique (e.g., open cardiac massage), gross spillage of viscus contents such as from the intestine, or incision through inflamed, albeit nonpurulent tissue.

• Examples: appendicitis

- Dirty wounds (class IV) include traumatic wounds in which a significant delay in treatment has occurred and in which necrotic tissue is present, those created in the presence of overt infection as evidenced by the presence of purulent material, and those created to access a perforated viscus accompanied by a high degree of contamination.
- Examples:

perforated appendicitis, Perforated diverticulitis, Necrotizing soft tissue Infections.



Case 1

• 13y with acute abdominal pain and a diagnosis of appendicitis. You complete a laparoscopic appendectomy and it is inflamed but no perforation.

What is the wound classification?

Case 2

• 40 yrs old female with a follicular adenocarcinoma and you performed a total thyroidectomy.

What is the wound classification?

Common bacteria involved in surgical infections

Streptococci

- The most important is the group A, β-haemolytic Streptococcus, also called Streptococcus pyogenes.
 - It has the ability to spread, causing cellulitis, and to cause tissue destruction through the release of enzymes such as streptolysin, streptokinase and streptodornase.
- Both Streptococcus pyogenes and Streptococcus faecalis may be involved in wound infection after bowel surgery.
- All the streptococci are sensitive to penicillin and erythromycin.
- The cephalosporins are a suitable alternative in patients who are allergic to penicillin

Staphylococci

- Staphylococcus aureus is the most important pathogen in this group
 - It can cause suppuration in wounds and around implanted prostheses.
 - Some strains are resistant to many common antibiotics (especially MRSA) and so are difficult to treat
- Staphylococcus epidermidis, also known as coagulase-negative Staphylococcus, is now recognised as a major threat in vascular and orthopaedic prosthetic surgery and in indwelling vascular cannulae/catheters.
 - The bacteria form bioflms that adhere to prosthetic surfaces and limit the efectiveness of antibiotics.

Clostridia

- Clostridial organisms are Gram-positive, obligate anaerobes that produce resistant spores .
- Clostridium perfringens is the cause of gas gangrene
- Clostridium tetani causes tetanus.
- Clostridium difcile (C. dif.) is the cause of pseudomembranous colitis, in which destruction of the normal colonic bacterial fora by antibiotic therapy allows an overgrowth of the normal gut commensal C. dif. to pathological levels.
 - the colitis may lead to perforation and the need for emergency colectomy with an associated high mortality.
 - Treatment involves resuscitation and antibiotic therapy.
 - The fbrinous exudate is typical and diferentiates the colitis from other infammatory diseases

Aerobic Gramnegative bacilli

- These bacilli are normal inhabitants of the large bowel.
- Escherichia coli and Klebsiella spp. are lactose fermenting; Proteus is non-lactose fermenting
- Most organisms in this group act in synergy with Bacteroides to cause SSIs after bowel operations
- Most of them can be involved in UTI, particularly in relation to urinary catheterisation.
- Pseudomonas spp. tend to colonise burns and tracheostomy wounds, as well as the urinary tract
- There is increasing concern about the development of ESBLs in many of this group of bacteria, which confer resistance to many antibiotics, particularly cephalosporins

Bacteroides

- Bacteroides are non-spore-bearing, strict anaerobes that colonise the large bowel, vagina and oropharynx.
- Bacteroides fragilis is the principal organism that acts in synergy with aerobic Gramnegative bacilli to cause SSIs, including intraabdominal abscesses after colorectal or gynaecological surgery.
- They are sensitive to the imidazoles (e.g. metronidazole) and some cephalosporins (e.g. cefotaxime).

Localised infection (abscess)

- Most abscesses relating to surgical wounds take 7–10 days to form after surgery
- If it is not drained or resorbed completely, a chronic abscess may result.
- If it is partly sterilised with empirical antibiotics, an antibioma may form.
- Persistent chronic abscesses may lead to sinus or fistula formation.
- In a chronic abscess,
 - lymphocytes and plasma cells are seen.
 - There is tissue sequestration and later calcifcation may occur.
 - Certain organisms are associated with chronicity (Mycobacterium and Actinomyces).
- USS, CT and MRI can be used for diagnosis and may allow image-guided aspiration and drainage
- Management includes Incision and drainage.
 - Antibiotics used if the abscess is not localized (eg: evidence ofcellulitis) or if the abscess is closed after drainage

Localised infection (cellulitis)

- Cellulitis is a non-suppurative, invasive infection of tissues
- typically caused by organisms such as β -haemolytic strepto cocci , staphylococci and Clostridium perfringens
- There is poor localisation
- Systemic signs are common, with chills, fever and rigors.
- Tissue destruction, gangrene and ulceration may follow, which are caused by release of proteases.
- Treatment :Analgesics, antipyretic, antibioticAntibiotic of choice is penicillin erthromycin is used as alternative in pt allergic topenicillin



Figure 5.9 Streptococcal cellulitis of the leg following a minor puncture wound.



Figure 5.10 Staphylococcal cellulitis of the face and orbit following severe infection of an epidermoid cyst of the scalp.

Localised infection (lymphangitis)

- The presence of infectious bacteria in the lymphatic vessele of an area of the body
- Most common cause streptococcus pyogenes
- presents as painful red streaks in affected lymphatics draining the source of infection
- often accompanied by painful lymph node groups in the related drainage area
- Treatment: antibiotics



Systemic inflammatory response syndrome

- Systemic infammatory response syndrome (SIRS) is a systemic manifestation of sepsis (Table 5.1), although the syndrome may also be caused by multiple trauma, burns or pancreatitis without infection.
- SIRS should not be confused with bacteraemia, although the two may coexist
- Septic manifestations and multiple organ dysfunction syn- drome (MODS) in SIRS are mediated by the release of pro- infammatory cytokines such as interleukin-1 (IL-1) and tumour necrosis factor alpha (TNFα)
- In its most severe form, MODS may progress into multiple system organ failure (MSOF)

Sepsis

- Sepsis is a clinical syndrome that has physiologic, biologic, and biochemicalabnormalities caused by a dysregulated host response to infection. Sepsis and the inflammatory response that ensues can lead to multiple organ dysfunction syndrome and death.
- Bacteremia is unusual following superficial SSIs but common after anastomotic breakdown (deep space SSI). It is usually transient and can follow procedures undertaken through infected tissues (particularly instrumentation in infected bile or urine). It may also occur through bacterial colonization of indwelling intravenous cannulae.
- Bacteremia is important when a prosthesis has been implanted, as infection of the prosthesis can occur.
- Sepsis accompanied by MODS may follow anastomotic breakdown.
- Aerobic Gram-negative bacilli are mainly responsible, but S. aureus and fungi may be involved, particularly after the use of broad-spectrum antibiotics

TABLE 5.1 Definitions of systemic inflammatory response syndrome (SIRS) and sepsis

SIRS is

Presence of two out of three of the following:

- Hyperthermia (>38°C) or hypothermia (<36°C)
- Tachycardia (>90/min, no β -blockers) or tachypnoea (>20/min)
- White cell count >12 \times 10⁹/litre or <4 \times 10⁹/litre

Sepsis is SIRS with a documented source of infection

Severe sepsis or sepsis syndrome is sepsis with evidence of failure of one or more organs: respiratory (acute respiratory distress syndrome), cardiovascular (septic shock follows compromise of cardiac function and fall in peripheral vascular resistance), renal (usually acute tubular necrosis), hepatic, blood coagulation systems or central nervous system

Table 6-10

Summary of Surviving Sepsis Campaign guidelines

Initial Evaluation and Infection Issues

- *Initial resuscitation:* Begin resuscitation immediately in patients with hypotension or elevated serum lactate with resuscitation goal of at least 30 mL/kg IV crystalloid given in the first 3 hours.
- Ongoing fluid administration should be guided by physiologic response as measured by clinical variables (e.g., heart rate, blood pressure, urine output) and/or other invasive or noninvasive monitoring.
- Resuscitation goals include mean arterial pressure >65 mmHg, urine output >0.5 mL/kg per h, and mixed venous oxygen saturation >65%.
- Target resuscitation to normalize lactate in patients with elevated lactate levels.
- *Diagnosis:* Obtain appropriate cultures prior to antibiotics, but do not delay antibiotic therapy. Imaging studies should be performed promptly to confirm a source of infection.
- Antibiotic therapy: Begin IV antibiotic therapy as early as possible and within the first hour after recognition of severe sepsis/ septic shock. Use broad spectrum antibiotic regimen with penetration into presumed source, reassess regimen daily with de-escalation as appropriate, discontinue antibiotics in 7 to 10 days for most infections, stop antibiotics for noninfectious issues. Consider the use of serial procalcitonin levels, which may allow earlier cessation of antibiotic therapy.
- *Source control:* Establish anatomic site of infection as rapidly as possible; implement source control measures as soon as possible after initial resuscitation. Remove intravascular access devices if potentially infected.

Hemodynamic Support and Adjunctive Therapy

- *Fluid therapy:* Fluid resuscitate using crystalloid, with continued fluid challenges so long as hemodynamic parameters continue to improve (i.e., for so long as the patient remains fluid-responsive). Albumin may be used as an adjunct if large volumes of crystalloid are required, but hydroxyethyl starch and gelatin-based fluids should not be used.
- *Vasopressors/Inotropic Therapy:* Maintain MAP of >65 mmHg. Centrally-administered norepinephrine is the first-line choice. Add vasopressin if needed to raise MAP or to reduce norepinephrine requirement. Epinephrine is an alternative to vasopressin but has greater risk of reduced splanchnic blood flow. Dopamine is an appropriate alternative only in select patients (bradycardia, low risk of arrhythmia), and there is no role for low-dose "renal protection" dopamine. Phenylephrine is not recommended. Insert arterial catheters for patients requiring vasopressors. Consider dobutamine infusion for persistent hypotension after appropriate resuscitation and use of vasopressor agents.
- Steroids: Consider intravenous hydrocortisone (dose <300 mg/day) for adult septic shock when hypotension responds poorly to fluids and vasopressors.

Other Supportive Therapy

- *Blood product administration:* Transfuse red blood cells when hemoglobin decreases to <7.0 g/dL in the absence of extenuating circumstances (e.g., myocardial ischemia, hemorrhage). It is not necessary to use fresh frozen plasma to correct INR abnormalities in the absence of bleeding. Consider prophylactic transfusion of platelets when counts are less than 10,000/mL in the absence of bleeding, <20,000/mL if there is a risk of bleeding, and <50,000 in the setting of active bleeding or need for procedure.
- *Mechanical ventilation:* Target an initial tidal volume of 6 mL/kg body weight and plateau pressure of <30 cm H₂O in patients with acute lung injury. Use PEEP to avoid lung collapse. Adopt a conservative fluid strategy. In the setting of sepsis-induced ARDS with PaO₂/FiO₂ ratio <150, use prone ventilation over continued supine position or high-frequency oscillatory ventilation. Use a weaning protocol to evaluate the potential for discontinuing mechanical ventilation. Pulmonary artery catheter placement is not indicated for routine monitoring.
- Sedation: Minimize sedation using specific titration endpoints.
- *Glucose control:* Use protocolized approach to blood glucose management targeting upper blood glucose target of 180 mg/dL. *Prophylaxis:* Use stress ulcer (proton pump inhibitor or H_2 blocker) and deep venous thrombosis (low-dose unfractionated or fractionated heparin) prophylaxis.
- Limitation of support: Discuss advance care planning with patients and families and set realistic expectations.

Data from Rhodes A, Evans LE, Alhazzani W, et al: Surviving Sepsis Campaign: International Guidelines for Management of Sepsis and Septic Shock: 2016, *Intensive Care Med.* 2017 Mar;43(3):304-377.

Specific wound infections

Gas gangrene (clostridial myonecrosis)



- Microorganism/ Clostridium perfringens.
- Widely found in nature, particularly in soil and feces.
- Military persons, traumatic surgery and colorectal operations.
- Severe local wound pain and crepitus. The wound produces thin, brown, sweet smelling exudate.
- Edema and spreading gangrene (after release of proteases and alpha toxin).
- If not treated > systemic complications with circulatory collapse.

Summary box 5.12

Gas gangrene

- Caused by C. perfringens
- Gas and smell are characteristic
- Immunocompromised patients are most at risk
- Antibiotic prophylaxis is essential when performing amputations to remove dead tissue

Large doses of IV penicillin

Management

Aggressive debridement of affected tissue

Hyperbaric oxygen could be helpful

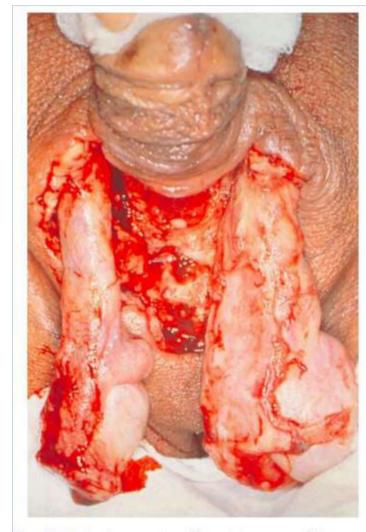
Necrotizing Fasciitis

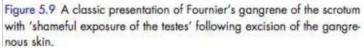
Also known as Synergistic spreading gangrene This condition is not caused by clostridia coliforms, staphylococci, Bacteroides spp., anaerobic streptococci have all been implicated, acting in synergy. Meleney's synergistic hospital gangrene (abdominal wall) Fournier's gangrene (scrotal infection)



FIGURE 6-4. Necrotizing soft tissue infection. (A) This patient presented with hypotension due to severe late necrotizing fasciitis and myositis due to β -hemolytic streptococcal infection. The patient succumbed to his disease after 16 hours despite aggressive debridement.

Necrotizing Fasciitis







Meleney's synergistic hospital gangrene

Necrotizing Fasciitis

- Patients are almost always immunocompromised.
- Severe wound pain, signs of spreading inflammation with crepitus and smell are all signs of the infection spreading.
- Untreated, it will lead to widespread gangrene and MSOF.
- Broad-spectrum antibiotic therapy must be combined with aggressive circulatory support. Locally, there should be wide excision of necrotic tissue and laying open of affected areas. The debridement may need to be extensive, and patients who survive may need large areas of skin grafting.

Pre-operative phase

- Shower
- Shaving
- patient dress
- Theater staff dress
- Hand washing
- Antibiotic prophylaxis
- >One hour before incision
- Before induction
- Before tourniquet application
- ▶1 dose vs 3 doses
- Additional dose:
 - -Prolonged operation
- -Excess blood loss

Intra-operative phase

- Patient skin preparation
 O lodine / chlorhexidine
 O Allowed to dry and avoid spillage
- Incision drapes
- Sterile gown and gloves
- Patient homeostasis
- Theater discipline
- Wound dressing

Treatment of SSI

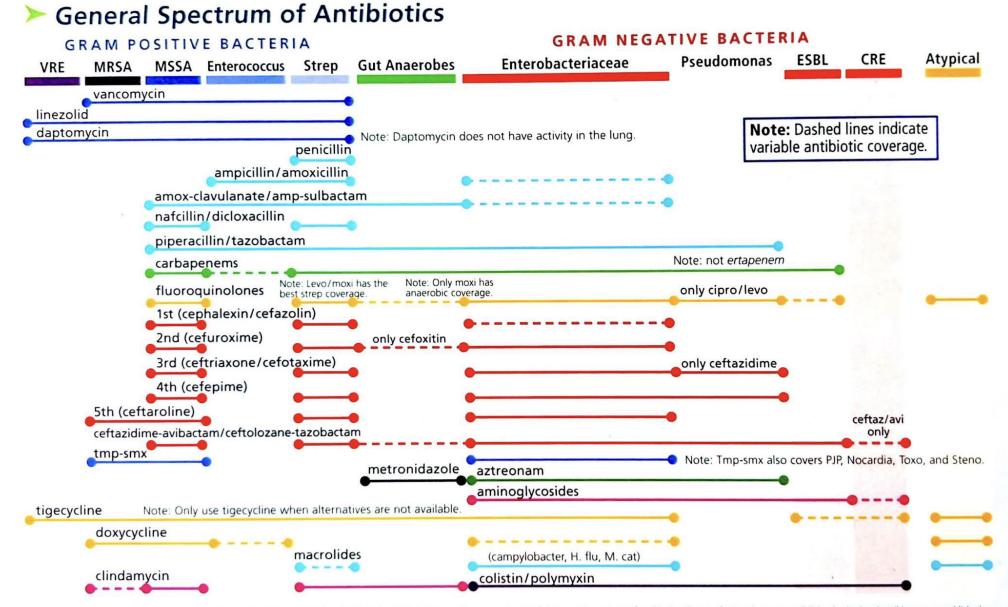
- Surveillance
- Drainage of pus
- Culture and sensitivity
- MRSA
- VRE
- ESBL strains
- ➢ Debridement
- Antibiotics
- ➢Removal of implant

Treatment of established surgical infection

- It is unusual to have to treat SSIs with antibiotics, unless there is evidence of spreading infection, bacteraemia or systemic complications (SIRS and MODS).
- The appropriate treatment of localized SSIs is interventional radiological drainage of pus or open drainage and debridement.

Antibiotic	МОА	Active against
Penicillin	Inhibit cell wall synthesis	Most gram +ve
Cephalosporin	Inhibit cell wall synthesis	Gram +ve & -ve
Erythromycin	Inhibit protein synthesis 50s	Mycoplasma & actinomycosis
Tetracycline	Inhibit protein synthesis 30s	Gram +ve & -ve bacteria that not sensitive to penicillin
Chloramphenicol	Inhibit protein synthesis 50s	Broad spectrum
Aminoglycoside	Inhibit protein synthesis 30s	Gram +ve, -ve and mycobacteria
Sulfonamide/Trimethoprime	Inhibit folate synthesis	Community acquired gram -ve
4-Fluoroquinolones	Inhibit DNA replication	Nosocomial infections and all gram -ve

Common antibiotic agents



This document is for educational purposes only. It is not intended as a guideline for clinical practice. It is a graphic representation of the general spectrum of antibiotics. Please refer to culture susceptibilities, institutional antibiogram, or published antibiotic guides for additional guidance. Also consider Infectious Disease consultation for further assistance.

References

- Schwartz's principles of surgery 11th edition
- Washington Manual of Surgery 8th edition
- Bailey & love's short practice of surgery 28th edition
- UpToDate

Thank you