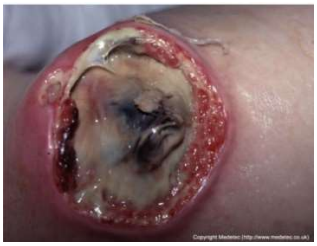
Infectious complications of pressure-induced skin and soft tissue injury

Pressure ulcers are localized areas of tissue necrosis that tend to develop when soft tissue is compressed between a bony prominence and an external surface for a prolonged period of time.



Factors that contribute to the infection of pressure ulcers:

1️⃣ Breaks in the integrity of the skin barrier

2️⃣ Pressure-induced changes

3️⃣ Contamination from contiguous dirty areas, such as from fecal incontinence

- Pressure ulcer colonization by microorganisms precedes development of infection. The ulcer is first colonized with **skin flora**, which is rapidly replaced by bacteria from the **local environment and the urogenital or gastrointestinal tracts**. The pressure ulcers are **polymicrobial.**

***Critical colonization**: Higher concentrations of bacteria in pressure ulcers may **inhibit normal wound healing** without inducing a host response. (Number of colony forming units that are required to delay the healing, if the count of CFU less than 10^2 the ulcer will progress with almost normally healing).

(Important) Predominant organisms in Infected pressure ulcers are:

✳️ Enterobacter ✳️ Enterococcus faecalis ✳️ Staphylococci

(Important) The major organisms that cause pressure ulcer-related bacteremia are:

✳️ Staphylococci (including methicillin-resistant Staphylococcus aureus, methicillin-susceptible S. aureus, and coagulase-negative staphylococci).

✳️ Streptococci.

✳️ Proteus mirabilis, and anaerobes.

👉 Bloodstream infection is common in patients with sepsis.

A poorly healing **superficial ulcer** with increased drainage but **no surrounding erythema** is suggestive of **a limited superficial infection** of the ulcer base; **spreading erythema** around an ulcer is suggestive of **cellulitis**, and a deep ulcer with **necrotic muscle, undermined tissue, or sinus tracts** is suggestive of a **deeper infection** of the soft tissue or bone.

Diagnosis:

▪ Diagnosis of soft tissue infection of a pressure ulcer is based on clinical features and is straightforward in patients with obvious changes (erythema, warmth, induration, fluctuance, tenderness, drainage, or tissue necrosis) in surrounding skin and underlying soft tissue.

▪ Exudate alone without additional signs or symptoms of soft tissue or systemic involvement should prompt consideration of a limited, superficial infection that is amenable to **local debridement and wound care**.

▪ Identification of causal pathogen(s) with in vitro antimicrobial susceptibility testing are essential to define targeted **antibiotic therapy**.

▪ The most useful specimen for culture is a biopsy of the deepest tissue involved, which is usually obtained during debridement of an ulcer. Aspiration of material below the ulcer margin is an alternative specimen source.

▪ Imaging is not necessary for evaluation of all patients specially if its superficial infection.

▪ magnetic resonance imaging (MRI) (or computed tomography, if MRI is not feasible) may be useful in identifying underlying necrotic tissue or abscess requiring debridement when there are patients with systemic manifestations of infection, positive blood cultures, or indwelling medical devices

▪ When **osteomyelitis** is suspected in patients with pressure ulcers, every effort should be made to obtain a **bone biopsy**, with **histopathologic and microbial analysis** to confirm the diagnosis of osteomyelitis and guide the selection of antibiotic therapy.

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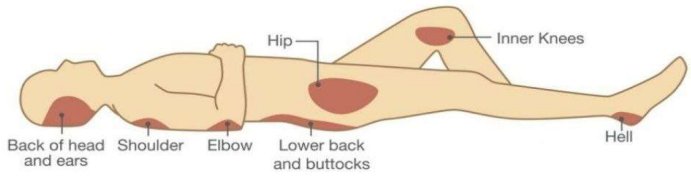
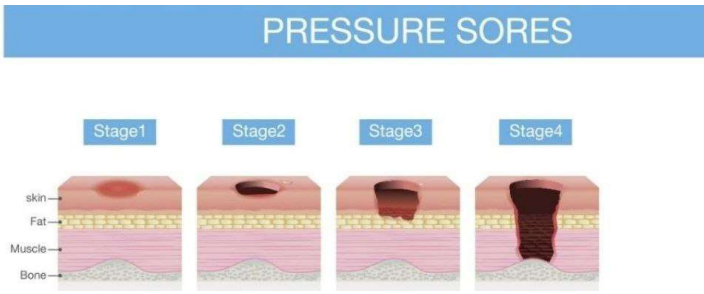




Burn wound infection and sepsis

The most common cause of morbidity and mortality in burn patients.

A variety of factors increase the risk of developing invasive burn wound infection (burn wound sepsis):

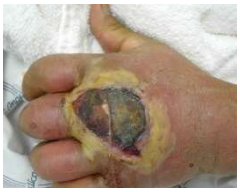
* Individuals who sustain a TBSA burn >20 percent are at particularly high risk.

TBSA → Total Body Surface Area

* Delays in burn wound excision, extremes in age (very old, very young), and impaired immunity.

* Microbial factors, such as type and number of organisms, enzyme and toxin production and motility.

Why may the burn wound get infected?



* Profound alterations in metabolic and host defense mechanisms and immune function, which predisposes burn patients to local and systemic invasion by microbial pathogens.

👉 The burn wound is also susceptible to opportunistic colonization by endogenous and exogenous organisms (Colonization occurs as biofilms)

- The spectrum of microorganisms causing infections in burn patients varies with time and location

Memorize the first table in last page :)

Diagnosis:

Early diagnosis and treatment of burn wound infection relies on ⏭ recognition of an infected burn wound site.

Clinical features of burn wound infection:

✳ Rapid change in the appearance of the wound ❓ conversion of a partial-thickness injury to full-thickness injury.

✳️ Loss of previously viable tissue or skin graft and erythema, wound drainage.

Acute bacterial infection manifests with the development of:

👉 discoloration, pain, purulent exudate, edema, tenderness, swelling, drainage, or malodor from a burn.

Local signs and characteristics of burn wound infections caused by fungi include:

- Unexpectedly rapid separation of the eschar; due to fat liquefaction.
- Rapid spread of subcutaneous edema with central ischemic necrosis

**After the observation of clinical features, cultures should be done such as: qualitative and quantitative wound cultures, Surface wound cultures, Swab cultures and tissue histopathology is obtained by biopsy.

Noninvasive infection	invasive infection
* Typical clinical features of infection without systemic signs, and the bacterial count is $>10^5$ bacteria per gram of tissue obtained from a burn wound or eschar with no invasive component (ie, no microbial invasion into unburned tissue) as identified by tissue histopathology. *Treatment consists primarily of topical antimicrobial therapy and burn wound excision for unexcised wounds.	* Typical clinical features of infection with systemic signs, and bacterial count is $>10^5$ bacteria per gram of tissue obtained from a burn wound or eschar with an invasive component (ie, microbial or fungal invasion into unburned tissue) identified by tissue histopathology. * Treatment: For patients with burn wound sepsis, initial management is aimed at stabilizing the patient and restoring perfusion then, we initiate empiric systemic broad-spectrum antimicrobial therapy and excise all infected tissue to a healthy tissue bed

The most common outcomes of burn wound infections include: ●Graft loss for excised and grafted burn wounds ●Increased number of surgical interventions ●Increased nosocomial infections ●Increased length of stay ●Conversion of donor site.

Surgical site infection (SSI)

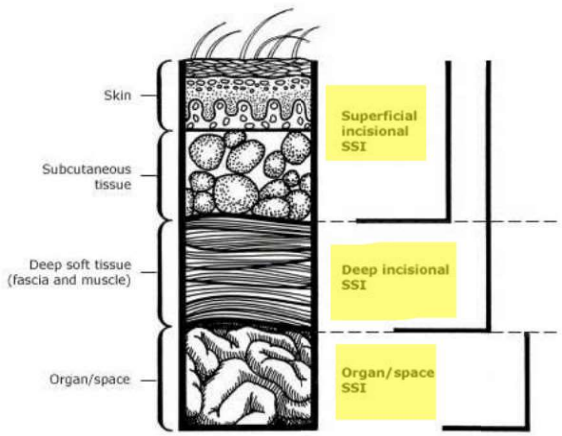
*The most common health care–associated infection following surgery.

An infection related to a surgical procedure that occurs near the surgical site within **30 days** following surgery (or up to **90 days** following surgery where an implant is involved).

Types:

▪ **Superficial incisional SSI**: only skin and subcutaneous tissues, localized swelling, warmth, drainage/ Treatment involves wound exploration and debridement and Antibiotics.

▪ **Deep incisional SSI**: involving deeper softer tissues, patients with clinical signs like those of superficial SSI, imaging (eg, ultrasound, computed tomography) may be helpful to estimate the depth and extent of infection/Treatment involves wound exploration and debridement and Antibiotics.



Surgeons can **reduce rates of SSI** using preventive measures that include **avoiding elective surgery** in patients with active infection, timely administration of **prophylactic antibiotics**, **proper skin preparation**, and maintenance of **sterile conditions**

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Chronologic appearance and characteristics of organisms in burns

Organism, chronology	Characteristics
Commensal skin organisms (gram positive) result in early colonization of burns	▪ *Streptococcus* and *Staphylococcus* species primarily ▪ Other sources are upper respiratory tract and environment ▪ Topical antimicrobials help decrease colonization
Gram-negative species dominant >5 days	▪ 2 to 4 days post-burn, gram-negative bacteria colonize wound ▪ Patient skin, upper respiratory tract, gastrointestinal tract, and hospital environment are typical sources ▪ *Pseudomonas aeruginoasa, Acinetobacter baumannii, Escherichia Coli, Klebsiella pneumoniae, Enterobacter cloacae*
If gram-negative cover is initiated, yeast often appears	▪ Yeast and fungi colonization follows ▪ Majority are *Candida* species, other fungi are increasing in frequency
Finally, more resistant bacteria and fungi invade the wound	▪ MRSA, VRE, multi-drug-resistant *Pseudomonas* and *Acinetobacter* species, and fungi ▪ Usually secondary to broad-spectrum antibiotics, or inadequate host response or therapeutic measures (excision burn, topical and systemic antibiotics) ▪ Transition from colonization to invasion

MRSA: methicillin-resistant Staphylococcus aureus, VRE: vancomycin-resistant Enterococcus

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Surgical site infection definitions

	Time to event*	Extent of tissue involvement	Clinical features	Criteria for diagnosis
Superficial incisional SSI[¶]	Within 30 days of NHSN procedure[Δ]	Skin and subcutaneous tissue	▪ Peri-incisional pain or tenderness ▪ Localized peri-incisional swelling ▪ Peri-incisional erythema or heat	At least one clinical feature **AND** at least one of the following: ▪ Purulent drainage from the superficial incision ▪ Organisms are identified by culture (or non-culture-based microbiologic testing method) performed for clinical diagnosis or treatment (eg, not surveillance) ▪ Incision opened by the surgeon (or other designated clinician) because of concern for superficial SSI[◇]
Deep incisional SSI[¶]	Within 30 or 90 days of NHSN procedure[Δ]	Deep soft tissues of the incision such as the fascia and muscle layers	▪ Fever (>38°C) ▪ Localized pain or tenderness	▪ Purulent drainage from the deep incision ▪ Deep incision that spontaneously dehisces or is opened by the surgeon (or other designated clinician) because of concern for deep SSI **AND** organisms are identified by culture (or non-culture-based microbiologic testing method) performed for clinical diagnosis or treatment (eg, not surveillance). Presence of at least one clinical feature, in absence of microbiologic testing
Organ/space SSI	Within 30 or 90 days of NHSN procedure[Δ]	Any part of the body deeper than the fascia/muscle layers that was opened or manipulated during the procedure	Clinical features for specific organ/space can be found at the CDC website[§] As an example, for intra-abdominal infection, at least two of the following: ▪ Fever (>38°C) ▪ Hypotension ▪ Nausea, vomiting ▪ Abdominal pain or tenderness ▪ Elevated transaminases ▪ Jaundice	Appropriate clinical features specific to the organ/space **AND** at least one of the following: ▪ Purulent drainage from a drain placed into the organ/space[¥] ▪ Organisms identified from culture of fluid or tissue obtained from a superficial incision[‡] ▪ Abscess or other evidence of infection involving the organ/space detected on gross anatomical exam or histopathologic exam ▪ Radiographic imaging findings suggestive of infection

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Surgical wound classification

Class I/Clean

An uninfected operative wound in which no inflammation is encountered and the respiratory, alimentary, genital, or uninfected urinary tract is not entered. In addition, clean wounds are primarily closed and, if necessary, drained with closed drainage. Operative incisional wounds that follow nonpenetrating (blunt) trauma should be included in this category if they meet the criteria.

Class II/Clean-Contaminated

An operative wound in which the respiratory, alimentary, genital, or urinary tracts are entered under controlled conditions and without unusual contamination. Specifically, operations involving the biliary tract, appendix, vagina, and oropharynx are included in this category, provided no evidence of infection or major break in technique is encountered.

Class III/Contaminated

Open, fresh, accidental wounds. In addition, operations with major breaks in sterile technique (eg, open cardiac massage) or gross spillage from the gastrointestinal tract, and incisions in which acute, nonpurulent inflammation is encountered are included in this category.

Class IV/Dirty-Infected

Old traumatic wounds with retained devitalized tissue and those that involve existing clinical infection or perforated viscera. This definition suggests that the organisms causing postoperative infection were present in the operative field before the operation.