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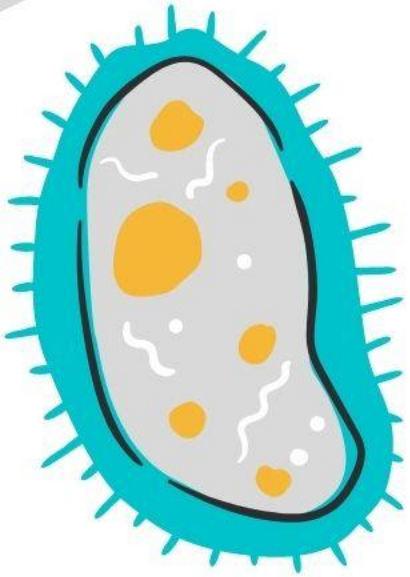
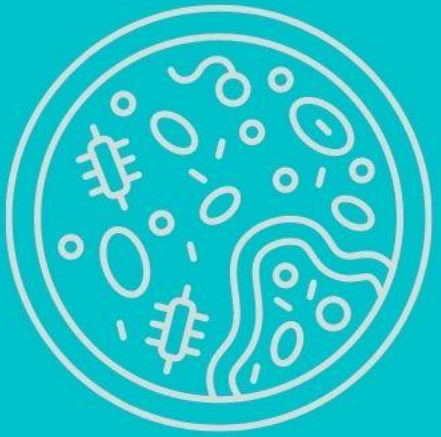


Microbiology

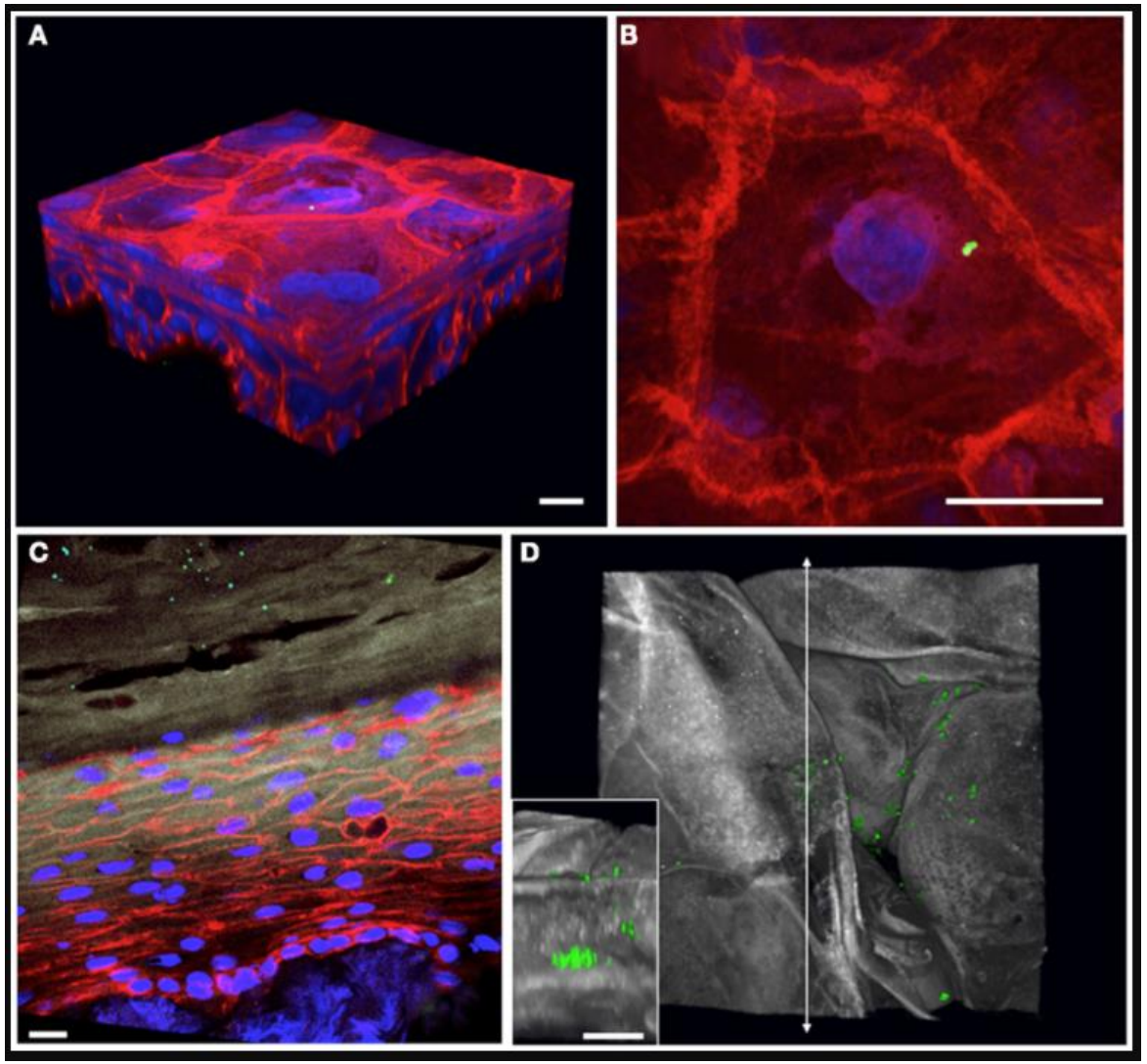
| Modified slides

Written by: Insaf Alammouri
Correction: Saif AL-Tayar
Doctor: Anas Abu-Humaidan



Musculoskeletal System Microbiology

Anas Abu-Humaidan
M.D. Ph.D.



Clinical scenarios associated with SSI

Overview

In this lecture, we will discuss:

- Infectious complications of pressure-induced skin and soft tissue injury
- Burn wound infection and sepsis
- Surgical site infection

Also, we will discuss clinical scenarios of :

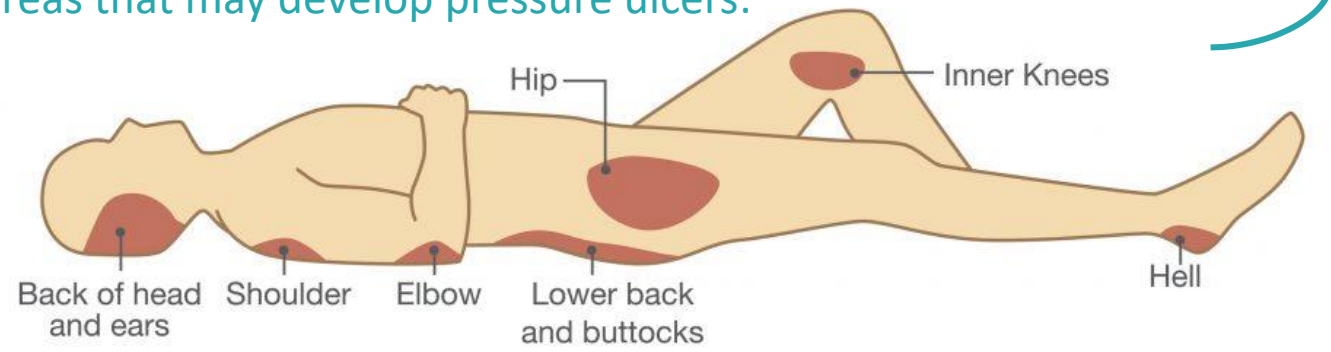
- Necrotizing fasciitis (https://link.springer.com/article/10.1007/s11739-014-1139-7)
- Burn wound infections (https://www.sciencedirect.com/science/article/pii/S246891221830021X#f0015)

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 or skin is compressed between a bony prominence and an external surface for a prolonged period of time (no blood supply → ischemia → ulcer formation). They are a significant problem in critically ill patients, older adults, and in persons with spinal cord injury.
- Pressure ulcers cause pain, decrease quality of life, and lead to significant morbidity and prolonged hospital stays (also they don't heal properly), **in part due to complicating infection**. Infected pressure ulcers are a common problem.
- Local factors that contribute to infection of pressure ulcers include **breaks in the integrity of the skin barrier**, **pressure-induced changes**, and **contamination from contiguous dirty areas**, such as from fecal incontinence (lower back and buttocks can be contaminated by GIT & GUT so we can see skin, GIT, GUT normal flora in these ulcers).
- Pressure ulcer **colonization** by microorganisms precedes development of infection (pressure ulcers are almost always colonized). The ulcer is first colonized with **skin flora**, which is rapidly replaced by **bacteria from the local environment and the urogenital or gastrointestinal tract**s, often from direct fecal contamination

Infectious complications of pressure-induced skin and soft tissue injury

Body areas that may develop pressure ulcers:



a pressure between a bony prominence can cause a pressure ulcer

Diabetic foot ulcer:

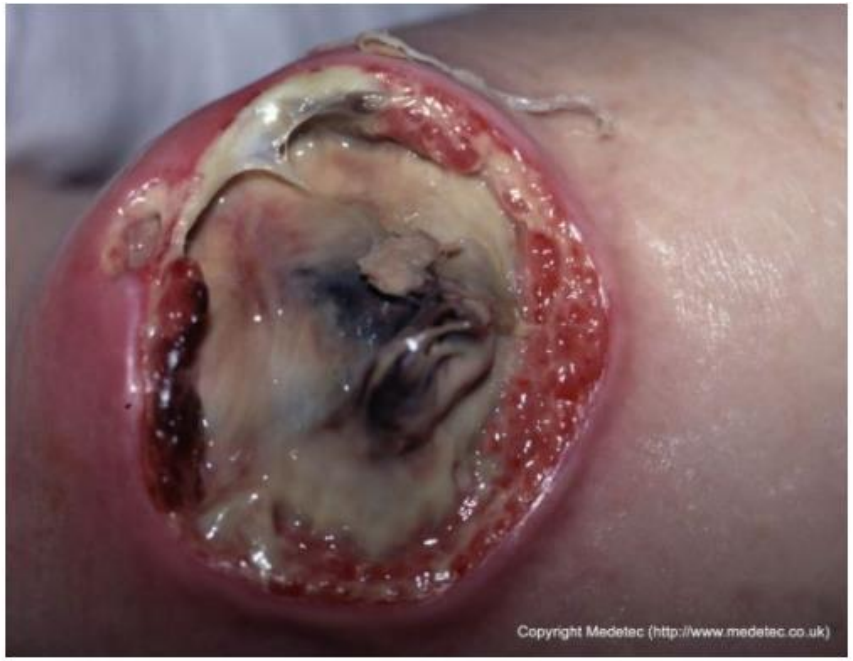
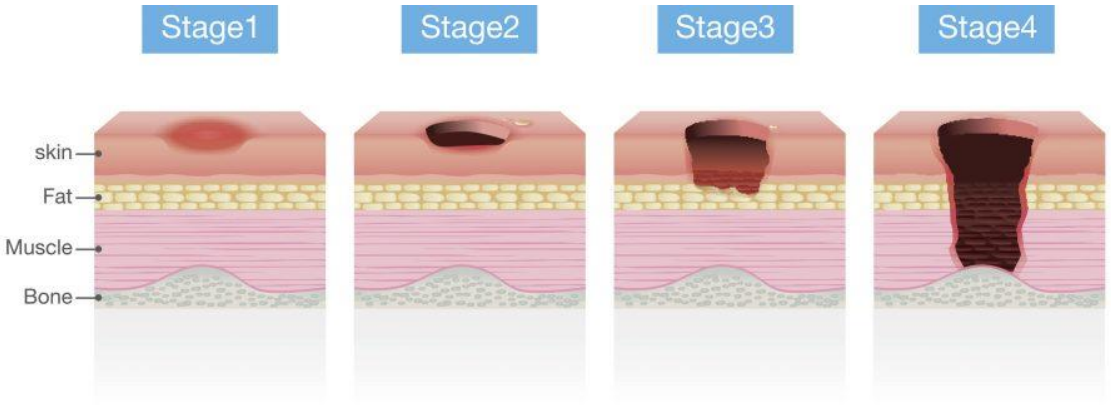


Figure 3: Slough covered pressure ulcer, unstageable

PRESSURE SORES



PRESSURE SORES

Infectious complications of pressure-induced skin and soft tissue injury

- Pressure ulcers are generally associated with a bacteria count of $<10^2$ colony forming units (CFU) per gram of tissue if cared for and kept free of necrotic tissue. Higher concentrations of bacteria in pressure ulcers may inhibit normal wound healing without inducing a host response, a state known as **critical colonization.** (number of colony forming units of bacteria required to delay healing of ulcers (<10^2 CFUs/g of tissue → almost normal healing / >10^2 CFUs/g of tissue → delayed healing))
- The presence of biofilm is also being increasingly recognized as a cause of delayed healing.
- The microbiology of pressure ulcers is polymicrobial and similar in superficial and deep ulcers. Nearly all infected pressure ulcer cultures yield multiple organisms. pressure ulcers are always colonized so if you swap them you find growth of bacteria (skin → staph & strep/ GIT, GUT → enterobacter & enterococcus…)
- A retrospective study of 168 surgical samples evaluated the microbiology of infected pressure ulcers in 101 spinal cord injury (SCI) patients (always in bed, thay have the risk of contamination). The predominant organisms were **Enterobacter** (29 percent), **staphylococci** (28 percent), and **Enterococcus faecalis** (16 percent). In a separate study of pressure ulcer-related bacteremia in SCI patients, the major organisms were **staphylococci** (including methicillin-resistant Staphylococcus aureus, methicillin-susceptible S. aureus, and coagulase-negative staphylococci), **streptococci**, **Proteus mirabilis**, and anaerobes. (Staphylococcus is better than Gi normal flora in overcoming immune defenses in such case, in many deep ulcers bacteremia can occur mostly of s.aureus)

Infectious complications of pressure-induced skin and soft tissue injury

- Infection occurs when microorganisms invade normal tissues, replicating and overwhelming host immune defenses. The extent of local infection associated with a pressure ulcer ranges from an **infection limited to the superficial ulcer base** to one with surrounding **cellulitis** to more extensive involvement of deeper structures (**including fascia, muscle, and bone**).

- 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** (like necrotizing fasciaitis → surgical exploration & debridment with culturing samples of dead tissue).

- Bloodstream infection **is common in patients with sepsis associated with pressure ulcer-related soft tissue infections**; in one study, the bloodstream infection rate **was as high as 79 percent.**

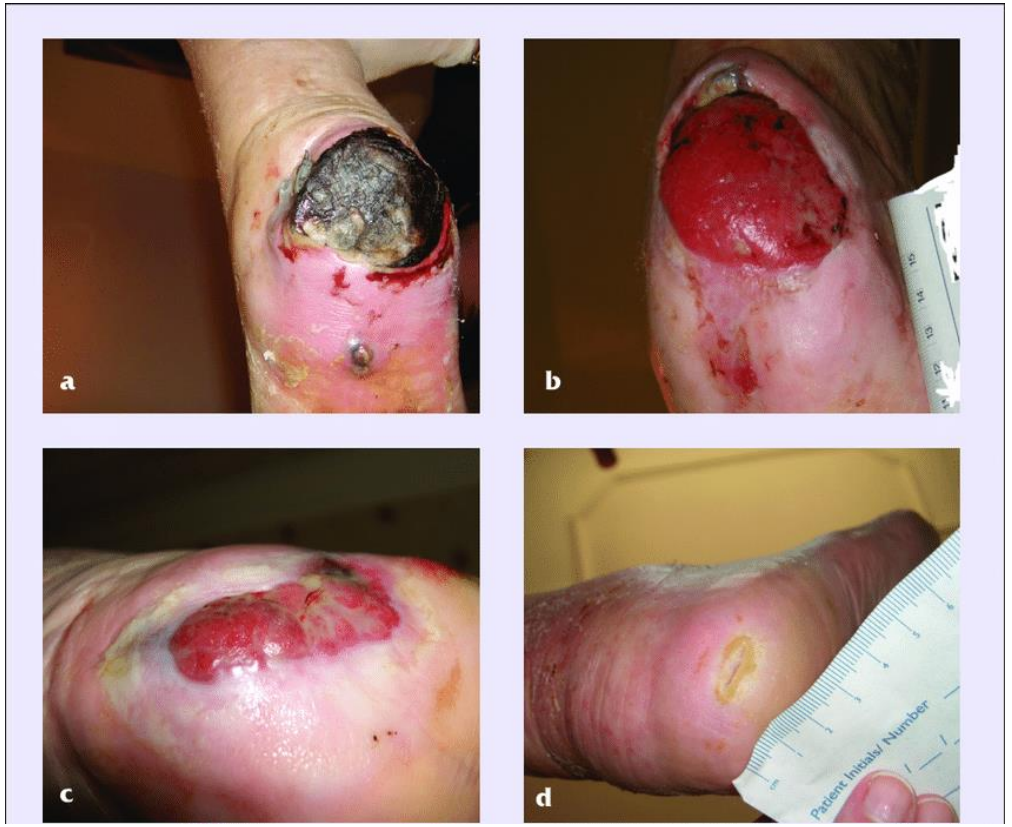
- Diagnosis of soft tissue infection of a pressure ulcer is based on clinical features and is straightforward in patients with obvious changes (erythema, warmth, induration, fluctuance, tenderness, drainage, or tissue necrosis) in surrounding skin and underlying soft tissue. We must determine if its superficial or deep to know if we need empiric antibiotics and culturing.
- Conversely, **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. A limitation of microbiologic evaluation is that it does not distinguish between bacterial invasion and colonization, since **all pressure ulcers are colonized with microorganisms.**
- **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 with pressure-ulcer related soft tissue infections (not needed for superficial infections). However, in patients with **systemic manifestations** of infection, **positive blood cultures**, or **indwelling medical devices** (eg, cardiac valves, cardiac implantable electronic devices, vascular grafts, prosthetic joints) → then we think of sepsis → , magnetic resonance imaging (MRI) (or computed tomography, if MRI is not feasible) may be useful in determining depth of infection & **identifying underlying necrotic tissue** or **abscess** requiring debridement.
- When **osteomyelitis** is suspected in patients with pressure ulcers, every effort should be made to **obtain a bone biopsy** (ulcers can reach bone so a bone biopsy is the best way to identify it before it becomes chronic), with histopathologic and microbial analysis in order to confirm the diagnosis of osteomyelitis and guide the selection of antibiotic therapy (Radiographic imaging can be not specific in Acute phase, but quite clear in chronic phase, we need to catch it in acute phase to start treatment).
- **Full debridement of all necrotic bone and tissue, treatment with antibiotics targeting the pathogen(s) recovered from bone biopsy** (or **empiric antibiotic therapy** based on likely pathogens), and associated flap coverage, when feasible, are essential components of management.

Infectious complications of pressure-induced skin and soft tissue injury

Case study : Pressure ulcer on patient's right heel, with signs of sub-epidermal abscess and infection tracking down the foot (a); heel at first assessment (with a lot of necrotic tissue & spreading erythema), following surgical debridement as an inpatient, care continuing in nursing home with Octenilin, topical antibiotics, padding and elevation (b); wound 6 months after initial presentation, post surgical debridement, healing with continuing care (c); 9 months after the original presentation, the wound healed (d).

With proper care of wound & patient position ulcer can heal completely, unlike diabetic foot ulcers because hyperglycemia is still there



New way of treating ulcers! Maggots (يرقات) can be sterilized and used to eat necrotic tissue, they also produce anti inflammatory products

Research articles

Bacteriological investigation of infected pressure ulcers in spinal cord-injured patients and impact on antibiotic therapy.

STUDY DESIGN Retrospective.

OBJECTIVES To improve the use of bacteriological results for treating spinal cord-injured patients with infected pressure ulcers.

SETTING Microbiology and Orthopaedics Department, Ambroise ParéUniversity Hospital, Boulogne-Billancourt, France.

METHODS Tissue specimens, sampled at the end of the surgical intervention from unbridled and cleaned ulcers were analysed. Drainage liquids were cultured at day 1 (D1) and day 5 (D5) postsurgery. For part of the patients, a presurgery superficial sample was analysed and compared with the surgical and postsurgical samples.

RESULTS In all, 168 surgical samples from 101 patients, 183 D1 and 104 D5 wound drainage liquids were included in this study. Out of the 168 surgical samples 17 (10%) had a negative culture, whereas 151 (90%) had a positive culture. For drainage liquids, the culture was negative in 48% and 56% of the samples at D1 and D5, respectively. The most frequently isolated species were enterobacteria, followed by staphylococci and streptococci.

CONCLUSION Culturing deep tissue specimens sampled from the surgically cleaned and unbridled ulcers allows for the isolation of the bacterial species that are really involved in the ulcer infection. As the identification of these bacteria and their antibiotic susceptibility are available, when the culture results of the D1 postsurgical drainage liquid is also available, it is easier to choose targeted antibiotic treatment.

Burn wound infection and sepsis

- Burns break epithelial barrier → compromise immunity → infections occur

- Infection remains the most common cause of morbidity and mortality in burn patients. The diagnosis and management of burn wound infection remains challenging due to the many physiologic features unique to burn injury (we are not only dealing with the site of burn but with systemic & pathologic consequences of burns especially large ones).

- A variety of factors increase the risk of developing invasive burn wound infection (burn wound sepsis). Individuals who sustain a **TBSA burn >20 percent** (burn surface area) are at particularly high risk; however, burn wound infection and sepsis can occur in smaller burns. Other risk factors include **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, also contribute

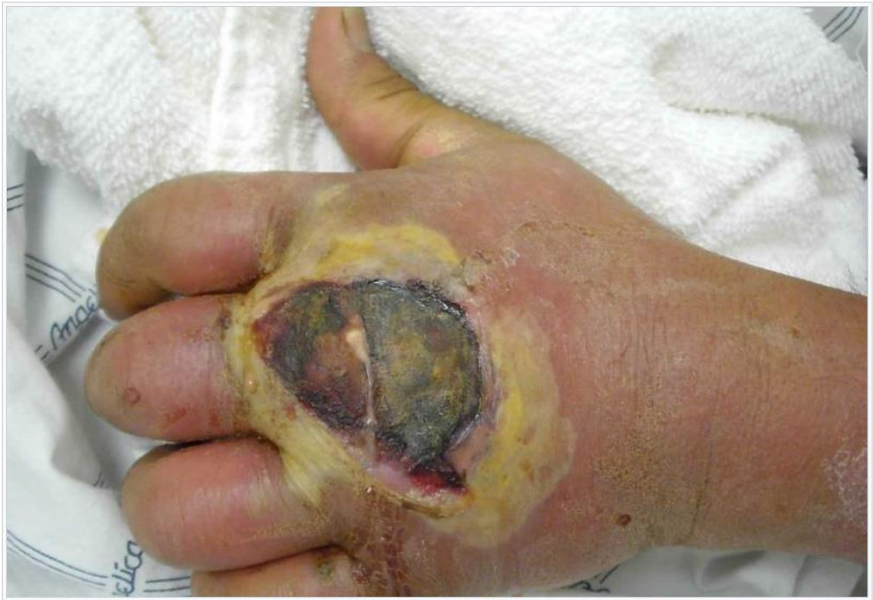
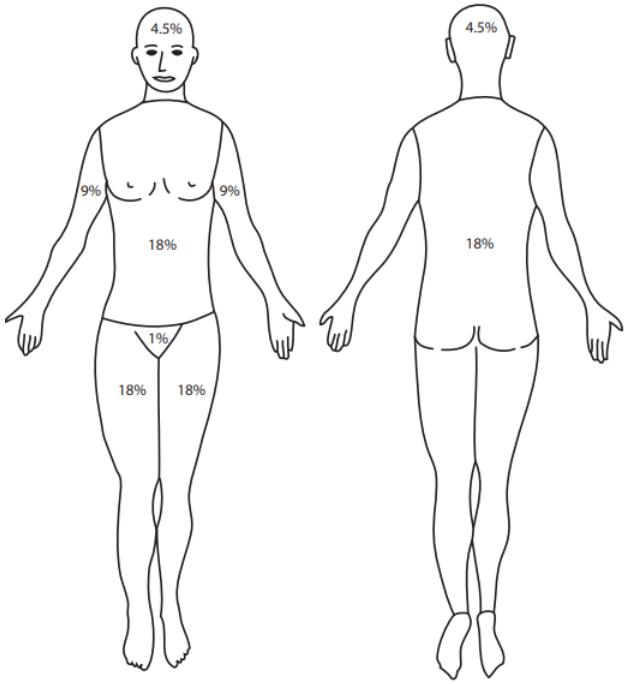


Figure 2. Infection of the hand caused by methicillin-resistant *Staphylococcus aureus*. Photo Credit: Gregory Moran, M.D., from the Center for Disease Control and Prevention.



Burn wound infection and sepsis

- Burn injury is associated with profound alterations in metabolic and host defense mechanisms and immune function, which predisposes burn patients to local and systemic invasion by microbial pathogens (leads to sepsis → high morbidity and mortality). The burn wound is also susceptible to opportunistic colonization by endogenous and exogenous organisms. Colonization of burn wounds by endogenous and/or exogenous organisms often occurs as biofilms

- The spectrum of microorganisms causing infections in burn patients **varies with time and location (table 1).** The organisms causing burn wound **infection typically appear at varying stages post-burn-injury.**

Chronologic appearance and characteristics of organisms in burns

Organism, chronology	Characteristics
Commensal skin organisms (gram positive) result in early colonization of burns Early stages	▪ *Streptococcus* and *Staphylococcus* species primarily ▪ Other sources are upper respiratory tract and environment ▪ Topical antimicrobials help decrease colonization
Gram-negative species dominant >5 days Wound not healing properly	▪ 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

→ give combination of antibiotics or broad spectrum antibiotics

Antibiotics lead to the appearance of fungi →

← Using of antibiotics & antifungals lead to the appearance of multidrug resistant bacteria and fungi

Burn wound infection and sepsis/ diagnosis and management

- Early diagnosis and treatment of burn wound infection relies on **recognition of an infected burn wound site.** The most common clinical feature is a **rapid change in the appearance of the wound**, which may include conversion of a partial-thickness injury to full-thickness injury, or **loss of previously viable tissue or skin graft** (refusal of a burn skin graft may indicate an infection).

- Acute bacterial infection manifests with the development of **discoloration, pain, purulent exudate, edema, tenderness, swelling, drainage, or malodor from a burn** or burn-related wound (previously reepithelialized grafted burn site, skin donor site). The appearance of infection may involve only a portion of the burn wound.

- Local signs and characteristics of burn wound infections **caused by fungi include unexpectedly rapid separation of the eschar**, presumably due to **fat liquefaction**, and rapid spread of subcutaneous edema with central ischemic necrosis

- When burn wound infection is suspected clinically based upon the clinical features discussed above, **qualitative wound cultures** can identify the presence of flora, but **quantitative wound cultures** (number of bacteria per gram of tissue) and histopathology obtained by biopsy of the wound are required to confirm the diagnosis of burn wound infection. The presence of bacteria at concentrations **$>10^5$ bacteria per gram of tissue** in adjacent unburned tissue defines invasive burn wound infection.
- We should also take biopsy from nearby uninfected unburnt wounds because we fear invasive wounds (if bacteria is present → invasive)
- Surface wound cultures are useful for identifying predominant organisms of the burn wound flora. Swab cultures assist in the surveillance of the bacterial flora colonizing burn patients.
- Burn wounds are **swabbed on admission and again if there are any concerning changes in appearance**. In the absence of changes, weekly surveillance swabs are suggested.
- **Tissue histopathology** allows for **quantification and evaluation of infection depth** and extent of involvement

- **Noninvasive infection** — Noninvasive burn wound infection is present when there are typical clinical features of infection **without systemic signs**, and the bacterial count is $>10^5$ bacteria per gram of tissue (or recovery of mold or yeast by culture) obtained from a burn wound or eschar with no invasive component (ie, **no microbial invasion into unburned tissue**) as identified by tissue histopathology.

- For noninvasive burn wound infection, treatment consists primarily of topical antimicrobial therapy and burn wound excision for unexcised wounds, and possibly reexcision for excised wounds.

- **Invasive infection** — Invasive burn wound infection is present when there are typical clinical features consistent with burn wound infection (eg, erythema, wound drainage), associated 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**. Also we can identify invasion by clinical signs like erythema, fever & organ failure.

- For patients with burn wound sepsis, initial management is aimed at stabilizing the patient and restoring perfusion. Concurrently, we initiate **empiric systemic broad-spectrum antimicrobial therapy**, and once the patient is stabilized, we take the patient to the operating room to widely **excise all infected tissue to a healthy tissue bed** as determined by intraoperative biopsy.

Burn wound infection and sepsis/ Prognosis

- It is estimated that 75 percent of the mortality following thermal injuries is related directly to infections.
- Burn intensive care units (ICUs) have the **highest rates of primary bloodstream infection** in patients with central venous catheters among all ICUs
- Emerging multidrug-resistant strains of bacteria and fungi have caused an unanticipated rise in burn wound infections, sepsis, and associated deaths worldwide.

Infection of burn wounds is not without consequence. The most common outcomes of burn wound infections include:

- Graft loss for excised and grafted burn wounds
- Increased number of surgical interventions (no response to antibiotics, more systemic manifestations, invasive infection→ debride more tissue & using more antibiotics)
- Increased nosocomial infections (in immunocompromised patients)
- Increased length of stay
- Conversion of donor site

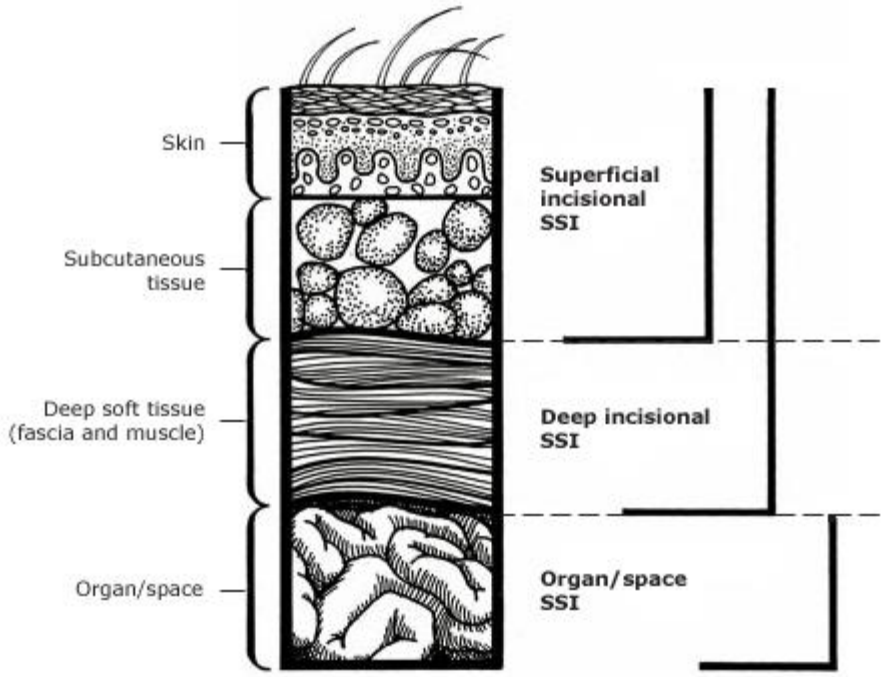
Surgical site infection

- Surgical site infection (SSI) is the most common health care–associated infection following surgery and is associated with significant morbidity and mortality, transfer to an intensive care unit setting, prolonged hospitalizations, and hospital readmission.

- Among those who undergo surgical procedures annually in the United States, 2 to 4 percent will develop an SSI, representing a significant burden on the health care system as a whole.

- The CDC defines an SSI as 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).

- **Incisional SSIs** are further divided into those involving **only skin and subcutaneous tissues** (**superficial** incisional SSI) and those involving **deeper softer tissues of the incision** (**deep** incisional SSI).



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$^{\Delta}$	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$^{\diamond}$
Deep incisional SSI¶	Within 30 or 90 days of NHSN procedure$^{\Delta}$	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$^{\Delta}$	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

SSI: surgical site infection; NHSN: National Health Safety Network; CDC: Centers for Disease Control.

* Day 1 is the procedure date.

¶ SSI can occur in a primary incision or a secondary incision among those undergoing an operation with one or more incisions.

Δ Depends on the surveillance period of the specific procedure.

◇ Typically copious drainage from the incision sufficient to warrant inspection of the fascia to ensure its integrity and to rule out incisional hernia or fasciitis. Fluid and tissue cultures should be obtained. The following do NOT meet the criteria for superficial SSI: cellulitis, stitch abscess, stab (eg, drain not laparoscopic port) or pin site infection, infection in non-NHSN procedures (eg, circumcision), burn wound infection.

§ Additional information can be found at: https://www.cdc.gov/nhsn/PDFs/pscManual/17pscNosInfDef_current.pdf.

¥ Percutaneous or surgical drain.

‡ Based on microbiologic testing performed for purposes of clinical diagnosis or treatment (eg, not surveillance).

Adapted from: Centers for Disease Control and Prevention. Procedure-associated Module: Surgical Site Infection (SSI) Event, January 2018. Available at: https://www.cdc.gov/nhsn/PDFs/pscManual/9pscSSIcurrent.pdf (Accessed on January 24, 2018).

Surgical site infection/ epidemiology

- The incidence of SSI varies widely, ranging from 5 to 30 percent depending upon the operative site and wound classification. It is estimated that SSI develops in 2 to 5 percent of patients undergoing inpatient surgical procedures each year in the United States

- Surgical site infection in middle- to low-income countries may be higher compared with high-income countries, possibly related to cumulative risk factors and resource limitations. In the FALCON study, the incidence of surgical site infection was 22 percent for clean-contaminated cases and 30 percent for contaminated/dirty cases in low- to middle-income countries

- 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**

Surgical site infection

Surgical Site Infections in a Tertiary Referral Hospital in Amman: Causative Bacteria and Antibiotic Susceptibility

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Received October 17, 2017; Revised November 5, 2017; Accepted November 11, 2017

Abstract

Surgical site infections (SSIs) represent a major healthcare challenge. This retrospective study aimed to assess the frequency of SSIs, the bacterial profile, and the antibiogram of the isolates, from a tertiary hospital in Jordan. Data regarding SSIs were obtained from hospital records throughout the year 2015. The prevalence rate of SSIs was 5.4%. Gram-negative bacteria were more common than Gram-positive (57% vs. 43%). *Staphylococcus aureus* (35%) and *Escherichia coli* (24.5%) were the most common etiologies. Among Gram-positive isolates resistance was highest for ampicillin, and least for linezolid, teicoplanin, and vancomycin. Among Gram-negative isolates, resistance was highest for ampicillin, ciprofloxacin, and amoxicillin-clavulanate, and least for gentamicin, piperacillin-tazobactam, and meropenem. Surveillance of SSIs, the causative bacteria, and the bacterial antibiograms, are necessary for implementing strict infection control measures, and in the selection of effective antibiotic treatments to decrease the mortality and morbidity rates associated with SSIs. Most SSI cases were detected in individuals aged fifty-five years old or more, and combined with comorbidities, ex. diabetes mellitus.

Keywords: Surgical site infection, Prevalence, Antibiogram, Jordan, *S. aureus, E. coli*.

Useful for surgions to decide the need of antibiotics or prophylactic antibiotics and determine the risk of infection

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.

Reproduced from: Mangram AJ, Horan TC, Pearson ML, et al. Guideline for Prevention of Surgical Site Infection, 1999. Infect Control Hosp Epidemiol 1999; 20:250. Available at: https://stacks.cdc.gov/view/cdc/7160 (Accessed on October 7, 2021).

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Surgical site infection/ diagnosis and management

- **Superficial SSI** – For patients with clinical signs of superficial SSI (eg, localized swelling, warmth, drainage) and active drainage or surgical wound separation, additional imaging is generally unnecessary. Treatment of superficial SSI involves wound exploration and debridement. Antibiotics are administered only if there is associated cellulitis.

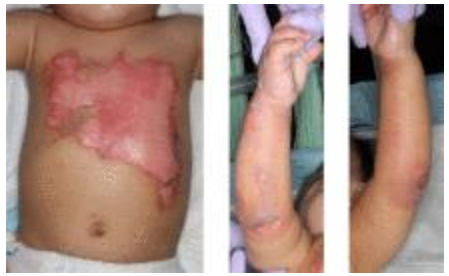
- **Deep incisional SSI** – For patients with clinical signs of deep SSI, which are like those of superficial SSI, imaging (eg, ultrasound, computed tomography) may be helpful to estimate the depth and extent of infection to guide the approach to source control. Treatment of deep SSI requires antibiotic administration and wound exploration/debridement.

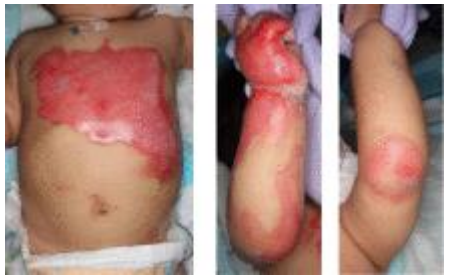
- For sites associated with implanted material, the risk of incomplete treatment of SSI must be weighed against the risks associated with removal of the implanted materials. "Treating through" an infection with intravenous antibiotics without removal of implanted materials may allow disease progression and further deterioration of the surgical site.

Dr didn’t read the rest slides and he said we can read them and ask him about them if we have questions

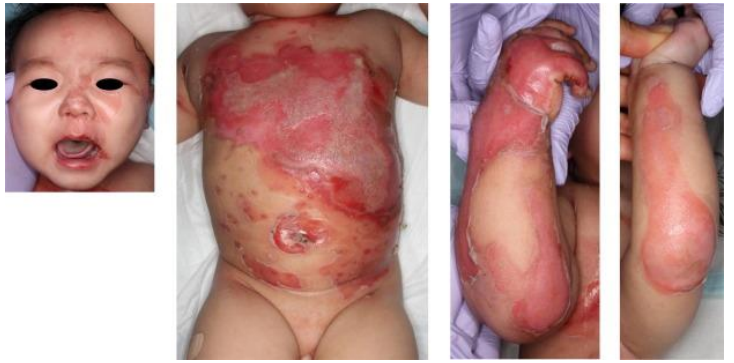
Burn wound infection and sepsis/ Staphylococcal scalded skin syndrome caused by burn wound infection in an infant: A case report

- An 8-month-old boy burned by hot water had partial thickness burns on 10% of his total body surface area. He was admitted to hospital and received fluid therapy and wound care treatment. During hospitalization, he developed a high fever and exfoliation of the skin, except for the burns. He then received antibiotic infusion treatment daily. Three days after initiating antibiotic therapy, he had epithelization of the raw surface, except for his burns. Skin exfoliation affected 36% of the total body surface area.

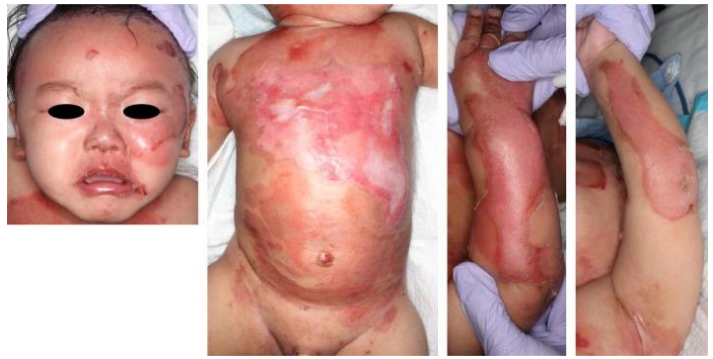




On Day 3, the patient's burn wounds show no infection.



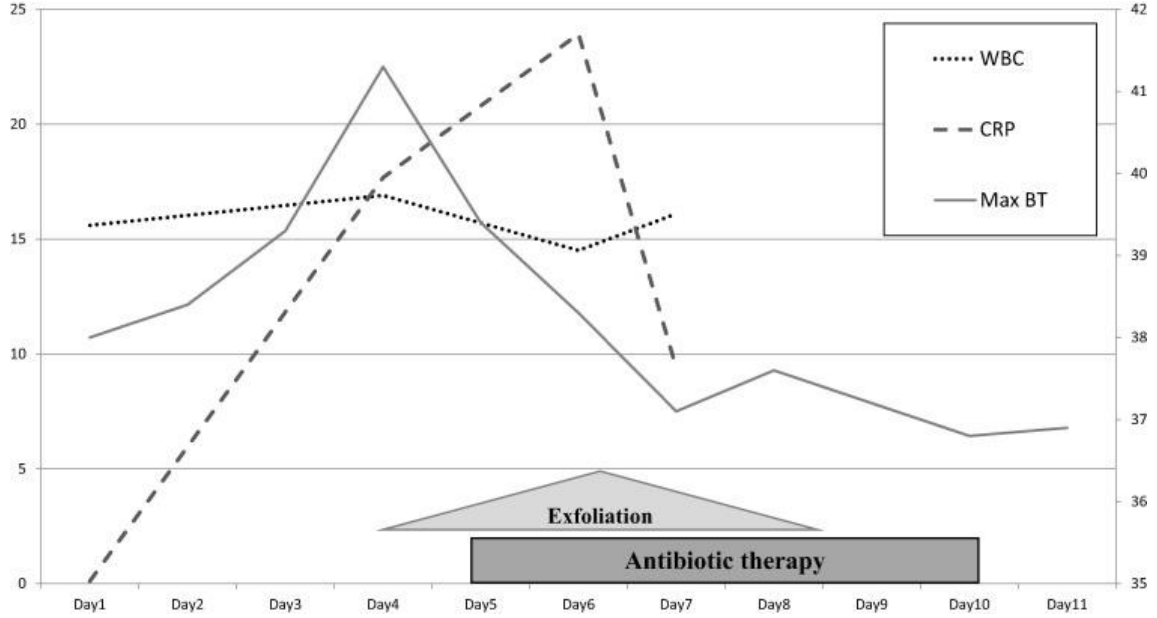
By Day 5, the patient had sudden enlargement of the exfoliation.



By Day 7, the exfoliated area, excluding the burn wound, has formed scabs.

Burn wound infection and sepsis/ Staphylococcal scalded skin syndrome caused by burn wound infection in an infant: A case report

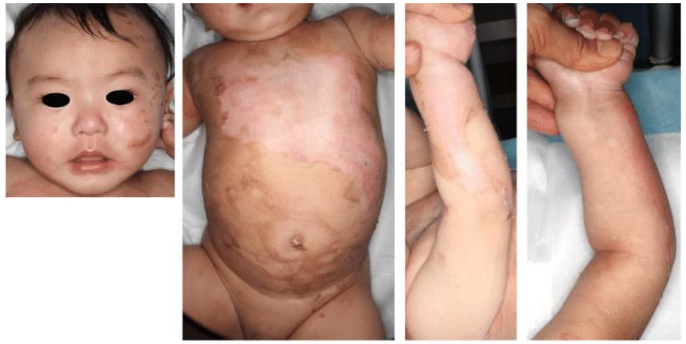
- Staphylococcal scalded skin syndrome developed 3 days after his being scalded.
- His exfoliative areas involved up to 36% total body surface area.
- His wounds without burns epithelized on day 10.
- Only burn wound swabs revealed **exfoliative toxin-positive Staphylococcus aureus.**



Tokyo, Japan) and covered with silver-containing polyurethane foam (Mepilex Ag; Mölnlycke Health Care AB, Götenborg, Sweden) on alternate days. On Day 2, he drank 600 mL water. Therefore, we decided to end fluid therapy on Day 2. On Day 3, his wound began to epithelize without infection (Fig. 2).

On Day 4, sudden epidermal exfoliation occurred on the abdomen and his body temperature rose to 39.3 °C. On Day 5, the exfoliation was larger (Fig. 3) and his body temperature further increased to 41.3 °C. Nikolsky's sign was positive around his chest. His blood examination showed his WBC was 16,900/μL and CRP level was 17.68 mg/dL. We suspected that he had SSSS.

We obtained culture swabs from his throat, arms, and chest, and we collected a blood sample. We started antibiotic therapy, which consisted of an intravenous drip injection of cefazolin and vancomycin. We also initiated fluid therapy because he had dehydration. The fluid therapy (800 mL/day; Ringer's solution) was restarted. On Day 6, all exfoliated areas were on the head and neck, chest, abdomen, both arms, and affected up to 36%TBSA. His blood examination showed his WBC was 14,500/μL and CRP level was 23.93 mg/dL. The CRP level was at its peak during this time. On Day 7, his epidermal exfoliation had become scabs (Fig. 4). The blood examination showed his WBC was 16,100/μL and CRP level was 9.40 mg/dL. On Day 10, his wounds were mostly epithelized (Fig. 5). We finished his antibiotic therapy and fluid therapy. While on fluid therapy, we were able to maintain his urinary volume over 1.5 mL/h. On Day 12, all of his wounds were healed. *Staphylococcus aureus* (ET^+) was cultured from a swab of the chest. *Staphylococcus aureus* (ET^-) and *Streptococcus* species were cultured from the swabs of both arms. No pathogen was cultured from the swabs of the throat or blood sample. We diagnosed SSSS.



All wounds are mostly epithelized (Day 10).

Necrotizing fasciitis

- A 60-year-old woman with a history of pre-diabetes and hyperlipidemia, presented to a local hospital with severe left flank pain and skin discoloration on the same area for 2 days. She had been taking care of her grandson who was diagnosed with streptococcal sore throat. On examination, the patient was hypotensive and tachycardic. There was a large grayish blue area over the left flank, extending to the left lower back (Fig. 1a, b). This area was very tender to palpation. There were no bullae, crepitus, or external drainage. A CT scan of the abdomen revealed a **diffuse inflammatory process** within the skin and soft tissue without any gas in the adjacent area.

- A **diagnosis of necrotizing fasciitis** was pursued. Broad-spectrum intravenous antibiotics (vancomycin, piperacillin–tazobactam, and clindamycin), and vasopressors were initiated, and she was transferred to our institution for further management.

Necrotizing fasciitis

- Upon arrival, she was **immediately taken to the operating room for surgery**. The operative findings showed there were **extensive soft tissue necrosis** and edematous fascial layers. A full-thickness **debridement** of skin and subcutaneous tissue of the left flank (24 cm × 21 cm × 1.5 cm) was performed (Fig. 1c). **The tissue culture was positive for group A β-hemolytic Streptococcus** (Streptococcus pyogenes). The pathogen was sensitive to penicillin (minimal inhibitory concentration, MIC 0.032 mg/L), ceftriaxone (MIC 0.064 mg/L), vancomycin (MIC 1.0 mg/L), and clindamycin (MIC not reported). **Blood cultures were negative** throughout the admission. Antibiotics were tailored to intravenous penicillin and clindamycin

Necrotizing fasciitis

The photos illustrated remarkable progression of streptococcal necrotizing fasciitis of left abdominal wall and left axilla (a through h). i, j represented pre- and post-skin grafts. Days were calculated from the first day of hospitalization

Further reading:

The following titles can be found on Uptodate:

- Infectious complications of pressure-induced skin and soft tissue injury
- Burn wound infection and sepsis
- Surgical site infection