Gangrene (myonecrosis, gas gangrene, flesh eating disease, necrotizing erysipelas, phagedena, putrid ulcer, Meleney’s gangrene, Fournier’s gangrene, gangrenous stomatitis, cancrum oris)

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**Are You Confident of the Diagnosis?**

- **What you should be alert for in the history**
  Gangrene is a life-threatening condition characterized by necrosis of a significant amount of body tissue caused by ischemia (dry gangrene), infection (gas gangrene), or both (wet gangrene); rather than by injury, tumors, vascular thrombosis, or snake envenomation. At the onset, patients usually complain of spontaneous intense local pain disproportionate to the physical findings, including the normal appearance of the skin. Violaceous bullae, cutaneous hemorrhage, skin sloughing, skin anesthesia, and gas in the tissue can successively appear.
Due to toxemia and systemic inflammatory response syndrome (SIRS), progression of disease is often very rapid (within hours).

- **Characteristic findings on physical examination**
  Symptoms begin locally, but at the outset, signs of inflammation may not be apparent if the bacteria are deep within the tissue. The skin may appear white, shiny, and tense; however, it is often absolutely normal. If the process is not deep, signs of inflammation such as redness and swollen or hot skin emerge very quickly. With the progression of disease, swelling develops, and the skin can become a violet to dusky bronze color. Blisters with dark-red fluid may form, resulting in subsequent necrosis of the subcutaneous tissues ([Figure 1](#)).

**Image Caption:** Figure 1. Necrotizing fasciitis upon chronic venous ulcers of the leg. The skin appears extensively dusky bronze for necrosis, with formation of bullae.
If broken, the bullae release a brown watery discharge with a bad smell. Bacteria release enzymes (such as hyaluronidase) and lipases that degrade connective tissue and fat, allowing the bacteria to spread along fascial planes. Thrombosis of dermal vessels can be caused by a local toxin-induced ischemia, together with subcutaneous necrosis and superficial nerve destruction. This causes anesthesia of the overlying skin.

Lymphangitis and lymphadenitis are rare. Crepitus, if present, is a late finding and is most frequent in type I infections in diabetics. Systemic manifestations include signs of bacteremia such as fever, tachycardia, hypotension, tachypnoea, irritability, and prostration. Patients may appear apathetic, with varied levels of confusion, stupor, delirium, and coma (toxic shock syndrome), manifesting the hallmarks of multi-organ failure (MOF).

- **Expected results of diagnostic studies**
  Routine laboratory tests may reveal metabolic acidosis, leukocytosis, anemia, thrombocytopenia, high levels of creatinine kinase, myoglobinemia and myoglobinuria, and coagulopathy (ie, disseminated intravascular coagulation). A Gram stain of the bullae fluid may show pleomorphic gram-positive bacilli, with or without spores. Blood cultures may identify the organism responsible. A definitive bacteriologic diagnosis is established by culture of tissue specimens obtained during surgery.

  The surgical debridement of the involved area reveals muscles that do not bleed when cut, do not retract when pinched, and appear cooked and dead.

  Histologic examination shows underlying cause of infection. Bedside goal-directed ultrasound examination can show fluid collection along fascial plane and illustrate irregularity of the fascia (Figure 2).

  The fascia can seem broken by a hypoechoic irregular area, with blurred contours and marked edge shadowing arising from deep subcutaneous tissue that corresponds to the region of maximum pain (Figure 3).

  Myonecrosis could be detected by the disappearance of the regular arrangement of muscular architecture (Figure 4, Figure 5). If the organism responsible is gas-forming, the ultrasound investigation can reveal the presence of many hyperechoic spots without shapes, occasionally involving subcutaneous tissue, which converged and produced distal artifacts such as comet tails, easily attributable to gas origin.

  Color doppler ultrasonography is useless in these cases because it is only able to detect a reduced vascularization in an affected area, but this is a nonspecific sign, as even a rise of vascularization in surrounding areas may be seen.
Plain radiography may reveal gas in the tissues; it is more sensitive than physical examination for crepitis.

Magnetic resonance imaging (MRI) helps to differentiate acute cellulitis from necrotizing fasciitis, but it takes much time to be performed and often, given the rapid progression and fatal outcome of these conditions, it is not justified if used, and delays treatment.

Computed tomography (CT) scan findings may be minimal in the early stage; however, CT remains the main imaging modality to detect even small fluid and/or air collections (Figure 6) and asymmetric fascial thickening (Figure 7). While examining the area of interest, it is crucial to evaluate surrounding vessels for their patency for the prompt recognition of a compartment syndrome.

Image Caption: Figure 2. Bedside goal-directed ultrasound examination of polymicrobial (type I) necrotizing fasciitis. Fluid collection (asterisk) along fascial plane (arrows), with thickness and irregularity of the fascia, are shown.
Figure 3. Bedside goal-directed ultrasound examination in a case of polymicrobial (type I) necrotizing fasciitis at the onset, in a 65-year-old patient suffering from metastatic colon cancer, without skin modifications. The image shows a hypoechoic irregular area with blurred contours and marked edge with shadows (asterisk), arising from fascial plane (arrow) and corresponding to dense infiltration of neutrophils and fat necrosis; this area seems to break the fascial plane in the posterior side of left calf.
Figure 4. Bedside goal-directed ultrasound examination on longitudinal scanning detects non-clostridial myonecrosis of right shoulder after a therapeutic injection procedure in a 72-year-old man. Box A: The disappearance of the regular arrangement of muscular architecture is shown; it presents a diffuse increased echogenicity and appears with a “clod pattern” due to infiltrative foci and initial colliquative necrosis (asterisk). Box B: normal parallel arrangement of muscular fibers (asterisk) on longitudinal scanning of contralateral arm. Fascial plane upwards (empty arrow) and homerus periosteum surface downwards (full arrow) are indicated on both boxes.
Figure 5. Bedside goal-directed ultrasound examination on longitudinal scanning detects non-clostridial myonecrosis of right shoulder after a therapeutic injection procedure in a 72-year-old man. Box A: The disappearance of the regular arrangement of muscular architecture is shown; it presents a diffuse increased echogenicity and appears with a “clod pattern” due to infiltrative foci and initial colliquative necrosis (asterisk). Box B: normal parallel arrangement of muscular fibers (asterisk) on longitudinal scanning of contralateral arm. Fascial plane upwards (empty arrow) and homerus periosteum surface downwards (full arrow) are indicated on both boxes.
Figure 6. CT scan image detecting the wide air infiltration of the posterior part of the arm (triceps muscle), involving even subcutaneous tissue in a 51-year-old woman with rapidly evolutive spontaneous gas gangrene by Clostridium septicum.
• **Diagnosis confirmation**

Even for the most experienced physicians, clinical findings are not precise enough to make diagnosis, so both clinical examination and diagnostic tools should be used.

Diagnosis is based on clinical findings and on a strong suspicion. The main clinical findings are severe constant pain, bullae, skin necrosis or bruising that precedes skin necrosis, gas in the soft tissues, edema that extends beyond the margin of erythema, cutaneous anesthesia, systemic toxicity (fever, leukocytosis, delirium, renal failure), and rapid spread and worsening, even during antibiotic treatment. A strong suspicion should rise from the presence of predisposing factors such as trauma and immunosuppression or tumors (cancer).

Gas gangrene should be distinguished from other nonclostridial deep soft-tissue gas-forming infections.

Wong et al. created a score to distinguish necrotizing soft-tissue infections from non-necrotizing ones. This score is based on six independent laboratory variables; each gives a specific number of points toward the final score. The total score ranges from 0 to 13. Patients were divided into three groups according to the risk of necrotizing soft tissue infection: low (≤ 5 points), intermediate (6-7
points), and high (≥8 points). This score provides an important tool in distinguishing and managing necrotizing soft tissue infections, but it is useful only if associated with a strong suspicion of severe infection.

Among other signs, crepitance, even if a late sign, should be also differentiated from subcutaneous emphysema, pneumomediastinum, and fractured larynx, trachea, or bronchi.

The presence of subcutaneous gas does not necessarily indicate a clostridial infection, as E coli, Peptostreptococcus species, and Bacteroides species may produce gas under appropriate conditions. Misleading subcutaneous gas can also result from the undermining of tissue planes during surgical debridement (Figure 8). Air microbubbles can contaminate penetrating wounds up to several hours after the trauma; ultrasound examination can define the characteristics of the gas bubbles in order to distinguish a normally healing wound from a life-threatening infection.

**Image Caption:** Figure 8. Bedside goal-directed ultrasound examination reveals the presence of many hyperechoic spots involving fascial plane (arrow), which converged and produced distal artifacts such as comet tails (asterisks), easily attributable to gas collection by external contamination after fasciotomy (same case depicted in Figure 3).
Who is at Risk for Developing this Disease?

General risk factors are diabetes mellitus, peripheral vascular disease, intravenous drug use, alcoholism, immunodeficiency, immunosuppression, malnutrition, obesity, and old age. Particular conditions leading to the possible development of gangrene are trauma with open and dirty wounds, animal bites, burns, needle stick injury, and vascular problems.

The epidemiology of this kind of disease has changed during recent years. There has been a reduction in the number of cases related to war injuries, probably due to better wound management and the availability of antibiotics. Conversely, there has been an increase in the number of spontaneous cases of gangrene, caused primarily by Clostridium septicum, because of the increasing number of people with immunodeficiencies (caused by sepsis, malignancy, HIV, chemotherapy, steroids, immunosuppressors, etc.).

Approximately 1000 cases of gas gangrene are reported each year to the Centers for Disease Control and Prevention.

What is the Cause of the Disease?

- Etiology
  
  Because of the microbiological findings, gas gangrene or clostridial myonecrosis could be included among necrotizing fasciitis type I. Type I necrotizing fasciitis is classified under polymicrobial etiology and type II to group A streptococci.
  
  Type I, or polymicrobial necrotizing fasciitis, usually is associated with trauma or surgery in the abdomen, perineum, or external genitalia.

  This type I fasciitis includes all non-group-A streptococci infections and at least one or more anaerobes (Bacteroides, Peptostreptococcus, or Clostridium) or Enterobacteriaceae (Escherichia coli, Klebsiella, or Proteus). The percentages of their presence in cultures are: Enterococcus (100%), Staphylococcus (71%), α-Streptococci (57%), E. Coli, Klebsiella, Proteus, and Bacteroides (43%). Less common are Peptostreptococcus, Bacillus, Citrobacter, Enterobacter, and Morganella (14%). In specific cases, normal commensals become virulent, aiding in further spreading of the infection. For example, in Fournier’s gangrene, Escherichia coli and Candida albicans have been isolated from the wound, together with lactobacilli, coliforms, Klebsiella, streptococci, staphylococci, clostridia, Bacteroides, and corynbacteria.

  In gangrenous stomatitis, Fusobacterium necrophorum and Prevotella intermedia can interact with other bacterial organisms such as Borrelia vincentii, Porphyromonas gingivalis, Tannerella forsynthesis, and Treponema denticola. This form, usually mild in presentation, may initially be
underestimated and mistaken for a simple wound cellulitis. However, associated symptoms such as severe pain and systemic toxicity reflect widespread tissue necrosis underlying apparently normal skin. This process may also be observed in association with urogenital or anogenital infections (Fournier’s gangrene).

Another variant of necrotizing fasciitis type I is “saltwater necrotizing fasciitis” in which an apparently minor skin wound is contaminated with saltwater containing a Vibrio species. Vibrio vulnificus infection is, however, due to raw oyster ingestion. Usually, it needs some underlying conditions to develop such as cirrhosis, alcoholic liver disease, gouty arthritis, chronic renal failure, diabetes mellitus, or long-term treatment with steroids.

Its pathogenicity is caused by the lack of neutrophil and macrophage functionality. In cirrhotic patients, these marine organisms can enter the bloodstream, gaining access by the gastrointestinal tract and escaping phagocytosis by the Kupffer cells of the hepatic reticuloendothelial system because of shunting through the portal-systemic circulation. Due to this easy way to evade host defense, V. vulnificus can spread rapidly, causing septic shock and leading to a fatal sepsis quicker than that caused by Streptococcus.

Type I necrotizing fasciitis can include gas gangrene or clostridial myonecrosis. However, this form could represent an independent entity, sometimes classified as type III necrotizing fasciitis. The most common bacteria responsible for gas gangrene are Clostridium species. Among them, Clostridium perfringens, Clostridium histolyticum, and Clostridium novyi are the most common. These organisms are often associated with recent surgery, severe penetrating trauma, or crush injuries that interrupt blood supply.

Clostridium perfringens and C. novyi have recently been described among heroin abusers, following intracutaneous injection of black tar heroin. A rapidly progressive necrotizing fasciitis following a stonefish sting has also been described. Clostridium septicum, more aerotolerant, is the most common cause of spontaneous gas gangrene, especially in patients with colonic lesions (i.e. diverticular disease, inflammatory bowel disease), adenocarcinoma, neutropenia, and any case of immunosuppression.

Type II infections typically involve group A Streptococcus (Streptococcus pyogenes) as monomicrobial fasciitis or in combination with a Staphylococcus organism. This kind of fasciitis is the so-called “flesh-eating bacterial infection.” Historically, monomicrobial necrotizing fasciitis by S. aureus is uncommon, except in neonates. However, recently another serious form of monomicrobial necrotizing fasciitis has been observed with increasing frequency, caused by methicillin-resistant Staphylococcus aureus (MRSA) strains associated with purpura fulminans.
**Pathophysiology**

The first mechanism of pathogenesis is the direct inoculation of the germ through an open wound, with crushed or ischemic edges that promote an anaerobic environment. The second is the hematogenous spread, most common in immunocompromised patients. The incubation period for gas gangrene is 1 to 4 days; the onset of the disease being estimated in 6 to 24 hours.

Anaerobic bacteria, usually in combination with aerobic gram-negative organisms, proliferate in an environment of local tissue hypoxia. Facultative aerobic organisms grow because polymorphonuclear (PMN) leukocytes exhibit decreased function under hypoxic wound conditions. This growth further lowers the oxidation/reduction potential, enabling more anaerobic proliferation, thus accelerating the disease process.

Carbon dioxide and water are the end products of aerobic metabolism. Hydrogen, nitrogen, hydrogen sulfide, and methane are produced from the combination of aerobic and anaerobic bacteria in a soft-tissue infection. These gases, except carbon dioxide, gather in tissues because of reduced water solubility. The synergistic activity of aerobes and anaerobes also leads to the production of various exotoxins and enzymes such as collagenase, heparinase, hyaluronidase, streptokinase, and streptodornase, which aid in tissue destruction.

The platelet aggregation and complement fixation induced by the aerobes, and the heparinase and collagenase produced by anaerobes, lead to microvascular thrombosis, especially of the deep blood vessels that traverse the fascia or muscle compartments. In addition, the phagocytic activity is impaired in the necrotic tissue, aiding in further spread of the infection.

Clostridia are large spore-forming gram-positive anaerobic organisms, usually found in the human gastrointestinal tract and female genitourinary system, as well as in soil. They produce over ten exotoxins, among which the most aggressive is the α-toxin that produces hydrolization of cell walls, tissue necrosis, leukocytes inactivation, hemolysis, and a direct cardiodepression.

Streptococcus pyogenes bases its pathogenicity on M proteins types 1 and 3, present on its cell membranes, which allow it to adhere, colonize, and then invade the host. It also produces exotoxins A and B, mitogenic factors that, acting as superantigen, stimulate the immune system to cause cytokine storm, leading to shock, organ failure, and myocardial and immune depression.

**Systemic Implications and Complications**

Gas gangrene can be difficult to recognize in its early stages, but it leads very rapidly to septic shock. The expected clinical course varies from patient to patient. The mortality rate for necrotizing
fasciitis ranges from 16%-24%, and the overall morbidity and mortality for gas gangrene is estimated at about 60%.

Anaya et al. worked out a score to categorize patients on their risk of mortality. Variables included age greater than 50 years, white blood count (WBC) greater than 40,000 cells/mm3, hematocrit greater than 50%, heart rate greater than 110 beats/min, temperature greater than 36°C, and creatinine level greater than 1.5 mg/dl. Patients are divided into three groups. This prognostic score helps in distinguishing patients at high risk who may benefit from novel therapeutic management.

The main complications are renal failure, septic shock, and multi-organ failure; for survivors, marred scars with cosmetic deformity and limb loss are possible. Therefore, affected patients require immediate aggressive fluid resuscitation, and antibiotic therapy should be started in the Emergency Department, using crystalloid, plasma, and packed cells to restore red blood cells lost by hemolysis and to correct hypotension. Vasoconstrictors should be avoided because of the possible reduction of blood support to already ischemic muscles.

**Treatment Options**

Treatment options are summarized in [Table I](#).

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<th>Surgical</th>
<th>Medical - Antibiotic</th>
<th>Physical - Hyperbaric Oxygen Therapy (HBO)</th>
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<td>Prompt and aggressive surgery that may include fasciotomy, debridement, or amputation. To guide the width of debridement, it is important to look at the muscles.</td>
<td>Penicillin 2-4MU every 4 hours intravenously + Clindamycin 600-900mg/kg every 8 hours intravenously + (optional) Meropenem 1g every 8 hours intravenously Metronidazole 500mg every 8 hours intravenously Vancomycin (for resistant strains) 30mg/kg/day in 2 divided doses intravenously</td>
<td>HBO therapy should be undertaken after the aggressive surgical debridement and should never delay surgery it consists of 100% oxygen at 3atm of pressure for 90 minutes immediately following surgery, with 3 dives in the first 24 hours, followed by 2 dives a day for 4-5 days.</td>
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The first step of the therapeutic ladder for the management of gas gangrene is the prompt and aggressive surgical debridement of the necrotic and infected soft tissues and muscles. The width and extension of the debridement is guided by the muscle appearance, and varies from a simple debridement, to fasciotomy, up to amputation. The incision should be made directly over the affected area, parallel to the neurovascular bundles, and carried down to the deep fascia.

Necrotic tissue seems dull grey and avascular, with brown dishwater-like fluid. Once opened, the fascial planes underlying muscles should be inspected, then all necrotic and nonviable tissues must be removed. The debridement should be extended until the subcutaneous tissue doesn’t split easily from the deep fascia. During surgery, tissue and fluid samples should be collected and sent for histopathological and microbiological analysis.

The second step, even if it is synchronous to the previous one, is to start therapy with a wide-spectrum antibiotic. The first choice is a combination of high-dose penicillin and clindamycin, which seems to suppress toxins and modulate cytokine (i.e. tumor necrosis factor) production. In addition, especially when there is no information on the organisms responsible for the infection, meropenem, metronidazole, or vancomycin (for resistant strains) should be added. In patients with penicillin hypersensitivity, clindamycin or metronidazole with an aminoglycoside or fluoroquinolone are recommended.

Mortality rates for this kind of infection remain high, especially in cases due to Aeromonas or Vibrio. In these cases, faster management and an earlier surgical debridement are required. A “second look” surgery, usually done within 12-24 hours of the first one, is often needed.

Intravenous immunoglobulins (IVG) are used to aid the response to streptococcal superantigens. This treatment seems to supply clinical benefit in terms of increased survival but because of the low number of patients treated with iv immunoglobulins, this treatment is not yet a common recommendation.

HBO should be initiated soon after surgical debridement, but the rarity of immediately available hyperbaric chambers reduces its use. HBO means the use of oxygen at increased pressure in a monoplace or a multiplace chamber. Potential benefits are increasing the normal oxygen saturation in the infected area by a thousand-fold, leading to a bactericidal effect, improved polymorphonuclear (PMN) function, and improved wound healing.

Some authors have noted a higher oxygen saturation (PO2) in infected necrotic tissue secondary to HBO-induced vasodilation. No large controlled randomized studies have been published to support
the complete effectiveness of HBO in necrotizing fasciitis. Some authors suggest that HBO is able to shorten the time of treatment. Other authors state that oxygen could provoke side effects due to the specific oxygen toxicity and to high atmospheric pressure. However, HBO therapy should never delay surgery. The rapid progression of this disease determines the poor prognosis.

Potential side effects of HBO include claustrophobia, tympanic membrane rupture, seizures, and central nervous system oxygen toxicity. A typical treatment protocol involves HBO, given aggressively after the first surgical debridement. Three treatment sessions, in a multiplace chamber at 3 atmosphere absolute (ATA), 100% oxygen, for 90 minutes each, can be given in the first 24 hours. Appropriate air breaks are given, as necessary. In a monoplace chamber, 2.5-2.8 ATA, 100% oxygen, for 90 minutes per session, can be given. On the second day, twice daily treatments can ensue until granulation is obtained (to a total of 10-15 treatments).

### Patient Management

Continued medical care in the form of a multidisciplinary approach is necessary. After the prompt debridement, the patient needs aggressive fluid resuscitation and blood component therapy, and scrupulous intensive care unit (ICU) management, in order to avoid multi-organ failure (MOF). ICU management should include obtaining intravenous access, taking care to not use an infected extremity, placing the patient on continuous cardiac monitoring, and placing a Foley catheter to monitor urine output (this procedure should probably be avoided in patients with Fournier gangrene).

Antibiotic therapy should be continued until the patient's condition improves to the point that the patient no longer requires antibiotics. Antibiotics are narrowed down based on the first results of blood, wound, and tissue cultures, but should be continued at least for 48 hours after temperature and white blood cell (WBC) normalization, or after stabilization of the clinical picture. In some cases, surgery could be devastating and sometimes reconstructive procedures of the affected site are needed.

Appropriate nutritional support is required until the last day of admission and beyond. These patients lose a huge quantity of proteins and fluid from their wounds and, in general, they need twice their basal caloric requirements to replace this loss. For this reason, a nasogastric tube for feeding is required to provide adequate enteral nutrition.

Once the first debridement has been performed, further debridements at intervals of 6-48 hours are recommended until no more necrosis or infected tissue is present. Serial photography taken during the different phases of the infection could allow identification of a demarcation zone of cellulitis or necrotic tissue that can be used as a monitor to avoid insidious spread of infection.
When the infection is controlled, wound management becomes very important. Daily dressings under sedation, sometimes followed by secondary suturing of the wound, help to avoid secondary infection, promote granulation tissue formation, and drain inflammatory exudates. Postoperatory dressings with hydrogel and alginate have been used for this reason. If the debridement has created a vast scar, conforming foam dressings can be inserted to fill the dead space. This dressing should be changed frequently, but the procedure is very painful for the patient, and sometimes a general anesthetic is required.

Another technique for wound management is topical negative pressure (TNP) therapy. This can reduce edema fluid and stimulate granulation tissue formation as a result of the applied tension forces. TNP is even able to reduce bacterial growth and help tissue expansion, aiding the wound closure. Although expensive, a vacuum-assisted wound-closing (VAC) device could be used in nonhealing limb wounds, with reduced morbidity in comparison to other conventional techniques.

If your hospital is not equipped to handle the aggressive care, monitoring, or serial surgical debridements that these patients require, then arrangements for transfer should be made. But only hemodynamically stable patients should be considered for transfer.

**Unusual Clinical Scenarios to Consider in Patient Management**

Spontaneous or idiopathic cases represent approximately 10% (7%-27%) of all cases of gas gangrene, being nontraumatic and almost exclusively caused by Clostridium septicum, a common host in gastrointestinal and genitourinary tracts and often associated with malignancy or immunodeficiency. Its detection could be delayed and lead to a bad prognosis, due to the lack of the strong suspicion and to the poor clinical findings at the onset. Unfortunately, from an insidious onset, the disease can develop a fulminant progression.

**What is the Evidence?**

(The chapter analyzes all soft-tissue infections. The first paragraph describes necrotizing soft-tissue infections, including gas gangrene, streptococcal myositis, necrotizing fasciitis, and necrotizing cellulitis. Then, cellulitis, erysipelas, and cutaneous abscesses are examined.)

Sixteen patients with necrotizing fasciitis were monitored for clinical observation and for laboratory specimen collection, preservation, and culture. Two clear-cut groups of culture and Gram stain results were found, suggesting that the clinical entity of necrotizing fasciitis can occur after infection by different infecting organisms. Necrotizing fasciitis can be divided into type 1, a polymicrobial infection, and type 2, sustained by group A Streptococcus with or without coexisting staphylococcal infection.)

(This article includes the Infectious Diseases Society of America guidelines for the diagnosis and management of skin and soft-tissue infections. Both superficial and deeper infections are examined, and the best management options and antibiotic choices are discussed. Moreover, particular cases such as infection in the immune-compromised host and soft-tissue infections following animal contact are examined.

(In this work, the authors reviewed the records of 843 patients whose wound cultures grew MRSA. Among this cohort, fourteen were identified as patients presenting from the community with clinical and intraoperative findings of necrotizing fasciitis, necrotizing myositis, or both. The authors assert that necrotizing fasciitis caused by community-associated MRSA is an emerging clinical entity, requiring antibiotics predictably active against this pathogen, especially in areas in which community-associated MRSA infection is endemic.

(This work, a review of the literature based on the authors’ clinical experience, proposes a new classification based on clinical presentation and suggests an algorithm to facilitate the management of this devastating condition. Increasing awareness should be given to the management of the large wounds resulting from the surgical debridement of necrotizing fasciitis.)

The chapter describes all soft-tissue infections, including cellulitis and abscesses such as hydradenitis suppurativa, carbuncle, and Bartholin’s cyst abscess. Then, necrotizing fasciitis and myositis are described.

(This work describes a case of spontaneous gas gangrene in the arm of a patient suffering from spondylitis. Diagnosis was made by ultrasonographic evaluation of the overthrow of the normal muscular architecture and the presence of air in soft tissues. Cultures of the tissue specimens and pus reveal Clostridium septicum.)

(The authors develop a novel diagnostic scoring system for distinguishing necrotizing fasciitis from other soft-tissue infections, based on laboratory tests routinely performed for the evaluation of severe soft-tissue infections: the Laboratory Risk Indicator for Necrotizing Fasciitis (LRINEC) score. The LRINEC score was constructed by converting into integer the regression coefficients of independently predictive factors in the multiple logistic regression model for diagnosing necrotizing fasciitis. The cutoff value for the LRINEC score was six points, with a positive predictive value of 92.0% and negative predictive value of 96.0%. The authors conclude that the LRINEC score is a robust score, capable of detecting even clinically early cases of necrotizing fasciitis. Patients with a LRINEC score of greater than or equal to six should be carefully evaluated for the presence of necrotizing fasciitis.)

(The study reports the possible air contamination of penetrating wounds. It describes sonographic findings to distinguish contaminating air from bacterial gas production. This work underlines the importance of point-of-care ultrasound examination.)

(The aim of this work is the identification of patients at high risk of mortality for selection of those that may benefit from future novel treatments and for development and comparison of future trials. The laboratory risk indicator for necrotizing fasciitis score can be helpful for distinguishing between cases of cellulitis, which should respond to medical management alone, and necrotizing soft-tissue infection, which require operative debridement in addition to antimicrobial therapy.)