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Gas Gangrene

Jerome B. Buboltz; Heather M. Murphy-Lavoie.

Author Information and Affiliations

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Continuing Education Activity

Gas gangrene is a highly lethal infection of soft tissue caused by Clostridium species, with Clostridium perfringens being the most common. It is synonymous with myonecrosis and is characterized by rapidly progressive gangrene of the injured tissue and the production of foul-smelling gas. This activity explains the evaluation and management of gas gangrene and highlights the role of the interprofessional team in improving care for patients with this condition.

Objectives:

- Identify the etiology of gas gangrene.
- Assess the evaluation of gas gangrene.
- Evaluate the use of antibiotics, early surgical debridement, intravenous fluid resuscitation, ICU monitoring, and hyperbaric oxygen therapy in the management of gas gangrene.
- Communicate the importance of collaboration among the interprofessional team to improve outcomes for patients affected by gas gangrene.

Access free multiple choice questions on this topic.

Introduction

Gas gangrene is synonymous with myonecrosis and is a highly lethal infection of deep soft tissue caused by *Clostridium* species. *Clostridium perfringens* is the most common (see **Image.** Gas Gangrene of a Diabetic Foot). *Clostridial myonecrosis* historically was a common war wound infection with an incidence of 5%. Still, with improvement in wound care, antisepsis, and the use of antibiotics, the incidence has fallen to 0.1% of war-related wound infections since the Vietnam War era. Puncture and surgical wounds, especially GI surgeries done on the biliary tract or intestinal surgeries, are causes of clostridial infections due to inadvertent inoculation of the surgical wound with gut bacteria.[1][2]

Etiology

Clostridial infections usually arise in traumatized tissue but also can arise spontaneously. The infection involves deeper tissue, such as a muscle, which can lead to a rapidly spreading infection along tissue planes, and patients often present with sepsis. The infection may develop hours to weeks after the initial trauma and inoculation. The inoculation of the bacteria does not always cause gas gangrene, and there are host and organism factors that determine the progression to infection. Immunocompromised patients and those with local tissue hypoxia (due to trauma or poor vascular supply) are most at risk. The most common organisms that cause these

infections are C*lostridium perfringens, Clostridium septicum,* and *Clostridium histolyticum*. *C. septicum* is the most common cause of spontaneous gas gangrene associated with G.I. abnormalities, such as colon cancer. *C. perfringens* and *C. histolyticum* are more commonly associated with post-traumatic infections.[3][4]

More recently, in the literature, *Clostridium sordellii*, an uncommon pathogen, has been reported to cause fatal shock syndrome and gas gangrene of the uterus after medical abortion with oral mifepristone and vaginal misoprostol. *Clostridium sordellii* is also on the rise associated with the use of black tar heroin injections, more commonly referred to as “popping.” This organism has also had increased incidence as the cause of deep tissue infections associated with childbirth and infections after gynecologic procedures, including septic abortions, which can cause gas gangrene of the uterus.[1][5][6]

Epidemiology

In the United States, the incidence of myonecrosis is only about 1000 cases per year. The incidence is probably higher in less developed countries with decreased access to healthcare and antibiotics, but the exact number is unknown. With the best of care, including early recognition, surgical care, antibiotic treatment, and hyperbaric oxygen therapy, the overall mortality rate is 20% to 30% and, in some studies, as low as 5% to 10%; however, if not treated, the disease has a 100% fatality. Host factors such as an immunocompromised state, diabetes mellitus, and spontaneous infections can have higher mortality rates of 67% or higher. If the infection involves the abdominal soft tissue or chest wall, the mortality rate can be as high as 60% compared to extremity infections with more favorable mortality of 5% to 30%.[7][8]

Pathophysiology

C. perfringens causes 80% to 90% of gas gangrene cases, but other species can cause infection. In order of prevalence, they are *Clostridium novyi* (40%), *C. septicum* (20%), *C. histolyticum* (10%), *Clostridium bifermentans* (10%), *Clostridium fallax* (5%), and *C. sordellii*. These organisms are in the soil and organic waste, especially if contaminated with fecal material.

Healthcare workers should suspect gas gangrene if anaerobic gram-positive bacilli are present in a wound with necrosis of soft tissue and muscle. The organisms produce a gas identifiable on X-ray or CT scans. Only about 5% of the wounds colonized with clostridial organisms develop an infection. Therefore, host factors and the anatomic location of the organism's injection help determine whether the bacteria develop into a clostridial myonecrosis infection. For example, a deep penetrating wound into muscle tissue where the host is immunocompromised is more likely to develop an infection than a host with a healthy immune system and good nutritional status. More open superficial wounds are less likely to become infected, especially if properly cleaned and dressed, compared to deeper penetrating wounds or wounds with crush injury and tissue ischemia.[9]

The clostridial organisms produce alpha and theta toxins that cause extensive tissue damage. The infection can spread quickly, and within a matter of several hours, the patient may develop overwhelming shock, sepsis, and death. A better-oxygenated tissue with 70mmHg oxygen tension inhibits the organism's growth because clostridial species are facultative anaerobes. A facultative anaerobe is an organism that makes ATP by aerobic respiration if oxygen is present but can switch to fermentation if oxygen is absent. If the oxygen tension of the tissue is less than 30 mm Hg, the clostridial organisms grow more quickly. The infection can develop slowly over

weeks or rapidly over hours, depending on the oxygen tension of the tissue and the amount of organisms inoculated.[10]

The virulence of the organism depends on the exotoxins produced; Clostridium perfringens is the most pathologic with 17 known toxins, with the most toxic being the alpha-toxin, a lecithinase. Alpha toxin is a phospholipase (lecithinase) that breaks down cell membranes, triggering platelet aggregation, thrombosis, and histamine release. Also present are collagenase, hyaluronidase, hemagglutinins, and hemolysins. Theta toxins cause direct vascular injury and breakdown of leukocytes, causing a blunted host inflammatory response to the infection. Collagenase breaks down connective tissue, allowing the organism to spread rapidly across tissue planes. This is 1 of the main reasons the infection can cross over connective tissue plains, spreading into the deeper muscle tissues.[5][11][12]

Histopathology

Clostridium's Gram stain shows large gram-positive rods with a paucity of leukocytes (as is typical of anaerobic infections).

Toxicokinetics

Common toxins produced by *C. perfringens*:

- Alpha toxin: Lecithinase (or phospholipase) that breaks down cell membranes, resulting in cell death and tissue necrosis. This toxin is also hemolytic and cardiotoxic.
- Beta toxin: Necrosis of tissue
- Delta toxin: Hemolysin
- Epsilon toxin: Acts to increase cell membrane permeability; permease.
- Iota toxin: Necrosis of tissue
- Kappa toxin: Collagenase, gelatinase, and necrosis of tissue. It especially destroys blood vessels and connective tissue.
- Lambda toxin: Protease
- Mu toxin: Hyaluronidase
- Nu toxin: Deoxyribonuclease, hemolytic and necrosis of tissue
- Phi toxin: Hemolysin, cytolysin[13]

History and Physical

Patients with gas gangrene (myonecrosis) present with signs of infection such as fever, chills, pain, and less superficial inflammation at the site of infection than one would expect, given the deep penetrating nature of these infections. The condition of the patient can rapidly progress to sepsis and death if not treated aggressively. The wound discharge is often dishwater-looking with a musty order. It can involve the vasculature that supplies large areas of infected tissue, leading to subcutaneous fat necrosis down to the fascia and extending into the deeper muscle. If the nerves are damaged, the severity of the pain is less than expected for the extent of infection. The drainage from the necrotic tissue often has a dishwater appearance and musty order. Signs of severe sepsis include septic shock, adult respiratory distress syndrome, disseminated

intravascular coagulation, and hemolysis, which may cause hemolytic anemia, which is often how patients present. Any patient with a cellulitis infection who develops additional signs of crepitus secondary to gas in the tissue and necrotic or dusky-looking skin should be evaluated for gas gangrene.[14][15][16]

Evaluation

Immediate workup of a patient with suspected gas gangrene includes CBC, CMP, urinalysis, PT, APTT, blood, and wound cultures. Additional blood tests such as ABG, lactic acid, and pre-calcitonin can be helpful in the evaluation of sepsis, which is often present in gas gangrene. Common imaging studies include X-rays, CT scans of the infected body part, and ultrasound. These can help identify the extent of the infection, abscess, and gas in the tissues. See **Image.** Gas Gangrene. Extensive lab and imaging should not delay definitive surgical debridement of the necrotic tissue. During the initial surgical debridement, a deep-wound aerobic and anaerobic culture can help determine the causative organism and direct antibiotic therapy.[17]

Treatment / Management

Because the infection is rapidly progressive, it is important to treat patients aggressively with antibiotics, early surgical consultation with debridement, intravenous fluid resuscitation, ICU monitoring, and adjuvant hyperbaric oxygen therapy. Getting early surgical consultation without delay is important as this is a true surgical emergency. Providers should not delay antibiotics to get cultures but begin empiric treatment with antibiotics. Reasonable broad-spectrum coverage includes vancomycin, tazobactam, a carbapenem, or ceftriaxone with metronidazole. If the provider suspects gas gangrene or a necrotizing soft tissue infection, penicillin plus clindamycin should be added, which treats group A streptococcal necrotizing fasciitis. Clindamycin should be strongly considered because it inhibits the synthesis of clostridial exotoxins and lessens the systemic effects of these toxins. Because clindamycin is bacteriostatic and not bactericidal, it should be used with a second anti-microbial, such as penicillin.[18][19][20][21][22]

Fasciotomy may be necessary to relieve compartment pressures. As the infection progresses into deep tissue along and under the fascia, compartment pressures increase, perpetuating further tissue ischemia and necrosis. Surgical debridement should focus on removing all the necrotic tissue and foreign bodies, such as soil, debris, and shrapnel. Irrigating the wounds with copious amounts of sterile normal saline is also important. Hyperbaric oxygen therapy should be added to standard antibiotics and surgical debridement therapy to help improve survival.[22][23] It is important to have coordinated care of these critically ill patients with an intensivist, general surgeon, orthopedic surgeon, urologist (in the setting of Fournier's gangrene of the testicles and perineal structures), gynecologist (in the setting of uterine gas gangrene), infectious disease specialist, hematologist/oncologist, gastroenterologist (in the setting of spontaneous gas gangrene), and hyperbaric oxygen therapy specialist. The consultation flow usually starts with an emergency department provider and early disease recognition.[24][25][24]

Early IV antibiotics with early surgical debridement followed by hyperbaric oxygen therapy can salvage patients with an otherwise nearly always fatal disease. Intravenous antibiotics and early surgical debridement of the necrotic tissue reduce the fatality rate to about 30%. Hyperbaric oxygen therapy can reduce this to 5 to 10%. Hyperbaric oxygen therapy helps by halting exotoxin production by the bacteria, helps to improve the bactericidal effect of the antibiotic, treats the tissue ischemia, improves reperfusion injury of the tissue, and promotes the activation and migration of stem cells and polymorphonuclear cells. Additionally, hyperbaric oxygen

induces vasoconstriction, reducing tissue edema while augmenting oxygenation. The oxygen tension of the tissue increases by a factor of 1000, and this increased oxygen in the tissue helps to resolve hypoxia, improve cellular activity, inhibit bacterial growth, and affect cytokinesis, which increases the migration of neutrophils to the injured tissue. Hyperbaric oxygen also increases the production of growth factors such as vascular epidermal growth factor, which induces neovascularization and tissue repair with capillary budding. This is recognized clinically as increased granulation tissue formation and is usually seen after several hyperbaric oxygen treatments.[26][27][28][27]

Hyperbaric oxygen therapy involves placing the patient in a pressurized chamber, which can be mono-place (single patient) or multi-place (multiple patients treated simultaneously). The mono-place chamber can only treat 1 patient at a time, and the attendant is outside the chamber with specialized equipment and pumps to run IVs and even mechanical ventilation equipment through ports in the chamber door or wall. The disadvantage of this setup is that it limits the therapies available in the chamber. If the patient requires direct contact with the attendant, the chamber has to be depressurized, and the patient is taken out of the chamber. The multi-place chamber has the added benefit of being able to treat multiple patients at the same time, and the attendant is in the chamber with the patients, allowing easier access to the patient for ventilator support, IV therapy, placement of a chest tube, or needle decompression of a pneumothorax. The treatment pressure for gas gangrene is 3 atmospheres absolute. The patient has air brakes every half hour to help reduce the risk of oxygen toxicity. These air brakes are usually 5 to 10 minutes in duration. The treatment at pressure is usually about 90 minutes, with 10 minutes for descent and 10 minutes for the ascent.[24][29][30][24]

When treating gas gangrene, the treatments start twice a day for the first 5 to 10 treatments, reducing to once-daily treatments when stabilized. Continuing hyperbaric oxygen therapy beyond the initial stabilization can speed tissue healing and preparation for eventual tissue grafting, often necessary to close the large defects left after surgical debridement of dead tissue. The risk of hyperbaric oxygen therapy includes oxygen toxicity, which can cause seizures, hypoglycemia, especially in insulin-dependent diabetics, and barotrauma, which can affect the ears, lungs, or any gas-filled structures, such as the stomach, and gas embolism. These complications are rare, except for ear barotrauma, which occurs approximately 43% of the time (84% of these are minor injections of the tympanic membrane).

It is crucial to get early surgical consultation without delay in the case of gas gangrene, as this is an immediate emergency. Broad-spectrum antibiotics should be initiated without any delay in getting cultures. Reasonable coverage should include vancomycin, tazobactam, or a carbapenem, or a third-generation cephalosporin (ceftriaxone) with metronidazole. Moreover, in case of any suspicion of gas gangrene or necrotizing fascitis, penicillin plus clindamycin should be added to cover group A streptococcal necrotizing fasciitis. Clindamycin is strongly recommended. Adjunctive measures in the treatment of gas gangrene include hyperbaric oxygen (HBO) therapy. The function of the existing toxin is not affected by hyperbaric oxygen therapy; thus, debridement is of paramount importance. Hemodynamically unstable patients may not be candidates for HBO therapy. Moreover, animal experimental studies in animals failed to document the therapeutic efficacy of HBO.[31][32][23] Providers should consider using negative pressure wound dressing therapy once adequate surgical debridement has resolved ongoing tissue necrosis.

Differential Diagnosis

The differential for gas gangrene includes the following:

- Abdominal abscess
- Abdominal trauma
- Bacteria sepsis
- Elective abortion
- Emphysematous cholecystitis
- Group A streptococcal infections
- Septic shock
- Toxic shock syndrome
- Vibrio infection[33]

Postoperative and Rehabilitation Care

Patients with gas gangrene need daily or repeated surgical debridement until the necrotizing infection is controlled and receive twice-daily hyperbaric oxygen therapy until tissue necrosis stops and signs of tissue recovery with granulation tissue formation occur. The patient also needs ongoing intensive care and may require hemodialysis for renal failure and extracorporeal membrane oxygenation for patients with severe adult respiratory distress syndrome. Once the infection resolves, many patients require further wound care, often with negative pressure wound therapy, advanced tissue regeneration techniques, and plastic surgical therapies such as skin grafting and flap procedures to close the surgical wounds. Many patients with gas gangrene required prolonged ICU stays, followed by long rehab to improve survival and restore function. Many patients require transfer to a long-term care facility for ongoing wound care, sometimes hyperbaric oxygen therapy, and therapeutic rehab programs with physical therapy and occupational therapy.[34][35]

Pearls and Other Issues

To enhance patient survival and reduce morbidity in gas gangrene, this diagnosis should be high on the differential if the patient presents with infection with signs of necrotic tissue, sepsis, or if gas is present in the tissue. It is important to diagnose early, consult surgery for emergent debridement, and transfer patients with gas gangrene to facilities that have the capability of taking care of such ill patients. They require coordinated care between surgery, intensive care, and hyperbaric oxygen/wound care.[36][37]

Enhancing Healthcare Team Outcomes

Best outcomes are achieved with coordinated care between multiple specialties and intensive care in a facility with personnel competent in caring for critically ill patients. Care has to be coordinated between the surgeons doing the debridements, the wound care/hyperbaric oxygen providers, and the intensivist. Photo documentation in the electronic health record helps improve coordination of care. The surgical team can take pictures in the operating room, and the wound care team can also take pictures when doing dressing changes. This helps nurses and other specialties, such as plastic surgery and infectious disease, know the progress and helps guide decisions. Rehabilitation should start as soon as the patient can, reducing the risk of blood clots

and muscle atrophy. The care team effort must be coordinated so that everyone is on the same page regarding expectations of outcomes. There needs to be peer review and evaluation of team performance for the improvement of patient care in a non-hostile and supportive manner so that all team members are able and willing to contribute to improved patient care.[14][38][39]

Review Questions

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Figure

Gas Gangrene of a Diabetic Foot Contributed by H Murphy-Lavoie, MD



Figure

Gas Gangrene. Plain x-ray showing gas in the tissue planes in a patient with gas gangrene. شهاب, Public Domain, via Wikimedia Commons

References

1. Takehara M. [Host Defense against Bacterial Infection and Bacterial Toxin-induced Impairment of Innate Immunity]. Yakugaku Zasshi. 2018;138(10):1249-1253. [PubMed: 30270267]
2. Stevens DL, Aldape MJ, Bryant AE. Life-threatening clostridial infections. Anaerobe. 2012 Apr;18(2):254-9. [PubMed: 22120198]
3. Huang YY, Lin CW, Yang HM, Hung SY, Chen IW. Survival and associated risk factors in patients with diabetes and amputations caused by infectious foot gangrene. J Foot Ankle Res. 2018;11:1. [PMC free article: PMC5755273] [PubMed: 29312468]
4. Perkins TA, Bieniek JM, Sumfest JM. Solitary Candida albicans Infection Causing Fournier Gangrene and Review of Fungal Etiologies. Rev Urol. 2014;16(2):95-8. [PMC free article: PMC4080857] [PubMed: 25009452]
5. Dempsey A. Serious infection associated with induced abortion in the United States. Clin Obstet Gynecol. 2012 Dec;55(4):888-92. [PubMed: 23090457]
6. Stevens DL, Bryant AE. Necrotizing Soft-Tissue Infections. N Engl J Med. 2017 Dec 07;377(23):2253-2265. [PubMed: 29211672]
7. Shindo Y, Dobashi Y, Sakai T, Monma C, Miyatani H, Yoshida Y. Epidemiological and pathobiological profiles of Clostridium perfringens infections: review of consecutive series of 33 cases over a 13-year period. Int J Clin Exp Pathol. 2015;8(1):569-77. [PMC free article: PMC4348875] [PubMed: 25755747]
8. Lehnhardt M, Homann HH, Daigeler A, Hauser J, Palka P, Steinau HU. Major and lethal complications of liposuction: a review of 72 cases in Germany between 1998 and 2002. Plast Reconstr Surg. 2008 Jun;121(6):396e-403e. [PubMed: 18520866]
9. Takazawa K, Otsuka H, Nakagawa Y, Inokuchi S. Clinical Features of Non-clostridial Gas Gangrene and Risk Factors for In-hospital Mortality. Tokai J Exp Clin Med. 2015 Sep

20;40(3):124-9. [PubMed: 26369267]
10. Janik E, Ceremuga M, Saluk-Bijak J, Bijak M. Biological Toxins as the Potential Tools for Bioterrorism. Int J Mol Sci. 2019 Mar 08;20(5) [PMC free article: PMC6429496] [PubMed: 30857127]
11. Srivastava I, Aldape MJ, Bryant AE, Stevens DL. Spontaneous C. septicum gas gangrene: A literature review. Anaerobe. 2017 Dec;48:165-171. [PubMed: 28780428]
12. Crum-Cianflone NF. Infection and musculoskeletal conditions: Infectious myositis. Best Pract Res Clin Rheumatol. 2006 Dec;20(6):1083-97. [PubMed: 17127198]
13. Carter GP, Cheung JK, Larcombe S, Lyras D. Regulation of toxin production in the pathogenic clostridia. Mol Microbiol. 2014 Jan;91(2):221-31. [PubMed: 24563915]
14. Garcia NM, Cai J. Aggressive Soft Tissue Infections. Surg Clin North Am. 2018 Oct;98(5):1097-1108. [PubMed: 30243450]
15. Roberts EJ, Martucci JA, Wu D. The Unusual Presence of Gas From a Puncture Wound: A Case Report. J Foot Ankle Surg. 2018 Jul-Aug;57(4):785-789. [PubMed: 29571810]
16. Cristoferi G, Fabris G, Ronconi AM, Bozza F, Gallassi GC, Bucca D, Caria GM, Duodeci S. [Gas gangrene. Clinical considerations, prognosis and therapeutic prospects in our experience]. J Chir (Paris). 1991 May;128(5):243-6. [PubMed: 1880179]
17. Sarvari KP, Vasas B, Kiss I, Lazar A, Horvath I, Simon M, Peto Z, Urban E. Fatal Clostridium perfringens sepsis due to emphysematous gastritis and literature review. Anaerobe. 2016 Aug;40:31-4. [PubMed: 27036998]
18. Finsterer J, Hess B. Neuromuscular and central nervous system manifestations of Clostridium perfringens infections. Infection. 2007 Dec;35(6):396-405. [PubMed: 18034207]
19. Nichols RL, Smith JW. Anaerobes from a surgical perspective. Clin Infect Dis. 1994 May;18 Suppl 4:S280-6. [PubMed: 8086576]
20. Shin SH, Park IK, Kang JW, Lee YS, Chung YG. Vacuum-Assisted Closure (VAC) Using Multiple Foam Pieces for Hidden Space Drainage through Less Exposure in Musculoskeletal Infections. J Hand Surg Asian Pac Vol. 2018 Sep;23(3):369-376. [PubMed: 30282543]
21. Yang Z, Hu J, Qu Y, Sun F, Leng X, Li H, Zhan S. Interventions for treating gas gangrene. Cochrane Database Syst Rev. 2015 Dec 03;2015(12):CD010577. [PMC free article: PMC8652263] [PubMed: 26631369]
22. Devaney B, Frawley G, Frawley L, Pilcher DV. Necrotising soft tissue infections: the effect of hyperbaric oxygen on mortality. Anaesth Intensive Care. 2015 Nov;43(6):685-92. [PubMed: 26603791]
23. Bakker DJ. Clostridial myonecrosis (gas gangrene). Undersea Hyperb Med. 2012 May-Jun;39(3):731-7. [PubMed: 22670554]
24. Mathieu D, Marroni A, Kot J. Tenth European Consensus Conference on Hyperbaric Medicine: recommendations for accepted and non-accepted clinical indications and practice of hyperbaric oxygen treatment. Diving Hyperb Med. 2017 Mar;47(1):24-32. [PMC free article: PMC6147240] [PubMed: 28357821]
25. Gacto-Sanchez P. Surgical treatment and management of the severely burn patient: Review and update. Med Intensiva. 2017 Aug-Sep;41(6):356-364. [PubMed: 28456441]
26. Pruskowski KA. Pharmacokinetics and Pharmacodynamics of Antimicrobial Agents in Burn Patients. Surg Infect (Larchmt). 2021 Feb;22(1):77-82. [PubMed: 33164665]
27. Ramos G, Cornistein W, Cerino GT, Nacif G. Systemic antimicrobial prophylaxis in burn patients: systematic review. J Hosp Infect. 2017 Oct;97(2):105-114. [PubMed: 28629932]
28.

Barone M, Grani G, Ramundo V, Garritano T, Durante C, Falcone R. Fournier's gangrene during lenvatinib treatment: A case report. Mol Clin Oncol. 2020 Jun;12(6):588-591. [PMC free article: PMC7179381] [PubMed: 32337042]

29. Chantre C, Foucher S, Le Hot H, Lefort H, Blatteau JÉ. [Hyperbaric oxygen therapy, a little-known discipline]. Rev Infirm. 2018 Jun-Jul;67(242):14-15. [PubMed: 29907169]
30. Burman F. Low-pressure fabric hyperbaric chambers. S Afr Med J. 2019 Mar 29;109(4):12574. [PubMed: 31084683]
31. Heyboer M. Hyperbaric Oxygen Therapy Side Effects - Where Do We Stand? J Am Coll Clin Wound Spec. 2016;8(1-3):2-3. [PMC free article: PMC6161636] [PubMed: 30276115]
32. Clark LA, Moon RE. Hyperbaric oxygen in the treatment of life-threatening soft-tissue infections. Respir Care Clin N Am. 1999 Jun;5(2):203-19. [PubMed: 10333449]
33. Chernyadyev SA, Ufimtseva MA, Vishnevskaya IF, Bochkarev YM, Ushakov AA, Beresneva TA, Galimzyanov FV, Khodakov VV. Fournier's Gangrene: Literature Review and Clinical Cases. Urol Int. 2018;101(1):91-97. [PMC free article: PMC6106138] [PubMed: 29949811]
34. Chen SY, Fu JP, Wang CH, Lee TP, Chen SG. Fournier gangrene: a review of 41 patients and strategies for reconstruction. Ann Plast Surg. 2010 Jun;64(6):765-9. [PubMed: 20407363]
35. McGinness K, Kurtz Phelan DH. Use of Viable Cryopreserved Umbilical Tissue for Soft Tissue Defects in Patients With Gas Gangrene: A Case Series. Wounds. 2018 Apr;30(4):90-95. [PubMed: 29718818]
36. Ingraham AM, Jung HS, Liepert AE, Warner-Hillard C, Greenberg CC, Scarborough JE. Effect of transfer status on outcomes for necrotizing soft tissue infections. J Surg Res. 2017 Dec;220:372-378. [PubMed: 29180205]
37. Mills MK, Faraklas I, Davis C, Stoddard GJ, Saffle J. Outcomes from treatment of necrotizing soft-tissue infections: results from the National Surgical Quality Improvement Program database. Am J Surg. 2010 Dec;200(6):790-6; discussion 796-7. [PubMed: 21146022]
38. Roloff D. [Prerequisites for the transfer of patients with gas gangrene to a specialized facility]. Anaesthesiol Reanim. 1991;16(1):49-58. [PubMed: 2043237]
39. Sison-Martinez J, Hendriksen S, Cooper JS. StatPearls [Internet]. StatPearls Publishing; Treasure Island (FL): May 31, 2023. Hyperbaric Treatment of Clostridial Myositis and Myonecrosis. [PubMed: 29763178]

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