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Gas Gangrene: Case Presentation and Literature Data

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Abstract

Although a disease with a particularly historical, war-related implication, gas gangrene still retains its importance in pathology through its highly severe prognosis and rapid death in the absence of immediate treatment. The most common occurrences of the infection in times of peace are traffic accidents and natural disasters, but this can also occur in a non-traumatic context, in carcinomas and digestive tract ulcers or diabetes. The most commonly cited etiological agent is *Clostridium perfringens*, but other anaerobic germs as well as various aero-anaerobic associations may be responsible for the appearance of gangrene. The accumulation of gas bubbles through the action of etiological agents, highlighted by the presence of crevices or radiological examinations, is the hallmark of this condition. In this paper, the authors present the case of a 26-year-old man who died as a result of the gas gangrene with a rapid evolution (60 hours), which occurred in the progress of a soil telluric wound in the right thigh despite doctors' efforts. The authors emphasize the importance of suspicion of the possibility of the occurrence of gangrene in wounds contaminated with vegetal remains or soil, on the one hand because the pathognomonic sign appears at an advanced stage of the infection and on the other hand because the prophylactic, surgical and medicinal treatment together with the resuscitation measures may be life-saving when applied in a timely manner.

Keywords: Wound contamination; Gas gangrene; *Clostridium perfringens*

Introduction

Gas gangrene is a rare infection, its incidence increasing during wars and natural disasters. The term was first used in 1882 by Molier and Ponget, replacing the "malignant edema"-a name that was given to the bacillus identified by Pasteur, Koch and Geffky as a result of the experiments conducted over several years: "bacillus of malignant oedema" [1]. The first cases mentioned in the literature date back to the period 1882-1902, being recorded at a hospital in Bombay, India, a geographical area where favorable conditions existed for the rapid growth of germs-damp heat and contamination [2]. The many cases identified during World War I were an important opportunity to study this pathology in detail [3]. Between January 1915 and October 1916, a French hospital near Paris found 107 cases of gas gangrene among soldiers wounded in the war [4]. Between 1924 and 1934, 17 were hospitalized in a New York hospital [3]. Between 1960 and 1972, there were 72 cases in Australia [5]. In the 1970s, following an airplane crash, eight of the 77 injured cases developed gangrene [5]. Closer to our day, in a 2001-2013 study, Shindo et al. identified 35 cases [6].

In about 80% of cases, the context of the gas gangrene is traumatic. Non-traumatic situations include pathologies that contribute as such by direct damage to the digestive tract, for instance carcinomas [7,8], ulcerations-Crohn's disease [8], or chemotherapy [7]. More notable pathologies are diabetes [7] or neutropenia [8] that predispose to gas gangrene by decreasing body immunity, prolonged treatment with proton pump inhibitors [8] which increases the pH, leading in turn to the intensifying of virulence and survival of gas gangrene-producing bacteria and severe arteriosclerosis affecting local vasculature [9]. Also, colon surgery can contribute to the development of gas gangrene through the risk of intraoperative dissemination.

The short incubation period is followed by the sudden onset of symptomatology, with rapid deterioration. The constant crevices found in the palpation of the skin, due to the accumulation of gas bubbles through the action of etiologic agents, are considered pathognomonic for the gas gangrene [10].

This severe infection immediately requires a complex and aggressive surgical treatment with wide debridements, possibly amputations, associated with antibiotherapy and general re-balancing general therapy [11]. Applying hyperbaric oxygen therapy improves prognosis [12]. Multivalent antigangrenoid serotonergy is efficient only until the toxins are attached to the tissues [11]. Often, despite the sustained treatment, clinical progression is rapid towards gloomy prognosis with increased mortality: 100% in the absence of treatment, 40% in the treated forms and around 75% if the infection exceeds the inguinal arcade [13].

Case Presentation

A 26-year-old man comes to the emergency room with a wound to the right thigh, caused by agricultural machinery, while working the field.

Five hours after the trauma, the patient is registered at the Emergency Triage with a contused wound of the right thigh. Five hours and 50 minutes after the trauma when the victim is examined surgically, it was found: a contused wound in the outer region of the right thigh in 1/3 medium, with ragged edges, containing various vegetal remains, with no bleeding (declarative with moderate bleeding at home); the patient was slightly restless. Local treatment is carried out: removal of plant debris, washing of the wound with hydrogen peroxide and betadine, compresses with hydrogen peroxide to the wound, and dressing; administration of an vial of ATPA was not possible due to lack thereof.

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Five hours and 55 minutes from the trauma, a thigh X-ray is performed that shows no changes and 6 hours and 10 minutes after the trauma the patient is hospitalized in the surgery ward where he is slightly agitated and accusing pain in the right thigh; the wound is washed with antiseptic solution and dressed.

11 hours and 20 minutes after the trauma, the culture of the secretion shows the aerobic germs Streptococcus fecalis (enterococcus) sensitive to Penicillin, Ampicillin, Nitrafuratoin, Vancomycin and Ciprofloxacin.

24 hours after the trauma, the first surgical intervention is performed with excision, debridement, lavage, drainage with 1 tube of polyethylene, suture, and dressing. The postoperative diagnosis was that of a contused wound on the anterior-outer part of the right thigh with the damage of the vast external muscle. In the intensive care unit, the overall condition of the patient is relatively good, draining about 200 ml serum-sanguinous fluids.

31 hours after the trauma, the patient claims to be in pain, which is why he is administred a vial of profen and 50 minutes later, since the pain does not give up, tramadol is administered. After 20 minutes, the pain does not cede and an ice bag is applied locally with a roller bed and one intravenous ketoprofen dose is administered in 0.9% saline.

43 hours and 15 minutes from trauma, the patient's examination reveals edema of the right thigh with crepitation, blood pressure of 90/50 mm Hg, and approximately 250 ml serohematic outwardly on the drainage tube. After 15 minutes the edema increases in size, the patient shows fever, tachycardia, hypotension, cyanosis, marbled tegument, sweaty, cold on the periphery.

44-45 hours after the trauma, a second surgical procedure was carried out, consisting of the incisions of the right thigh, calf and groin, fasciotomy, abundant oxygen and betadine lavage, drainage with mosses. The established diagnosis is that of gas gangrene appeared in the evolution of an infected wound of the thigh and toxico-septic shock. The patient was in a severe general condition, hypotensive, with oro-tracheal intubation and mechanically ventilated. General treatment with Penicillin, Clindamycin and Tavacin is established.

46-49 hours from the trauma, the patient's condition continues to worsen with the installation of psychomotor agitation for which drug sedation is applied; changes in blood count, in hepatic and renal functioning, hyperglycemia, and hematuria also appeared.

53 hours after the trauma, the doctors tried to transfer the patient to a higher echelon hospital that has the hyperbaric oxygen treatment room, but unfortunately it was not functional at that time. In those conditions, one hour later the patient is transferred to another higher echelon hospital in the region. Upon admission, the patient presented Glasgow score 7 points, tachypnea, tachycardia, hypotension, oxygen saturation 66%, and admission to the surgery department was decided.

59 hours from the trauma, a local surgical examination reveals large incisions at the level of the thigh, the groin and the right inguinal region, the presence of gas bubbles at the abdominal, preperitoneal muscular level, the tissues having a devitalized aspect. The patient was in a very severe condition, comatose, intubated oro-tracheal, with facies throbbing, fever, paleness, marbled aspect, dehydrated, tahipneic, tachycardic, hypotensive, filiform peripheral pulse and oligoanuria. Emergency surgery occurs and shortly the patient suffers a cardio-circulatory arrest for which resuscitation and cardioversion maneuvers were instituted. After resuscitation of the sinus rhythm, surgical maneuvers of debridement and excision of necrotic tissues were performed; the presence of subcutaneous gas up to umbilical and preperitoneal space is highlighted. When the presence of intraperitoneal haemorrhagic fluid and gas bubbles were detected, excision maneuvers were stopped. The patient soon suffered two cardio-circulatory stops, followed by death 60 hours after the trauma. The diagnosis of death in the clinic was: right thigh gas gangrene, extended to right calf and anterolateral abdominal wall, hemorrhagic peritonitis with anaerobic germs, preperitoneal anaerobic germ infection, toxic-septic shock, and multi-organ failure.

The autopsy was performed 24 hours after death. The external examination of the corpse revealed the installation of signs of death and icteric staining of the skin and mucous membranes.

Upon examination of the right lower limb, the right thigh and the right calf were increased in volume (diameter 63 cm right thigh, 55 cm left thigh, 39 cm right calf, 36 cm left calf); right thigh with reddish-green skin, multiple vesicles with gas and reddish-green liquid; crevices of the right thigh skin. On the abdomen, the right thigh and the right leg, multiple deep surgical incisions were observed, all the way to the abdominal muscles and to the muscular layer of the thigh and calf, the muscles displaying a “boiled meat” aspect (Figure 1).

In the internal examination, non-specific aspects were found: cerebral and pulmonary stasis and edema, pleural effusion and peritoneal fluid, liver steatosis, superficial gastric mucosal erosions and stomach blood content.

The microscopic examination performed on autopsy specimens revealed: lung with focal leukocyte alveolitis, stasis, interstitial and intraalveolar focal edema; liver with focal microscopic hepatocyte steatosis and sinusoidal congestion, red-brown acellular material in the lumen of the renal collector tubes; thigh skin with acute cellulitis; muscle tissue from the thigh with acute myositis lesions and fibrinous peritonitis.

Based on the macro and microscopic findings, it was concluded that the patient died as a result of the toxic-septic shock, the consequence of the gas gangrene-an infectious complication occurring in the evolution of a right thigh contused wound contaminated with soil and vegetal remains.

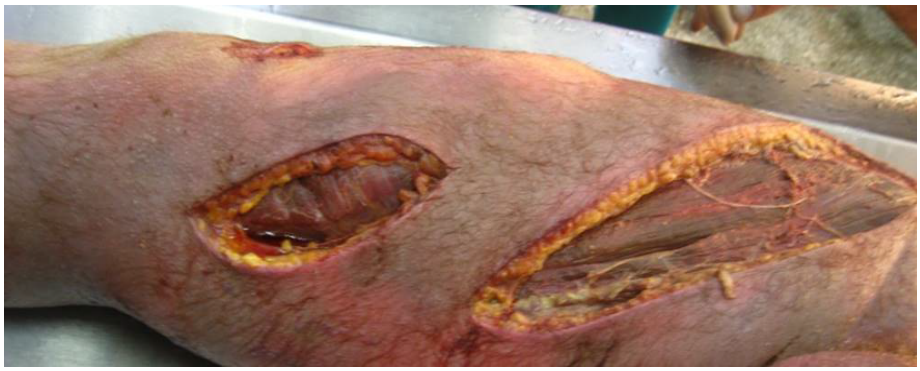

Figure 1: Aspect of the right thigh.

Discussion

Gas gangrene is a diffuse, necrotizing infection of the muscular tissue, characterized by a rapid extension, favored by the presence of devitalized tissues or by generic factors such as immunosuppressive therapy and organic tars.

The incriminated etiological agents are various: anaerobic germs (clostridium-most often, bacteroides, microfili-peptococci, peptostreptococci) or aero-anaerobic associates such as clostridium-staphylococcus streptococci [3]. They enter the body primarily through wounds contaminated with earth or feces. The development of clostridium germs, most commonly an etiological agent, requires anaerobic conditions initially created by traumatic tissue hypoperfusion, with consequent disruption of tissue oxygenation [4] and subsequently by the alpha toxin eliminated by the bacterium itself at entrance gate, where the rapid multiplication of the gas release germs takes place as well. Alpha toxins produce vasoconstriction, followed by platelet aggregation, which results in decreased blood flow and maintenance of the initial ischemic necrotic process in the infected area, with subsequent rapid expansion to the entire body segment [14]. Also, alpha toxins interfere with neutrophil chemotaxis and activate the C proteinkinase and arachidonic acid cascade, thus taking control of cellular metabolism. In addition to alpha-toxins, theta-toxins, which produce cell lysis, are also eliminated. From the entrance gate the toxins are released into circulation and, together with the formed gases, lead to tissue necrosis with extension of the infection until plurivisceral damage, which will eventually lead to the installation of toxic-septic shock [15].

Clinical signs in the gas gangrene occur after an incubation period of 1-2 days after wound production, during which the general condition of the patient is good and the evolution of the wound has no particular elements [16].

The onset is sudden, manifested by pain that exacerbates in the following few hours, becoming atrocious. Initially there is a local palor and then the skin around the wound becomes livid, marbled. The region is diffusely swollen, with the occurrence of blisters and skin necrosis that expands rapidly; the underlying muscles look like boiled meat. From the wound, a fetid, dirty secretion drips out, and in cases where the etiology is given by piogene associations, the secretion becomes purulent. The pathognomonic mark for gas gangrene is represented by gas gulps observed during palpation, generated by the formation of pressure gas bubbles around the wound [17,18].

General clinical examination reveals: rapid general worsening with toxemia, paleness, sharp features, rapid heart rate, fever (38°C), nerve disorders, cardiovascular collapse, and progression to death. Paraclinic the following are observed: hypovolaemia, acidosis, anemia (consequence of hemolysis), coagulation disorders, hepatic and renal failure [18,19].

The clinical diagnosis is based on local and general manifestations: sudden onset, presence of a wound, suggestive local signs, and serious condition with toxemia. Laboratory diagnosis involves making Gram stained smear with germination, and haematological tests reveal leukocytosis with neutrophilia, anemia with anezoinophilia (characteristic of anaerobic infections), elevated urea and glycemia as well as alteration of protein electrophoresis and ionogram. The imaging examination highlights gas bubbles at the subcutaneous level [16,20].

The prophylaxis of gas gangrene involves the identification and correct treatment of telluric wounds. Surgical treatment includes opening sutures-when the traumatic wound was sutured or the source of the infection is surgery in the abdominal perineal sphere; removal of secretions for laboratory examination; extensive debridement; large excisions and conter-incisions for wound ventilation; washing with hydrogen peroxide and betadine, as well as performing "open" amputations. Drug treatment consists of antibiotherapy (penicillin-gentamicin-metronidazole associations) as well as sustained rebalancing-volemic, hydro-electrolytic and protein, blood transfusions, hyperbaric oxygen therapy. Symptomatic treatment is also given in order to relieve pain and hyperthermia with analgesics and antipyretics, vitamins, tranquilizers, etc. The promptness of the diagnosis allows the administration of polyvalent antigangrenoid serum (400,000-600,000 i.u.), which can be effective before fixation of toxins on the tissues [11,12,21].

In the case presented in this paper, the starting point of the gas gangrene was a right thigh wound, produced with agricultural machinery, while the victim was pursuing agricultural activities. He came to the hospital 5 hours after the injury was produced, and the wound was treated from the outset as a lesion with septic potential. Since the appearance of the first signs of gangrene, the medical team made strong efforts to save the patient's life by applying surgical treatment with extensive tissue debridements and complex antibiotic treatment associated with sustained general rebalancing treatment. Limited material resources did not allow the application of hyperbaric oxygen therapy despite the efforts of the medical team. The evolution of the case was rapidly unfavourable, surpassing the inguinal arch, extending to the umbilical level.

Conclusion

Gas gangrene is a pathology rarely encountered in current medical practice. However, due to the sudden onset of rapid progression, gloomy prognosis and increased mortality, it is necessary for emergency and surgical department specialists to keep high suspicion of each contusive wound contaminated with earth and vegetal remains.

Early diagnosis, immediate multivalent antigangrenoid serotherapy, extensive surgical debridement, prompt antibiotic therapy with intensive care monitoring, application of hyperbaric oxygen therapy can lead to survival and rescue of the affected body segment.

Conflict of Interest

Authors have no conflict of interest to disclose.

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