Forensic Sci Med Pathol (2011) 7:278–282
DOI 10.1007/s12024-010-9211-8

CASE REPORT

Necrotizing fasciitis with fatal outcome: a report of two cases

Sarah Heinze · Klaus Püschel · Michael Tsokos

Accepted: 1 December 2010 / Published online: 19 December 2010

Abstract Two cases of sudden and unexpected death due to necrotizing fasciitis are presented with a short overview of this rare disease with special reference to pathological features and causative and epidemiological factors. One case occurred as a complication of liposuction surgery, and the second presented after minor trauma. Based on the autopsy findings and an interdisciplinary approach, medico-legal evaluation provides a substantial basis for later court hearings in such cases.

Keywords Necrotizing fasciitis · *Staphylococcus aureus* · Autopsy · Forensic pathology

Introduction

Necrotizing soft tissue infections cover a broad spectrum of clinical entities, ranging from mild pyodermas to life-threatening necrotizing fasciitis. Even though caused by many different organisms, these infections are most often due to Streptococci, Staphylococci or Clostridiae [1–3], and the prognosis depends on the rapidity of diagnosis and treatment. The term *necrotizing fasciitis* is used to describe the most consistent feature of the infection: necrosis of the fascia and subcutaneous tissue with relative sparing of the underlying muscle [4].

S. Heinze · M. Tsokos (✉)
Institute of Legal Medicine and Forensic Sciences,
Charité—Universitätsmedizin Berlin, Turmstr. 21 (Haus L),
10559 Berlin, Germany
e-mail: mtsokos@web.de

K. Püschel
Institute of Legal Medicine, University Hospital
Hamburg-Eppendorf, Hamburg, Germany

Necrotizing fasciitis is rare with approximately 500 to 1,500 cases reported annually in the United States [5]. However, its incidence seems to be rising [6, 7]. Pathologists and forensic pathologists are aware of necrotizing faciitis and the diagnosis is usually no challenge at autopsy. Nevertheless, necrotizing fasciitis might be recognized too late by clinicians for sufficient treatment, thereby resulting in a fatal outcome. In such instances, the forensic pathologist has to deal with cases of alleged clinical malpractice. The cases presented here were observed in our forensic autopsy practice. The pathogenic correlations and forensic assessments based on the case characteristics are presented, with a brief survey of the literature.

Case reports

Case 1

A 61-year-old woman was admitted to the intensive care unit (ICU) 2 days after abdominal liposuction surgery. Previous laboratory examinations were normal. She denied taking any medications or having any illnesses. On arrival at the ICU, she presented with diarrhea, general weakness, and acute pulmonary failure. The initial tentative diagnosis of pulmonary embolism could not be confirmed. Other findings included fever, low blood pressure, vomiting, and extreme erythema of the abdominal skin. Swabs taken from the wounds revealed *Staphylococcus aureus*. Antibiotic therapy was initiated, and the woman underwent surgical debridements, however, her condition deteriorated and she showed signs of multiple organ failure. The hemodialyzed and ventilated patient developed septic shock with disseminated intravascular coagulation. Laboratory findings revealed persistent elevation of inflammatory parameters

until death, with the clinical diagnosis of sepsis. The body was transferred to the Institute of Legal Medicine where an autopsy was performed. External examination showed swelling of the upper limbs, dry gangrene of the fingertips, and surgical debridement with removal of skin and subcutaneous fat from the entire abdominal wall. The open abdominal wound had greenish yellow fasciae and necrotic edges. Autopsy revealed shock lungs, shock kidneys, pericarditis, splenic infarction, and petechial hemorrhages in the brain. Histology showed marked neutrophil inflammation with numerous gram-positive cocci in the affected abdominal fasciae, but only minor myonecrosis in the underlying muscles. Based on the gross and microscopic pathological findings, the cause of death was multiorgan failure due to necrotizing fasciitis.

Case 2

A 47-year-old athletic man died 5 days after he sustained minor trauma to his left leg during a tennis match whilst on vacation. The same day he consulted a general practitioner (GP) because of increasing pain in the left inguinal region. The treatment consisted of analgesia. On returning home, he consulted a second GP because of increasing extreme pain in his left leg. The diagnosis of a "pulled muscle" was confirmed, and the patient received another intramuscular injection and different oral analgesics. On the same night, he called an ambulance due to the increasing pain and was taken to the emergency room (ER) of the University Hospital. The ER staff diagnosed moderate swelling and local pressure pain in the left thigh. A blood test revealed reduced hemoglobin and hematocrit, and increased fibrinogen, international normalized ratio (INR), total bilirubin, glucose, urea nitrogen, creatinine, aspartate aminotransferase, alanine aminotransferase, and C-reactive protein. Despite these laboratory values, the patient was transferred to a ward with the recommendation for leg elevation with cold compresses. Two hours later, his blood pressure dropped to 90/60 mmHg. Six hours after admission to the ward, the diastolic pressure was not measurable, while the systolic pressure had fallen to 80 mmHg. More analgesics were administered, with no effect. During the following hours, the patient became cold and sweaty and had decreased arterial oxygen saturation. He reported numbness in his left leg and a burning sensation in his right leg. The skin of the lower and upper extremities and abdomen had a mottled dusky blue tinge, and both legs were numb. The patient rapidly developed liver and kidney failure and livid discoloration of the skin of his lower body with bullae formation. The tentative diagnosis of crush kidneys and compartment syndrome led to explorative surgery 18 h after the patient had been transferred to the ward. Surgical incisions to the deep fasciae disclosed massive amounts of exudate. Microbiological screening revealed large masses of gram-positive cocci, and pathological examination of a muscle sample yielded the diagnosis of necrotizing fasciitis. Death occurred in the operating room with the clinical cause given as "heart failure".

Significant gross findings at autopsy included extensive soft tissue necrosis radiating from the left thigh with extensive involvement of the subcutaneous tissue of the left flank, the left buttock, back, side, and the lower abdomen (Fig. 1). There were also signs of sepsis with buffy coat clots in all heart chambers and adjacent large vessels, shock lungs and shock kidneys. Histology showed the typical features of necrotizing fasciitis with abundant neutrophil granulocytes (Fig. 2) and gram-positive cocci infiltrating the affected fasciae. Postmortem microbiological examination revealed group A streptococci. The cause

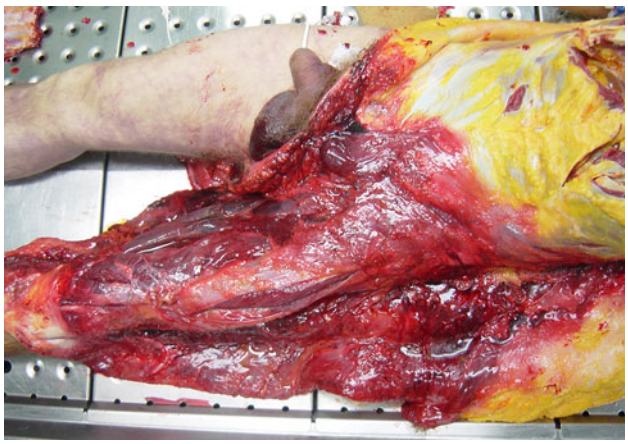

Fig. 1 Gross autopsy findings in necrotizing fasciitis: extensive soft tissue necrosis

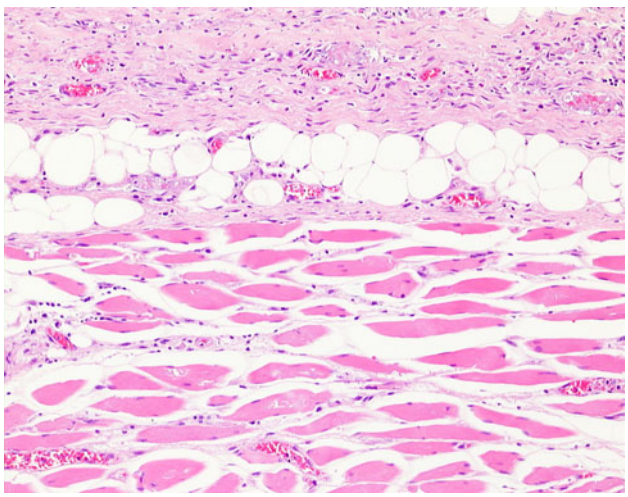

Fig. 2 Histological appearance of necrotizing fasciitis: infiltration of affected fasciae with neutrophil granulocytes (original magnification ×100; H&E)

of death was septic multiorgan failure due to necrotizing fasciitis. Surgical expert advice questioning malpractice found mistakes in the clinical management.

Discussion

A variety of terms have been used to designate the disease named *necrotizing fasciitis* by Wilson in 1952 [4]: hemolytic streptococcal gangrene, Fournier's gangrene, phagedena, phagedena gangrenosum, hospital gangrene, and progressive bacterial synergistic gangrene. The literature describes two groups of necrotizing fasciitis based on microbiological cultures. Two types of the disease can be differentiated: Type I involves synergistic polymicrobial infections usually caused by non-group A streptococci and aerobic or anaerobic organisms. Type II involves *Streptococcus pyogenes* alone or with staphylococci [8].

So far, the etiology of necrotizing fasciitis is not fully understood. Patients often have a prior history of trauma or abrasion. Predisposing risk factors for group A streptococcal necrotizing fasciitis include alcoholism, diabetes mellitus, male sex, and recent trauma (Table 1). The initial encounter of group A streptococci with the host takes place at the epithelial surfaces in the pharynx, tonsils or skin [9].

When affected by necrotizing fasciitis, patients usually suffer from severe pain. This may not be accompanied by erythema or edema at the affected site, which could lead to an initial incorrect diagnosis (e.g. cellulitis, erysipelis) due to subtle physical findings. A characteristic dusky, cyanotic, boggy plaque develops, followed by small serous-filled bullae, tissue necrosis, and extension of the condition. The presence of these bullae is an important diagnostic clue and should raise the suspicion of necrotizing fasciitis [5]. Bullae formed during the early phase of the disease in our second case. This phenomenon was described by Weiss and Laverdiere in 1997 [10]. Gas on plain radiographs is a more inconsistent sign [10, 11].

Characteristic surgical findings include the presence of foul-smelling exudate and a grey necrotic fascia [6] (Table 2).

Table 1 Risk factors for group A streptococcal necrotizing fasciitis [from Refs. 4, 14–19]

Advanced age
Alcoholism
Burns
Malignancy
Chronic illness, e.g. chronic cardiac disease, peripheral vascular disease with limb ischemia, pulmonary disease, asthma, renal failure, chronic liver disease
Chronic skin conditions, e.g. psoriasis, eczema, cutaneous ulcers
Cutaneous injury
Diabetes mellitus
Immunosuppression, e.g. corticosteroid use, solid organ transplant recipient, human immunodeficiency virus infection, asplenia, collagen vascular disease
Intravenous drug abuse
Male sex
NSAID usage
Recent surgery or childbirth
Reduced anti-M1 antibodies and antistreptococcal superantigen neutralizing antibodies (both antistreptococcal antibodies)
Spider bite, snake bite
Trauma
Varicella zoster infection

Table 2 Physical, surgical, and histopathological findings in necrotizing fasciitis

Physical findings in necrotizing fasciitis
Tenderness
Erythema
Warm skin on palpation
Bullae
Skin induration
Skin fluctuance
Crepitus
Skin necrosis
Sensory and motor deficits
Hypotension
Fever (>38°C)
Tachycardia (>100 bpm)
Surgical findings in necrotizing fasciitis
Grayish necrotic fascia
Lack of resistance of normally adherent muscular fascia to blunt dissection
Absence of bleeding during surgical dissection
Presence of foul-smelling exudate
Histopathologic findings in necrotizing fasciitis
Early lesions
Non-specific superficial epidermal hyaline necrosis
Focal edema and hemorrhage in the reticular dermis, subcutaneous fat, and superficial fascia
Few or no inflammatory cells or bacteria
Absent or subtle deep tissue necrosis
Advanced lesions
Diffuse necrosis at all tissue levels
Necrotic subepidermal blisters
Eccrine necrosis
Noninflammatory intravascular coagulation and thrombosis
Myonecrosis
Diffuse dense neutrophil-predominant inflammation
Gram stain: numerous gram-positive cocci

Histopathological examination of the early developing plaque often reveals non-specific epidermal necrosis, edema, and focal dermal hemorrhage with few inflammatory cells. Established plaque shows diffuse necrosis, thrombosis and suppuration of veins and arteries coursing through the fascia, neutrophilic inflammation, and numerous gram-positive diplococci.

Treatment of necrotizing fasciitis involves debridement of infected tissue, aggressive supportive care, and antimicrobial therapy. Rapid diagnosis and early initiation of aggressive surgical and medical therapies are associated with improved survival [12].

Wong et al. (2003) [5] analyzed twelve factors associated with mortality and found that the following reached statistical significance: advanced age, two or more associated comorbidities, and the delay of more than 24 h from admission to surgery. However, mortality associated with necrotizing fasciitis is still high. It has a reported cumulative mortality rate of 34% (range 6% to 76%) with death due to sepsis, respiratory failure, kidney failure, or multiorgan system failure [11, 13].

An early diagnosis may be difficult to make due to the paucity of cutaneous findings early in the course of the disease. In neither of the two cases presented was necrotizing fasciitis diagnosed at the time of admission. In both cases, the correct diagnosis was only made postmortem by medico-legal autopsy. Regardless of the diagnosis, both patients received surgical treatment within 24 h of admission. At the time of admission, both patients already showed symptoms of necrotizing fasciitis, e.g. severe pain disproportionate to local findings as well as erythema and laboratory values in accordance with necrotizing fasciitis. Risk factors predisposing to necrotizing fasciitis as described by Wong et al. (2003) were not apparent in our cases.

The use of broad-spectrum antibiotics prior to admission may modify the clinical picture at the time of presentation and temporarily mask the severity of the underlying infectious process.

The forensic pathologist may occasionally be confronted with patients who have died from necrotizing fasciitis. In the morgue, bodies with signs and symptoms of systemic toxicity associated with soft tissue infections should certainly raise the suspicion of necrotizing fasciitis. Tissue specimens for cell cultures and histology are crucial and should be performed in all suspicious autopsy cases without exception.

Early diagnosis, adequate antibiotic therapy, and aggressive surgical resection are crucial for patient survival in cases of necrotizing fasciitis. Knowledge of the medical history, disease progression and treatment is therefore essential for the medico-legal expertise. The medical records should provide insight into these facts.

Expert opinion has to include knowledge of both forensic pathology and surgery. Such an interdisciplinary approach should be mandatory. In light of the exact time of onset of symptoms and the time course of the following diagnostic procedures and therapy, we suggest that division of expertise into different sections might be helpful in cases of assumed malpractice, e.g. (1) signs, symptoms and cause of death, (2) diagnosis, and (3) therapy. An appraisal of the different sections sheds light on the recognition of the disease and on the appropriate action of the medical personnel in the questioned case.

In the cases presented here, surgical medical experts stated that neither the ambulant doctors nor the hospital medical staff could have been able to diagnose necrotizing fasciitis at presentation. In the second case, the surgical opinion was that even if the diagnosis of necrotizing fasciitis was established earlier, there would have been no difference in fatal outcome. In both cases, the legal charges were withdrawn in view of the surgical opinions.

Key points

1. Necrotizing fasciitis is most often caused by Streptococci, Staphylococci or Clostridiae and the typical pathological feature is necrosis of the fascia and subcutaneous tissue with relative sparing of the underlying muscle. The histopathological picture consists of epidermal necrosis, edema, and focal dermal hemorrhage with few inflammatory cells.
2. Necrotizing fasciitis might be recognized too late by clinicians for sufficient therapy and fatal outcome may be the subject of subsequent medico-legal investigations.
3. The etiology of necrotizing fasciitis is not completely understood. Patients often have some prior history of trauma or abrasion and predisposing risk factors include alcoholism, diabetes mellitus, male sex, and recent trauma.
4. Expert opinion should include signs and symptoms of the patient, correlated with the exact time of onset of diagnostic procedures and therapy. A division of the expertise into different sections such as (a) signs, symptoms and cause of death, (b) diagnosis, and (c) therapy, may be helpful in cases of assumed malpractice.

References

1. Dahl PR, Perniciaro C, Holmkvist KA, O'Connor MI, Gibson LE. Fulminant group A streptococcal necrotizing fasciitis: clinical and pathological findings in 7 patients. J Am Acad Dermatol. 2002;47:489–92.

2. Orlando A, Marrone C, Nicoli N, Tamburello G, Rizzo A, Pagliaro L, Cottone M, D'Amico G. Fatal necrotising fasciitis associated with intramuscular injection of nonsteroidal antiinflammatory drugs after uncomplicated endoscopic polypectomy. J Infect. 2007;54:145–8.
3. Tsokos M, Schalinski S, Paulsen F, Sperhake JP, Püschel K, Sobottka I. Pathology of fatal traumatic and nontraumatic clostridial gas gangrene: a histopathological, immunohistochemical, and ultrastructural study of six autopsy cases. Int J Legal Med. 2008;122:35–41.
4. Wilson B. Necrotising fasciitis. Am Surg. 1952;18:416–31.
5. Wong CH, Chang HC, Pasupathy S, Khin LW, Tan JL, Low CO. Necrotizing fasciitis: clinical presentation, microbiology, and determinants of mortality. J Bone Joint Surg Am. 2003;85: 1454–60.
6. Bakleh M, Wold LE, Mandrekar JN, Harmsen WS, Dimashkieh HH, Baddour LM. Correlation of histopathologic findings with clinical outcome in necrotizing fasciitis. Clin Inefct Dis. 2005; 40:410–4.
7. Stevens DL, Tanner MH, Winship J, et al. Severe group A streptococcal infections associated with a toxic shock-like syndrome and scarlet fever toxin A. N Engl J Med. 1989;321:1–7.
8. Elliott D, Kufera JA, Myers RA. The microbiology of necrotizing soft tissue infections. Am J Surg. 2000;179:151–8.
9. Chatwal GS, McMillan DJ. Uncovering the mysteries of invasive streptococcal diseases. Trends Mol Med. 2005;11:152–5.
10. Weiss KA, Laverdiere M. Group A streptococus invasive infections: a review. Can J Surg. 1997;40:18–25.
11. Fugitt JB, Puckett ML, Quigley MM, Kerr SM. Necrotizing fasciitis. Radiographics. 2004;24:1472–6.
12. Stamenkovic I, Lew PD. Early recognition of potentially fatal necrotizing fasciitis. N Engl J Med. 1984;310:1689–93.
13. McHenry CR, Piotrowski JJ, Petrenic D, Malangoni MA. Determinants of mortality for necrotizing soft-tissue infections. Ann Surg. 1995;221:558–63.
14. Aronoff DM, Bloch KC. Assessing the relationship between the use of nonsteroidal antiinflammatory drugs and necrotizing fasciitis caused by group A streptococcus. Medicine. 2003;82:225–35.
15. Basma H, Norrby-Teglund A, Guedez Y, et al. Risk factors in the pathogenesis of invasive group A streptococcal infections: Role of protective humoral immunity. Infect Immun. 1999;67:1871–7.
16. Kaul R, McGeer A, Low DE, Green K, Schwartz B. Population-based surveillance for group A streptococcal necrotizing fasciitis: Clinical features, prognostic indicators, and microbiologic analysis of seventy-seven cases. Ontario group A streptococcal study. Am J Med. 1997;103:18–24.
17. Majeski J. Necrotizing fasciitis developing from a brown recluse spider bite. Am Surg. 2001;67:188–90.
18. Simonarth T, Simonarth JM, Derdelinckx I, et al. Value of standard laboratory tests for the early recognition of group A beta-hemolytic streptococcal necrotizing fasciitis. Clin Infect Dis. 2001;32:E9–12.
19. Singh G, Sinha SK, Adhikary S, Babu KS, Ray P, Khanna SK. Necrotising infections of soft tissues—a clinical profile. Eur J Surg. 2002;168:366–71.