CASE REPORT

Necrotising fasciitis due to an infected sebaceous cyst

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SUMMARY

The current case presents a patient who was admitted to our hospital with the diagnosis of cellulitis of the right groin. In the following days, the patient's condition deteriorated and developed a septic shock. Exploration in the operating room showed a necrotising fasciitis of the adductor muscles, with an infected sebaceous cyst in the inguinal crease as port d'entrée. After extensive surgical debridement, antibiotic therapy, haemodynamic and respiratory support, the patient recovered. Necrotising fasciitis is a rare but very lethal condition, which necessitates aggressive surgical therapy and antibiotic support. The current case report is the first report to show a necrotising fasciitis due to an infected sebaceous cyst.

BACKGROUND

Necrotising fasciitis is a serious condition, which may prove to be lethal if not treated promptly and aggressively.^{1–3} This condition develops in the presence of gas-forming bacteria in the subcutaneous tissues, often due to blunt trauma or due to abrasion of the skin secondary to a cut, a bite, laceration, injection or a surgical incision.^{1–3} In the current case, the fasciitis developed due to an infected sebaceous cyst, an origin which has not been described in the literature.

An infected sebaceous cyst is often underestimated by clinicians as a minor problem. The current case underlines the potential devastating effect it might have.

CASE PRESENTATION

A 65-year-old woman was admitted to the emergency department with a painful right groin since 2 days. Her history consisted of an appendectomy, a laparoscopic sterilisation, bilateral total hip prostheses, adipositas, myocardial infarction, atrial fibrillation and polyneuropathy of the feet. The patient had a temperature of 38.4°C and was haemodynamically stable. Physical examination showed a 10×10 cm erythaema with oedema in the right groin with tenderness but no crepitations. In the right inguinal crease a 15 mm hard tumour was palpable, without signs of a port d'entrée.

Laboratory results showed a leucocytosis of $17.8 \times 10^9/L$, a C reactive protein of 212 mg/L and a BSE of 23 mm/h. An ultrasound was made and showed infiltrated subcutaneous fat without an abscess. The patient was diagnosed with cellulitis of the right groin and was admitted to the hospital for intravenous antibiotics (*flucloxacillin* 6 g24 h).

In the following 48 h, the patient's condition deteriorated, despite the intravenous antibiotics. The erythaema progressed to an area of approximately 40×40 cm and started to show blisters (figure 1). Furthermore, the patient became haemodynamically unstable (RR 85/45 mm Hg, p 110/min, diuresis 20 cc/h). Laboratory results showed an increase of the C reactive protein to 300 mg/L and a decrease of glomerular filtration rate from 65 to 35 mL/min/1.73 m². The Laboratory Risk Indicator for Necrotising Fasciitis Score System (LRINEC-score) was 9, making it a high risk for necrotising fasciitis.^{1–4} A CT scan was made, which showed free air over the fascia of the adductor muscles (figure 2A, B), which led to the diagnosis of a necrotising fasciitis.

TREATMENT

Emergency surgery was performed in which the affected skin, subcutaneous fat tissue and fascia was resected. There was extensive fat necrosis subcutaneous with 'dish-water' foul discharge. The fascia was clearly infected and had a typical golden/grey/brown aspect (figure 3). Exploration of the inguinal crease showed a sebaceous cyst with a small port d'entrée, the source of the infection (figure 4). All the infected subcutaneous tissue and the infected fascia were removed and the patient was transported to the intensive care unit for haemodynamic and respiratory support.

The next hours and following days, there was no progression of infection and no further debridement was necessary. The patient remained haemodynamically unstable and respiratory instability was noticed during the first days after the operation, needing inotropic support and mechanical ventilation. Antibiotic treatment continued with meropenem and clindamycin and was changed later to cefuroxime and metronidazole based on the available cultures. After 6 days the patient stabilised and it was possible to extubate the patient.

OUTCOME AND FOLLOW-UP

The specimens excised during the operation showed extensive necrosis of the fascia with polymorphonuclear infiltration, underlining the diagnosis of necrotising fasciitis. The clinical diagnosis of the sebaceous cyst was confirmed histopathologically. The cultures from the wound showed several different bacteria: *Stenotrophomonas maltophilia*, *Enterococcus faecium* and a *Staphylococcus haemolyticus*.

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Figure 1 The patient's right groin with the fistula of the infected sebaceous cyst (white circle) surrounded by redness and oedema.

After 3 days, the wound was treated with negative pressure system (V.A.C. VeraFlo Negative Pressure Wound Therapy System; KCI Medical; San Antonio, Texas, USA), which also allowed irrigation with an antiseptic solution (Prontosan Wound irrigation solution; B. Braun Medical AG; Sempach;

Figure 2 CT scan of the infected area. The red circle in (A) showing the sebaceous cyst, while the red circle in (B), more dorsocaudal in the upper leg, showing the presence of subcutaneous air over the fascia of the adductor muscles.

Figure 3 The fascia of the adductor muscles was clearly infected: the fascia had a grey brown aspect and was surrounded by foul smelling ('dish-water') fluid.

Switzerland). The negative pressure and irrigation therapy was continued for 18 days and the wound started to show granulation (figure 5A–C).

The plastic surgeon was consulted for the closure of the remaining defect. It measured 3.6 × 12.6 inches (9 × 32 cm; figure 5D). Different options such as split skin grafting, rhomboid flaps or a pedicled anterolateral thigh flap are examples of ways to close such a defect. In this situation a keystone flap seemed the best option as it closes the defect with similar sensate tissues with minimal donor site morbidity. After thorough debridement of the wound and a release of the scar tissue in the wound edges, a keystone flap, as described by Behan,⁵ was advanced into the defect. Closure was performed over a suction drain using vicryl 2.0 and staples (figure 5E). Five days after the keystone flap reconstruction, the patient was

Figure 4 Specimen of the sebaceous cyst found with the fistula shown in figure 1.

Figure 5 (A) Three days after initial debridement. (B) Seven days after debridement and after 4 days of treatment with negative pressure system and antiseptic irrigation. (C) Fourteen days after debridement and after 11 days of treatment with negative pressure system and antiseptic irrigation (dorsal side wound). (D) The residual defect measured 3.6×12.6 inches (9×32 cm) and was closed using a keystone flap reconstruction. (E) Closure was performed over a suction drain using vicryl 2.0 and staples. (F) Seven weeks after initial debridement and 3 weeks after wound closure by the plastic surgeon using a flap reconstruction.

discharged. A small wound dehiscence occurred, probably due to the tension caused by the patient's early mobilisation. This was closed in the outpatient clinic with VAC treatment (figure 5F).

DISCUSSION

Necrotising fasciitis is a serious condition, which may prove to be lethal if not treated promptly and aggressively.^{1–3 6} The condition was already described in 500 BC by the father of modern medicine, Hippocrates: "... the erysipelas all over the body, caused by a trivial accident or a very small wound ... the erysipelas would quickly spread widely in all directions. Flesh, sinews and bones fell away in large quantities ... There were many deaths..."^{6 7} In 1952, Wilson⁷ gave the disease the current well-chosen name of 'necrotising fasciitis', as the infection spreads horizontally over the fascia's of the underlying muscles.¹ It remains a relative rare condition, and especially patients with predisposing diseases such as diabetes mellitus, immunosuppression, end-stage renal failure, liver cirrhosis and malignancy or intravenous drug usage are at risk.^{1 8–18} The incidence is thought to be 0.40 case per 100 000 population.^{1 16} The mortality of 25–30% is extremely high and has been unchanged in the past 60 years.^{1 2}

This condition is due to the presence of gas-forming bacteria in the subcutaneous tissues, often due to blunt trauma or due to abrasion of the skin. This abrasion of the skin is often due to trauma,⁸ a cut,^{19 20} a bite, laceration,¹⁰ injection or a surgical incision.^{1 21 22} Often, a very ordinary accident may lead to the

lethal condition: Dias *et al*¹⁹ and Kushawaha *et al*²⁰ described near fatal cases of necrotising fasciitis after a finger while peeling food. Clinicians should be weary of a potential fasciitis, when a patient becomes septic early after recent surgery. Raghavendra *et al*²² showed a case in which an otherwise healthy 19-year-old man developed a rapidly spreading necrotising fasciitis after a minimal invasive laparoscopic appendectomy. Subramaniam *et al*²¹ described a case of Fournier's gangrene, after rubber band ligation of haemorrhoids, leading to extensive debridement and formation of a defunctioning loop sigmoid colostomy.

In the current case, the skin defect was due to an infected sebaceous cyst, an origin which had not been described in the current literature. Sharma *et al*²³ have described a case in which an infected sebaceous cyst presented itself as a necrotising ulcerative wound, according to the authors, mimicking a necrotising fasciitis. However, contrary to the current case, there were no systemic symptoms or a corroded fascia described. Although an infected sebaceous cyst is often underestimated by clinicians as a minor problem, the current case underlines the potential devastating effect it may have.

Necrotising fasciitis is not necessarily caused by a disruption of the skin. It can also be caused by a perforation of the gastrointestinal tract. Haemers *et al*¹⁸ described recently a patient with an extensive necrotising fasciitis of the pelvic area after perforation of a rectal tumour. Sometimes there is no clear port d'entrée and the origin may remain unclear. Chunduri *et al*²⁴ described a case of necrotising fasciitis probably due to an

infection of an odontogenic origin, leading to an extensive necrotising fasciitis of the head and neck region. Sharma *et al*¹² showed a case in which an 82-year-old man who developed a fatal necrotising fasciitis without a history of trauma, insect bites or abrasions. In another case, by Leaning *et al*, a 60-year-old woman receiving chemotherapy developed extreme bruising of the left thigh and buttocks, spontaneously without a history of trauma. Surgical exploration showed a necrotising fasciitis, after which a palliative regime was started as the patient was unfit for extensive debridement.¹¹

On the basis of microbiology, there are two main types of necrotising fasciitis.^{1 2 25} Type I is a polymicrobial infection with at least one anaerobic species, often members of Enterobacteriaceae. Location of the infection is often the perianal and groin area, the trunk, the abdominal wall and surgical wounds. They often take place with immunocompromised patients. The majority of necrotising fasciitis infections are type I (55–75%).¹ With the current case, the infection was also a polymicrobial infection with *Staphylococcus haemolyticus* as the anaerobic species. The *Stenotrophomonas maltophilia* in the culture is a difficult-to-treat germ and has been described as an (uncommon) originator of fasciitis.²⁶

Type II is a monomicrobial infection, with an invasive group A β -haemolytic *Streptococcus* or sometimes another *Streptococcus* or *Staphylococcus*. Predominant infection sites are head, neck and the extremities. Type II can take place in otherwise healthy, young, immunocompetent patients.² It can quickly progress (24–48 h) and takes place in 30% of the cases in combination with a streptococcal toxic shock syndrome.^{1 2}

Some state a third category, in the form of infections of marine *Vibrios*.^{1 2} Kushawaha *et al*²⁰ described a case in which an 81-year-old patient with a history of end-stage renal disease developed an infection by a *Vibrio vulnificus*, after a puncture injury due to cleaning crabs. Several surgical debridements were necessary, but the physicians were able to preserve the affected limb.

The diagnosis of necrotising fasciitis is above all a clinical diagnosis.²⁷ Important symptoms are painful erythema, (extreme) pain beyond borders of erythema, swelling, fever and sometimes skin anaesthesia, bullae and crepitus.^{1 2 27} The latter two are often thought to be a *conditio sine qua non* for necrotising fasciitis; however, they are only present in 13–31% of the cases.² Early recognition is essential: Vijayan *et al*¹⁰ recently described a lethal case, where an infected laceration with a necrotising fasciitis in a septic and unresponsive 91-year-old man was misdiagnosed for full-thickness burn, eventually leading to delay of treatment.

To assist the clinical diagnosis, Wong *et al*⁴ introduced the LRINEC, a score in which the variables total white cell count, haemoglobin, sodium, glucose, serum creatinine and C reactive protein are used to predict the probability of a necrotising fasciitis. A score of lower than 5 holds a low risk, score of 6–7 an intermediate risk, score of 8 or higher holds a high risk on necrotising fasciitis.

When in doubt, imaging modalities can be used. The MRI is the recommended modality to see if there is oedema of the fascia or other soft tissue fluid^{1–3}; however, in our case that was not possible due to the bilateral total hip prostheses in the affected area. Therefore, we choose to use a CT scan to see if there were subcutaneous air collections over the fascia. Another diagnostic modality is to perform a frozen section biopsy under local anaesthesia.²⁸ However, it should be noted very clearly that the use of diagnostic modalities should not lead to delay in operative intervention. Several studies have shown that delay in intervention leads to an increase in mortality.^{12 29–31}

Treatment of necrotising fasciitis consists most importantly of aggressive surgical debridement.^{1–3 7–10 13–22 24–27 29 30} If treatment consists only of antibiotic therapy and support, the mortality is believed to be 100%.^{1 11 12} All necrotic tissues should be removed, to reduce bacterial load and expose the anaerobic bacteria to oxygen. During the debridement, the wound should be bluntly probed in all directions to find additional pockets of infection.¹ The overlying skin should also be resected, even if appears normal: Andreassen *et al*²⁷ showed that resected overlying skin, which macroscopically appeared normal, was microscopically affected with extensive early vascular thrombosis and vasculitis.³ The first debridement should be extensive, as initial incomplete debridement increases the mortality significantly.² After initial debridement, the wounds should be dressed and patient should go to an intensive care unit for support and antibiotic therapy. Wounds should be inspected frequently and if necessary additional debridements must be performed.^{1–3 27} When the patient has stabilised and is free of infection, the residual wound can be treated with skin grafts or surgical flaps.^{1–3 5 27}

Learning points

- ▶ An infected sebaceous cyst may form a port entrée for bacteria and may lead to necrotising fasciitis.
- ▶ If a cellulitis or an other skin infection does not respond to antibiotic therapy, be cautious of necrotising fasciitis.
- ▶ Necrotising fasciitis is a serious condition, which needs an extensive aggressive surgical debridement.
- ▶ A keystone flap can be an adequate reconstruction option when the residual tissue is free from infection, as it closes the defect with similar sensate tissues with minimal donor site morbidity.

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