CASE REPORT

Necrotising fasciitis

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SUMMARY

A patient in his late 50s who suffered from necrotising fasciitis is described in this case report. The patient first presented at the chest pain unit, where he reported of respiratory distress and showed systemic signs of an infection. Because of progressive skin lesions indicative of necrotising fasciitis on both the lower extremities, the patient was immediately rushed into surgery and was treated at the intensive care unit. Despite receiving immediate treatment, the patient died of this rapidly spreading infectious disease. Because of the fast advance of this disease, we would like to emphasise the importance of early recognition of symptoms.

BACKGROUND

Necrotising fasciitis (NF) is a rare, but life-threatening infectious disease which is associated with a high mortality rate. Bacteria rapidly spread out along the fascial planes and therefore early recognition of symptoms is crucial. Patients have to be rushed into surgery immediately where a thorough debridement needs to be performed.

We would like to sharpen awareness of cases of NF. Patients frequently first present with systemic signs of infection at the internal medicine department and not the surgery department. It is essential that NF is taken into consideration as a potential issue when examining patients with symptoms as mentioned in our case.

CASE PRESENTATION

A patient in his late 50s presented with respiratory distress and hypotension at the chest pain unit. The patient had been able to walk into the hospital but collapsed shortly after arriving. As the laboratory diagnostic (C reactive protein (CRP) 50 mg/L, metabolic acidosis with a pH of 7.09 and a base excess of -18.7) suggested an acute infection, the patient was admitted to the hospital in search of concurrent infectious site(s).

The patient did not have fever; the white cell count was normal (4.59 /nL), platelets (67/nL) and haemoglobin were low (10.1 g/dL). The electrolytes were within the normal range (sodium 140 mmol/L, calcium 2.1 mmol/L, potassium 4.57 mmol/L) and renal function was impaired (creatinine 2.08 mg/dL, urea 68 mg/dL, glomerular filtration rate 34.1 mL/min).

Of note, the patient was on an immunosuppressive therapy (azathioprine, steroids) because of an interstitial lung disease. Furthermore, the patient suffered from pulmonary hypertension, chronic hepatitis C, hyperparathyroidism, arterial hypertension and a monoclonal gammopathy of undetermined significance.

Three years prior, the patient had suffered from pneumonia with septic shock which led to acute kidney failure.

After admittance to the hospital, a CT scan of the abdomen and thorax was performed, and there was no evidence of an infection. During the course of the day, the patient required increasing amounts of catecholamines and the laboratory diagnostic showed symptoms of disseminated intravascular coagulation (platelets 61/nL, D-dimer 1.92 mg/L, fibrinogen 1.5 g/L, activated partial thromboplastin time 40 s). An empiric antibiotic therapy was started (daptomycin, clindamycin and meropenem). During this short period of time the patient developed skin lesions progressing dorsally on both thighs (figure 1), and therefore colleagues from the dermatology department and the orthopaedic department were consulted.

The patient reported to have had a skin biopsy on the left lower leg just several days prior to his admittance because of a suspicious mole (figure 2), which might have served as an entrance portal for the bacteria. Because the skin lesions (haemorrhagic bullae, skin necrosis; figure 1) indicated necrotising fasciitis, the patient had to be operated on without any delay. The diagnosis was confirmed intraoperatively by necrosis of the fascia and later on by the analysis of tissue samples, which revealed group A streptococcal infection.

TREATMENT

Twelve hours had elapsed since admittance to the hospital. The patient was rushed into surgery and a thorough debridement with complete removal of the necrotic fascia was performed on both legs through incisions medially and laterally as shown on the images (figures 3–6). An amputation was

Figure 1 Rapidly progressive skin lesions had developed on both lower extremities. Skin lesions on the abdomen point towards spreading of the disease to the abdominal fascia.

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To cite: Dapunt U, Klingmann A, Schmidmaier G, et al. *BMJ Case Rep* Published online: [please include Day Month Year] doi:10.1136/bcr-2013-201906

Figure 2 A skin biopsy had been taken on the left lower leg, which might have served as an entrance portal.

not an option anymore because the disease had already spread to the abdominal fascia. A vacuum-assisted-closure (VAC) system was placed on the wounds. A broad spectrum antibiotic treatment was continued (daptomycin, clindamycin and meropenem).

Despite surgical intervention the patient demonstrated progressive signs of septic shock. Surgical debridement was repeated 24 and 48 h after hospital admittance, and during the last surgical intervention, necrotic tissue was no longer detectable.

OUTCOME AND FOLLOW-UP

Despite treatment at the intensive care unit, the patient died of septic shock only 68 h after admittance to the hospital (see rapid progression of the disease, figure 7).

Figure 3 Radical surgical debridement of the entire fascia was performed.

Figure 4 Radical surgical debridement of the entire fascia was performed.

DISCUSSION

NF is an infectious disease, characterised by rapid bacteria spreading along the fascial planes. It is a rare disease with an annual incidence of about 1.5 cases/100 000 population¹; but the mortality is rather high, reaching about 20–30%.^{1–3} In most patients, NF develops in the upper or lower extremities (about 57.8% as reviewed by⁴), but cases of cervical or abdominal NF have also been described.⁵

Several countries have reported an increase of NF in the past 20 years, an alarming trend, especially considering the aforementioned high mortality rate.^{6 7}

Two different types of NF have been described. Type 1 is usually caused by multiple organisms (70–90%, streptococcal species, staphylococcal species, enterococcal species, enterobacteriaceae, bacteroides species) and type 2 which typically involves group A streptococci (10%).^{8–10} Some authors have added a type 3 comprising rare cases including vibrio species or methicillin-resistant *Staphylococcus aureus* as causative agents.²

Even though only about 10% of all cases are caused by group A streptococci, it is the most widely known bacterium associated with this disease and has even been termed the ‘flesh-eating bacterium.’¹¹

The most common group A streptococcal infections are mild conditions such as impetigo or pharyngitis, but in rare cases, severe, life-threatening diseases such as NF or pneumonia occur.¹² Extensive research has been dedicated to the question, how the same pathogen may elicit different diseases ranging from mild infections to highly aggressive forms. One aspect to

Figure 5 Wide incisions on both legs were performed medially and laterally, as well as on the lower abdomen.

consider are bacterial virulence factors, such as the M-protein, by which the bacteria are believed to evade the immune system,¹² another, not yet well analysed inter-bacterial communication, which might result in highly aggressive infection, as it is suggested in the case of type 1 (multispecies) NF.²

Risk factors have been identified especially for type 1 NF and they include diabetes, immunosuppressive therapy, chronic infectious diseases, advanced age and others. For type 2 NF risk factors have not yet been identified, and even young and otherwise healthy patients have been described to develop streptococci-induced NF.^{4 5 13}

Figure 6 Wide incisions on both legs were performed medially and laterally, as well as on the lower abdomen.

Figure 7 Rapid progression of the disease over 68 h.

Any kind of skin lesion, ranging from insect bites to extensive injuries may serve as an entrance portal for the bacteria. In many patients, however, previous skin lesions cannot be detected and haematogenous spreading of bacteria is assumed.⁴ After having gained access to the blood circulation or after a penetrating trauma, the infection usually starts at an area of muscle injury,¹⁴ presumably because streptococci have the ability to bind to areas of muscle injury (ie, muscle strain). Bryant *et al*¹⁵ have reported that the vimentin expression is increased on muscle cells after injury and that streptococci may bind selectively to the cell-surface-associated protein.

In the early stages of the infection, the bacteria spread out along the fascial planes. Patients usually report of severe pain which cannot be explained by a visible injury, but because the skin is still unaffected and accompanying symptoms such as erythema or swelling are non-specific, early stages escape detection¹⁶ and can be misdiagnosed as cellulitis because the symptoms are very similar.¹⁷ Only in the more advanced stages, patients quickly develop blisters, bullae, skin fluctuance and skin induration, followed by formation of haemorrhagic bullae, skin anaesthesia, skin necrosis and crepitation.¹⁶ Patients also quickly develop systemic signs of infection such as hypotension and respiratory distress.

The diagnostic options are very limited and the most important determinant of a patient's survival is early recognition and immediate treatment,⁹ which, however, is hampered by the fact that the early symptoms are rather non-specific and hence difficult to detect. So far, there is no laboratory parameter specific for NF. The so-called Laboratory Risk Indicator for Necrotizing Fasciitis (LRINEC) score has been proposed to categorise the average risk of NF.¹⁸ However, prospective studies on the LRINEC score are not yet available. Furthermore, there are also reports of patients suffering from NF despite a normal LRINEC score.¹⁹ In our case described above, the LRINEC score at admission was 4 (CRP 50 mg/L, white cell count 4.59 /nL, haemoglobin 10.1 g/dL, sodium 140 mmol/L, creatinine 2.08 mg/dL, glucose 90 mg/dL) which is not highly indicative of NF.

Because of the lack of reliable laboratory parameters and specific symptoms, it is essential that patients are presented immediately to an experienced surgeon and rushed into surgery at the mere suspicion of NF. According to the literature, the mortality is about 6% when patients are operated on within 12 h of onset of symptoms, but up to 30% after 24 h.²⁰

It is required that all of the necrotic tissue is removed during the first surgical intervention.^{3 13} As demonstrated in our case above, the fasciectomy needs to be performed extensively (figures 3–6).

Usually more than one extensive surgical debridement is necessary with the use of a VAC system in between, and possibly followed by flap surgery.⁵ Therefore, a plastic surgeon should also be involved early on. In addition, a broad spectrum antibiotic treatment has to be started immediately, which can be changed to a more selective antibiotics when the microbiology results are available. Importantly, antibiotic treatment alone will not suffice and should never replace aggressive, surgical debridement.^{2 9}

Alternative therapeutic approaches have been proposed, including hyperbaric oxygen or intravenous immunoglobulin, but their effect is still controversially discussed.¹³

Learning points

- ▶ Patients frequently first present with systemic signs of an infection at the internal medicine department. Close attention should be paid to painful skin lesions.
- ▶ Patients might report of severe pain which cannot be explained by a visible injury, because the bacteria first spread out along the fascial plains.
- ▶ Any surgical discipline might be confronted with cases of necrotising fasciitis. It is vital to keep this disease in mind and to act quickly.
- ▶ Even though the Laboratory Risk Indicator for Necrotizing Fasciitis (LRINEC) score might be helpful, a negative LRINEC score does not rule out necrotising fasciitis.
- ▶ Treatment of necrotising fasciitis requires a multidisciplinary approach and various specialties should be involved early on.

Contributors All the authors have contributed to the conception and design, drafting of the article or revising it critically for important intellectual content and final approval of the version published.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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