BMJ Best Practice

Necrotising fasciitis

Straight to the point of care

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Summary

Necrotising fasciitis is a life-threatening subcutaneous soft-tissue infection that requires a high index of suspicion for diagnosis.

Always suspect necrotising fasciitis in a patient with a rapidly progressing soft-tissue infection and any of the following: severe pain (disproportionate to the clinical findings) or anaesthesia over the site of infection; oedema and erythema (oedema will typically extend beyond the erythema); systemic signs of infection. However, necrotising fasciitis can be easily missed because the patient may present earlier in the disease process with non-specific signs and symptoms.

If you suspect necrotising fasciitis, immediately refer the patient for urgent surgical debridement; do not wait for the results of investigations before referral. Necrotising fasciitis is a clinical diagnosis. However, investigations can support the diagnosis if this is unclear.

Surgical debridement should be repeated as necessary until the patient has no necrotic tissue remaining.

Adjunctive antibiotic therapy and supportive care is crucial. Start intravenous empirical antibiotics as soon as you have obtained blood cultures. Once culture results are available, tailor the antibiotics to target the causative organism.

Definition

Necrotising fasciitis is a life-threatening subcutaneous soft-tissue infection that progressively extends to the deep soft tissues including muscle fascia and overlying fat, but not into the underlying muscle. The causal organisms may be aerobic, anaerobic, or mixed flora. Type I necrotising fasciitis is a polymicrobial infection with anaerobes such as *Bacteroides* , *Peptostreptococcus* , or *Clostridium* , and facultative anaerobes such as certain Enterobacterales or non-group A streptococcus.[1] [2] [3] [4] [5] Type II necrotising fasciitis is a monomicrobial infection, most commonly with *Streptococcus pyogenes* (group A streptococci) and occasionally *Staphylococcus aureus* .[1] [3] [4] [5]

Other infectious aetiologies may rarely cause a monomicrobial necrotising infection that may be associated with specific exposures or risk factors. These include freshwater exposure associated with *Aeromonas hydrophila* , saltwater exposure or consumption of raw oysters associated with *Vibrio vulnificus* , and recent travel to (or living in) Taiwan, where *Klebsiella pneumoniae* is a common cause of monomicrobial infection.[6] Rarely, necrotising fasciitis can be caused by fungal pathogens.

This topic covers the diagnosis and management of necrotising fasciitis in adults only.

THEORY

Epidemiology

Absolute data for the incidence and prevalence of necrotising fasciitis are lacking. Type I (due to mixed anaerobic-facultative anaerobic infections) is more common than type II necrotising fasciitis.[21][22]

An estimated 500 people present with necrotising fasciitis each year in the UK, with an incidence of 0.4 to 0.53 cases per 100,000 population.[23]

US-based multi-site surveillance data from 2021 showed that necrotising fasciitis complicated about 4.5% of invasive group A streptococcal infections, with approximately 100 cases per year.[24]

The overall prevalence, incidence, and epidemiology remain stable.

Risk factors

Strong

inpatient contact with index case

Patient to patient spread of group A streptococcal infection (a common cause of necrotising fasciitis), with median interval of 4 days to spread from index case to second case.[1] [30]

Varicella zoster infection

Serves as a cutaneous portal of entry for infective organisms.[1] [30]

cutaneous injury, surgery, trauma

Serve as a cutaneous portal of entry for infective organisms.[1] [2] [16]

non-traumatic skin lesions

Chronic or acute skin conditions, for example, eczema, psoriasis, cutaneous ulcers, and burns, may serve as a cutaneous portal of entry for infective organisms[1] [2] [16][30]

intravenous drug use

Intravenous drug use provides a cutaneous portal of entry for infective organisms.[16]

Weak

chronic illness

A generalised immunosuppressed state as a consequence of long-standing disease (alcohol dependence, diabetes mellitus, cardiac or pulmonary disease, peripheral vascular disease, renal failure) may predispose to soft-tissue infections[1] [2] [4] [16][30]

immunosuppression

Immunosuppression due to malignancy and/or chemotherapy or radiotherapy, medications (especially chronic corticosteroid use), or infection (HIV) may predispose to soft-tissue infections.[4] Immunosuppressed status may lead to a delay in diagnosis and surgical management leading to greater risk of death.[31]

non-steroidal anti-inflammatory drugs (NSAIDs)

It has been suggested that use of NSAIDs may mask symptoms of necrotising fasciitis, delaying the diagnosis, and that supression of neutrophils and alterations of cytokine production caused by NSAIDs may impair response to infection and allow progression to severe disease. In an animal model of group A streptococcus soft-tissue infection, ibuprofen worsened disease and increased mortality.[32] However, good evidence for the association of NSAIDs and necrotising fasciitis in humans is not available.

Aetiology

Type I necrotising fasciitis is a polymicrobial infection caused by anaerobes such as *Bacteroides*, *Clostridium*, or *Peptostreptococcus* and facultative anaerobes such as certain Enterobacterales (*Escherichia coli*, *Enterobacter*, *Klebsiella*, *Proteus*) or non-group A streptococcus with or without *Staphylococcus aureus*.[1]

Type II necrotising fasciitis is a monomicrobial infection that is most commonly caused by *Streptococcus pyogenes* (group A streptococci) and occasionally *S aureus*.[16] Panton-Valentine leukocidin (PVL)-positive *S aureus*, and MRSA are also potentially causative organisms.[1] Other infectious aetiologies may rarely cause a monomicrobial necrotising infection associated with specific exposures or risk factors:

- *Aeromonas hydrophila*, associated with freshwater exposure[4] [9] [10]
- *Vibrio vulnificus*, from saltwater exposure or consumption of contaminated raw oysters[4] [9] [10]
- *Klebsiella pneumoniae*, in South East Asian countries, in particular Taiwan[11]
- *Clostridium*, can cause gangrenous necrotising fasciitis – see Gangrene.

Very rarely, necrotising fasciitis is a monomicrobial infection caused by fungal pathogens such as mucormycosis.[5] Mucormycosis has been reported as a cause in immunocompromised and immunocompetent patients.[12] [13] [14] Few cases of candida necrotising fasciitis have been reported following surgery.[25]

Predisposing risk factors may include diabetes mellitus, peripheral vascular disease, immunocompromising conditions, chronic renal or hepatic insufficiency, chickenpox or herpes zoster, intravenous drug use, trauma or surgery, or certain medications (e.g., corticosteroids).[1] [16][26] [27]

Pathophysiology

Bacteria are introduced into the skin and soft tissue from minor trauma, puncture wounds, or surgery. However, in up to 20% of cases no primary site of infection is identified. Infection extends through the fascia but not into the underlying muscle, and tracks along fascial planes extending beyond the area of overlying cellulitis. Systemic signs of necrotising fasciitis, such as fever, tachycardia, and hypotension, are primarily due to the action of bacterial toxins.[28] [29]

Classification

Clinical presentation

Necrotising fasciitis can be classified according to clinical presentation, which is based on clinical signs and symptoms, and their speed of onset.

Fulminant

This is the most severe type of necrotising fasciitis and has a poor prognosis.[7] The patient will have extensive tissue necrosis that progresses over hours and will be systemically unwell with sepsis.[7]

Acute

Symptoms and signs develop over days. Typically associated with an identifiable skin or history of trauma, with pain out of proportion to the clinical findings.[7] The patient may initially be systemically well, but can deteriorate over days to hours.[7]

Insidious

Non-specific or variable symptoms with an insidious onset.[7] Localised pain at the site of the skin lesion may be mild or absent.[7]

Causative organism

Necrotising fasciitis can be classified according to the causative organism, once this is identified from blood or tissue cultures.

Type I

Polymicrobial infection with anaerobes such as *Bacteroides* or *Peptostreptococcus* and facultative anaerobes such as certain Enterobacterales (*Escherichia coli* , *Enterobacter* , *Klebsiella* , *Proteus*) or non-group A streptococcus.[1] [3] [4] [5] It is most commonly seen in older patients and in those with underlying illnesses.[8]

Type II

Monomicrobial infection, most commonly with *Streptococcus pyogenes* (group A streptococci), anaerobic streptococci, or rarely other pathogens including Panton-Valentine leukocidin (PVL)-positive *Staphylococcus aureus* and MRSA.[1] [3] [4] [5]

Other infectious aetiologies may rarely cause a monomicrobial necrotising infection associated with specific exposures or risk factors:

- *Aeromonas hydrophila* : associated with freshwater exposure. Most common in patients with immunosuppression, burns, and trauma in an aquatic setting.[4] [9] [10]
- *Vibrio vulnificus* : from saltwater exposure or consumption of contaminated raw oysters. Predisposing risk factors include hepatic disease, diabetes mellitus, chronic renal insufficiency, and adrenal insufficiency.[4] [9] [10]
- *Klebsiella pneumoniae* : in South East Asian countries, in particular Taiwan.[11]
- *Clostridium* : can cause gangrenous necrotising fasciitis – see Gangrene .

Very rarely, monomicrobial infection is caused by fungal pathogens such as mucormycosis.[5] Mucormycosis has been reported as a cause in immunocompromised and immunocompetent patients.[12] [13] [14]

The classification above is based on the World Society of Emergency Surgery (WSES) global clinical pathways for patients with skin and soft-tissue infections, and on expert opinion.[4] Some references, including other publications from WSES, further sub-classify monomicrobial gram-negative infections including *Aeromonas* and *Vibrio* infections as type III and fungal infections as type IV.[3] [15]

Anatomical location

Fournier's gangrene is a type I necrotising fasciitis of the scrotum or male perineum.[1] [2] [4] [16]

Meleney's synergistic gangrene is gangrene of the tissues of the abdominal wall, with synergistic infection with enterobacteria and *Streptococcus* .[17]

Cervicofacial necrotising fascitis is a rapidly progressing gangrenous infection of the skin, subcutaneous tissue, and fascia of the neck and face.[18]

Further classifications exist and are sometimes used when discussing necrotising fasciitis in the context of surgical site infections or rare organisms.

Case history

Case history #1

A 35-year-old woman is admitted to hospital because of pain and swelling of the right thigh. The patient was well until the morning before admission, when she observed a spot on her right thigh. During the course of the day, the lesion enlarged, with increasing pain, swelling, and erythema, and was accompanied by nausea, vomiting, and delirium. Her temperature is 37.5°C (99.5°F), pulse is 128 bpm, and respirations are 20 breaths/minute. BP is 85/60 mmHg. On physical examination, the patient appears unwell and in pain. A small, indurated area of skin breakdown with surrounding erythema and warmth is present on the right thigh; no fluctuance is detected. She is unable to flex or extend the right hip without severe pain and reports pain on passive extension of the right ankle. Her temperature soon rises to 38.4°C (101°F), and BP drops to 70/40 mmHg. Haematocrit is 42, WBC count 5.9 x 10^9 /L (with 64% neutrophils, 19% band forms), serum creatinine 168 micromol/L (1.9 mg/dL), and serum urea 7.8 millimol/L (22 mg/dL). Contrast-enhanced computed tomography shows a diffuse, non-enhancing, honeycomb pattern within the subcutaneous tissue of the right thigh. Subcutaneous stranding and thickening of the skin are prominent in the posterolateral aspect of the thigh; there is also thickening of the posterolateral deep fascia.

Other presentations

Necrotising fasciitis should be considered in a patient with cellulitis who also has systemic symptoms and signs such as hypotension, tachycardia, tachypnoea, nausea, vomiting, or delirium. The area of cellulitis may be either severely and constantly painful (disproportionately to skin findings) or, conversely, anaesthetic. Examination of the skin overlying the area of cellulitis may reveal underlying induration extending beyond the area of cellulitis, ecchymoses, vesicles, bullae, greyish discoloration, or oedema extending beyond erythema. Crepitus may be noted on examination. Rapid extension of cellulitis despite the use of appropriate antibiotics should also raise suspicion for a necrotising process. About half of

cases occur in the extremities, with the remainder affecting the perineum, trunk, or head and neck.[1][2][3] [4] [5] [16][19] [20]

Atypical presentations include necrotising fasciitis that occurs without an obvious overlying skin lesion (approximately 20% of cases), or that arise from a Bartholin gland or perianal abscess. Fournier's gangrene is a form of type I necrotising fasciitis that occurs in the perineum.[1] [16]

Recommendations

Key Recommendations

Have a high index of suspicion; make the diagnosis at the earliest opportunity and have a low threshold for referral for **immediate surgical debridement**.

- Necrotising fasciitis is a life-threatening and time-critical surgical emergency.[3] [5]
- Discuss the patient early with the critical care team.

Always suspect necrotising fasciitis in a patient with a rapidly progressing soft-tissue infection and any of the following:[3]

- Severe pain (disproportionate to the clinical findings) or anaesthesia in the local area.[1] [2] [3][16] [19] [20][34] [35]
- Oedema that extends beyond the erythema.[3]
- Systemic signs of infection.[1] [3] [16][35] [36] Sepsis and multi-organ failure may be present.[3] [37] Think ' **Could this be sepsis**?' based on acute deterioration in an adult patient in whom there is clinical evidence or strong suspicion of infection.[38][39][40] See Sepsis in adults .

Practical tip

Think '**Could this be sepsis?**' based on acute deterioration in an adult patient in whom there is clinical evidence or strong suspicion of infection.[38][39][40]

- Use a systematic approach, alongside your clinical judgement, for assessment; urgently consult a senior clinical decision-maker (e.g., ST4 level doctor in the UK) if you suspect sepsis.[38] [40] [41] [42]
- Refer to local guidelines for the recommended approach at your institution for assessment and management of the patient with suspected sepsis.

If you suspect necrotising fasciitis, immediately refer the patient to the surgical team; do not wait for the results of investigations before referral.[2] [3] Necrotising fasciitis is a clinical diagnosis. However, investigations can support the diagnosis if this is unclear.[3]

Be aware that the diagnosis can be **easily missed** because the patient may present early in the disease process with non-specific signs and symptoms.[5]

This topic covers the diagnosis and management of necrotising fasciitis in adults only.

Full Recommendations

Clinical presentation

Always suspect necrotising fasciitis in a patient with a rapidly progressing soft-tissue infection and any of the following:[3]

- Severe pain (disproportionate to the clinical findings) or anaesthesia over the site of infection[1] [2] [3][16][19] [20][34] [35]
- Oedema and erythema; oedema will typically extend beyond the erythema[3]
- Systemic signs of infection.[1] [2][3] [16][35]

Other possible symptoms of necrotising fasciitis include lightheadedness, palpitations, and nausea or vomiting, or delirium.

Practical tip

Many cases of necrotising fasciitis begin as cellulitis or are misdiagnosed as cellulitis; the patient may present without systemic signs of infection.[5] [3] The early differential diagnosis between cellulitis and a necrotising infection that requires prompt surgical intervention may be difficult.[3] See Differentials and Cellulitis and erysipelas .

Be aware that necrotising fasciitis is a **clinical diagnosis** with signs and symptoms that change rapidly over time.[3] [5] Early recognition is critical because necrotising fasciitis is a life-threatening surgical emergency.

- Have a high index of suspicion; the diagnosis can be easily missed because the patient may present early in the disease process with non-specific signs and symptoms.[5]

If you suspect necrotising fasciitis, refer the patient immediately for **surgical debridement** and discuss the patient early with the critical care team; do not wait for the results of investigations.

History

Take a detailed history; specifically ask about risk factors, including:[1] [2] [16][24][30] [43]

- Preceding skin lesions or breakdown
- Trauma, surgery
    - Necrotising fasciitis in the context of recent abdominal surgery or in the groin is most likely to be polymicrobial
- Immunosuppression due to chronic illness (e.g., diabetes mellitus, alcohol dependence)
- Intravenous drug use
- Chickenpox
- Herpes zoster
- Hospitalisation.

Be aware that the **inciting insult may be minor** (e.g., an insect bite) and/or not recalled by the patient.[1] [26]

Exposure history may occasionally be helpful (e.g., freshwater exposure associated with *Aeromonas hydrophila* , saltwater exposure or consumption of raw oysters associated with *Vibrio vulnificus*); however, initial selection of empirical antibiotics should be broad and not guided solely by historical exposures.[9] [26]

Physical examination

Assess the patient for **systemic signs of infection** such as tachycardia, tachypnoea, and hypotension, and toxic shock syndrome (caused by infection with group A streptococcus).[1] [2][3] [16][35] See Toxic shock syndrome .

- Bear in mind that many patients present without systemic signs of infection.[5]

Sepsis and multi-organ failure may also be present.[3] [37] Think ' ***Could this be sepsis?*** ' based on acute deterioration in an adult patient in whom there is clinical evidence or strong suspicion of infection.[38] [39] [40] See Sepsis in adults.

More info: Sepsis

Think '**Could this be sepsis?**' based on acute deterioration in an adult patient in whom there is clinical evidence or strong suspicion of infection.[38] [39] [40]

- The patient may present with non-specific or non-localised symptoms (e.g., acutely unwell with a normal temperature) or there may be severe signs with evidence of multi-organ dysfunction and shock.[38] [39] [40]

- Remember that sepsis represents the severe, life-threatening end of infection.[44]
- Necrotising fasciitis is a rapidly progressive disease that can quickly lead to overwhelming sepsis and death; mortality in patients who develop shock and end-organ damage approaches 50% to 70%.[2]

Use a systematic approach (e.g., the National Early Warning Score 2 [NEWS2]), alongside your clinical judgement, to assess the risk of deterioration due to sepsis.[38] [39] [41] [45] Consult local guidelines for the recommended approach at your institution.

Arrange urgent review by a senior clinical decision-maker (e.g., ST4 level doctor in the UK) if you suspect sepsis:[42]

- **Within 30 minutes** for a patient who is critically ill (e.g., NEWS2 score of 7 or more, evidence of septic shock, or other significant clinical concerns)
- **Within 1 hour** for a patient who is severely ill (e.g., NEWS2 score of 5 or 6).

Follow your local protocol for investigation and treatment of all patients with suspected sepsis, or those at risk. Start treatment promptly. Determine urgency of treatment according to likelihood of infection and severity of illness, or according to your local protocol.[42] [45]

In the community: refer for emergency medical care in hospital (usually by blue-light ambulance in the UK) any patient who is acutely ill with a suspected infection and is:[40]

- Deemed to be at high risk of deterioration due to organ dysfunction (as measured by risk stratification)
- At risk of neutropenic sepsis.

If you suspect sepsis due to necrotising fasciitis, immediately refer the patient to the surgical team for inspection, exploration, and debridement of infected tissue.[2] [3] [5][42]

See Sepsis in adults .

Features of necrotising fasciitis that distinguish it from cellulitis include:

- Severe pain or anaesthesia over the site of infection[1] [2][3][16][19] [20] [34] [35]
    - The pain experienced with necrotising fasciitis may be disproportionate to the visible skin changes[35]

- Skin changes to the area overlying the infection such as crepitus, vesicles, bullae, greyish discoloration, or oedema extending beyond erythema
    - However, be aware that the patient can present with normal overlying skin, and that skin changes overlying group A streptococcal necrotising fasciitis are a late sign

- Subtle skin changes such as leakage of fluid and oedema precede the overt skin changes of blistering and redness.

The extremities are the most common site for necrotising fasciitis.

- About half of cases occur in the extremities, with the remainder affecting the perineum, trunk, or head and neck.[1][3] [4] [5][16] [19] [20]
- The most common site of group A streptococcal necrotising fasciitis is the thigh. Necrotising fasciitis of a limb, especially the arm, is more likely to be due to group A streptococci than a polymicrobial infection.
- Some cases of necrotising fasciitis may have associated myositis due to contiguous spread. This is more common in group A streptococcal than polymicrobial infections.

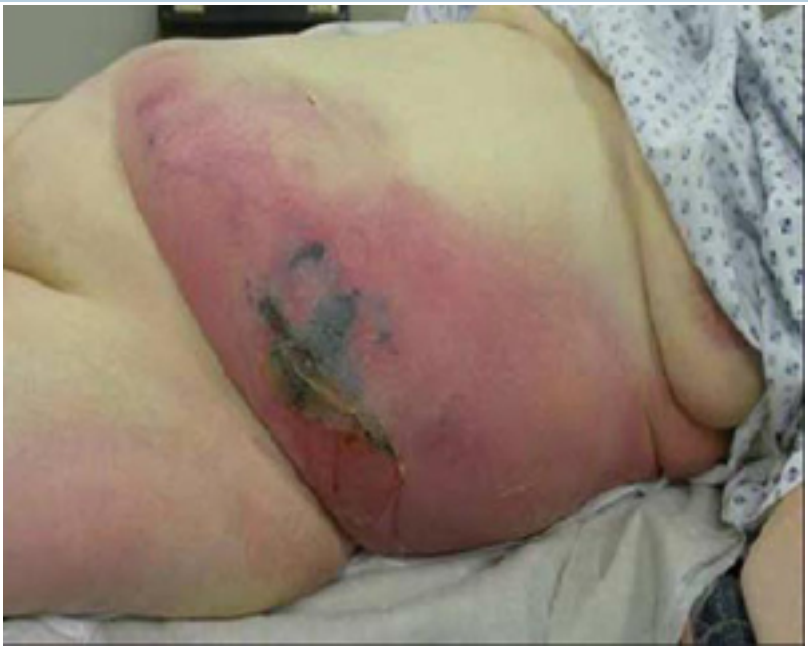
Emergency surgical exploration

If you suspect necrotising fasciitis clinically, immediately refer the patient for inspection, exploration, and debridement of infected tissue.[5] [46]

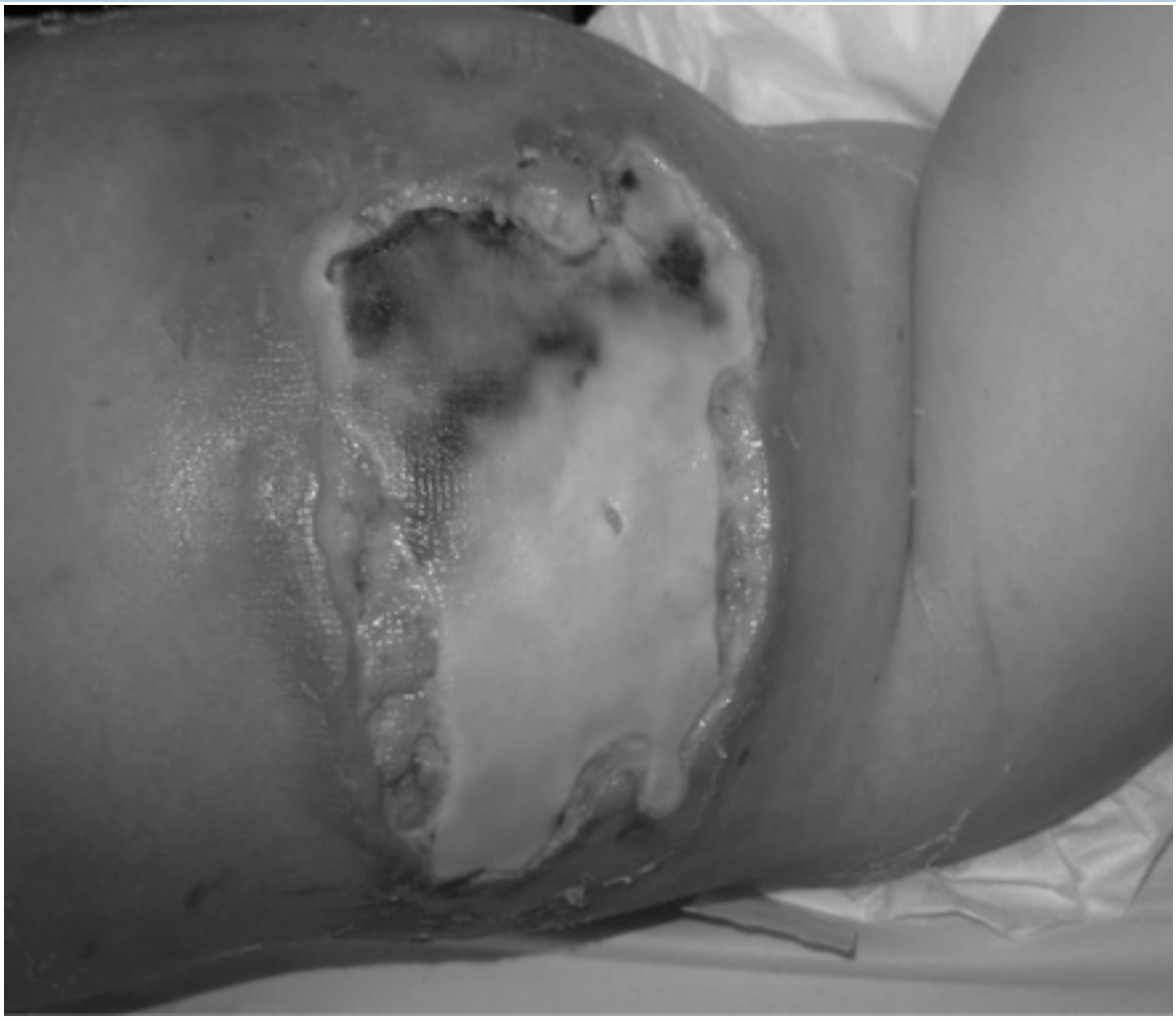
- The 'finger test' is a surgical method that can be performed under local anaesthesia at the bedside for the diagnosis of necrotising fasciitis.[3] It involves making a 2 cm incision down to the deep fascia. Findings that suggest necrotising fasciitis following incision include:[3]
    - Minimal resistance to finger dissection (a 'positive' finger test)
    - Absence of bleeding
    - Presence of necrotic tissue
    - Murky or greyish 'dishwater' fluid.

Definitive bacteriological diagnosis is best made from tissue specimens obtained from surgical debridement.[2] Gram staining of clinically affected tissue may provide an early indication of the causative organism(s). For example, small chains of gram-positive cocci suggest a streptococcal infection, whereas clumps of large cocci suggest *Staphylococcus aureus* .

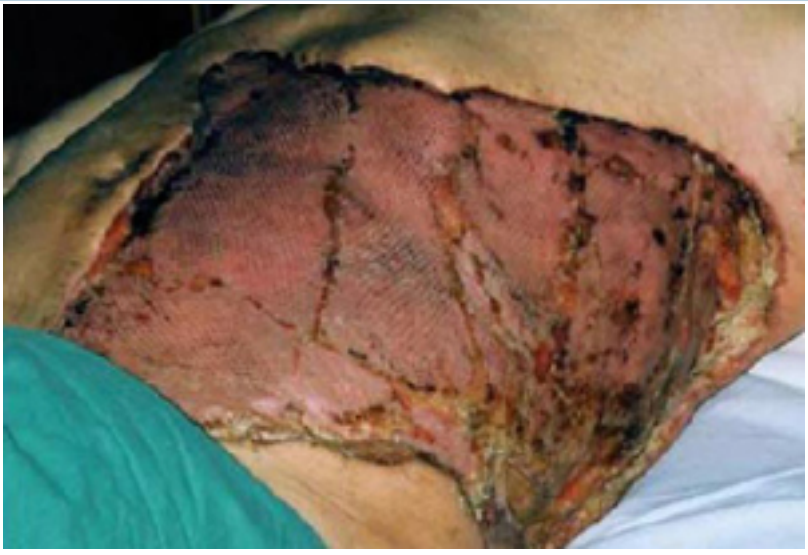
- Early frozen-section soft-tissue biopsy can provide a definitive diagnosis and may be used if the diagnosis is unclear clinically or radiologically.[3] However, frozen-section soft-tissue biopsy requires specialist pathology expertise, takes time to perform, and is not widely available in all regions, including in the UK.[3]
- Necrotising fasciitis is classified according to the underlying pathogen as type I or II – see Classification .[5]



Late signs of necrotising fasciitis with extensive cellulitis, induration, skin necrosis, and formation of haemorrhagic bullae
From: Hasham S, Matteucci P, Stanley PRW, et al. Necrotising fasciitis. BMJ. 2005 Apr 9;330(7495):830-3



Necrotising fasciitis on the right abdomen of a 2-year old girl following varicella infection
From: de Benedictis FM, Osimani P. Necrotising fasciitis complicating varicella. BMJ Case Rep. 2009;2009:bcr2008141994



Split thickness skin grafting after surgical debridement
From: Hasham S, Matteucci P, Stanley PRW, et al. Necrotising fasciitis. BMJ. 2005 Apr 9;330(7495):830-3

Investigations

If you suspect necrotising fasciitis, immediately refer the patient to the surgical team; do not wait for the results of investigations before referral.[2] [3]

Necrotising fasciitis is a clinical diagnosis. However, investigations can support the diagnosis if this is unclear.[3]

Laboratory tests

Always order:

- Blood cultures: obtain these as soon as possible and before starting antibiotics, to help identify the causative organism[3]
- Full blood count with white cell differential: may show abnormally high or low white blood cell count with or without a left shift (elevated percentage of polymorphonuclear leukocytes and/or bands)
- Urea, electrolytes, and creatinine: urea and creatinine may be elevated due to intracellular volume depletion; serum sodium may be low
- C-reactive protein: usually elevated creatine kinase: may be elevated
- Liver function tests: may be elevated if there is organ dysfunction due to sepsis
- Clotting screen: may show coagulopathy
- Blood gas (venous or arterial): lactate is usually elevated. Consider performing an arterial blood gas if you are concerned about respiratory compromise.

Imaging

Imaging may show **soft-tissue gas**, which is highly suggestive of the diagnosis; imaging may also demonstrate abnormalities in the involved soft tissue.[1][3] [4] [16]

Seek advice from a radiologist to determine the most appropriate imaging modality for your patient.

- Computed tomography (CT) is typically the radiological test of choice.[5]
    - Both CT and magnetic resonance imaging (MRI) offer higher sensitivity than x-ray. However, MRI may be difficult to organise in an emergency and is not recommended as the first-line imaging technique.[3] [4]
- Do not use a plain x-ray to rule out the diagnosis because x-ray is frequently normal during the early stages; subcutaneous gas may only be present as the disease progresses.[15]
- Bedside ultrasound may be performed if the patient is clinically unstable.[3] [4] In practice, however, bedside ultrasound is not widely used in all regions (including in the UK).
    - In one prospective study, ultrasound findings of diffuse thickening of the subcutaneous tissue, accompanied by fluid accumulation greater than 4 mm in depth, had a sensitivity of 88% and a specificity of 93%.[47]

Scoring tools for diagnosis and risk assessment

Use a scoring tool to:[3]

- Identify patients who are at risk of deterioration
- Identify those who need to be managed in a critical care setting

- Help distinguish necrotising fasciitis from less severe soft-tissue infections.

Commonly used examples include the following.

- The Laboratory Risk Indicator for Necrotising Fasciitis (LRINEC) score[3] [4]
  - LRINEC is based on laboratory parameters and was developed to assist with early discrimination of necrotising fasciitis from less severe skin and soft-tissue infections.[48]
  - Do not use LRINEC to rule out the diagnosis of necrotising fasciitis.[3] Validation studies have failed to demonstrate sufficient sensitivity or specificity to either diagnose or exclude necrotising fasciitis.[49] [50]
  - Suspect necrotising fasciitis if the patient scores 6 or more; a score of 8 or more is strongly predictive of necrotising fasciitis.[3]
  - An initial score of greater than 7 is associated with poorer outcomes and higher risk of death in necrotising fasciitis.[51]

- Portsmouth Physiological and Operative Severity Score for the enumeration of Mortality and Morbidity (P-POSSUM)
  - P-POSSUM is a tool that has been validated for estimating an individual patient's risk of death within 30 days of emergency general surgery, and is based on preoperative and perioperative factors.[52]
    - If you are using this tool preoperatively, estimate any perioperative factors and update these at the end of surgery.[52]
  - Transfer the patient to critical care if they have a predicted mortality risk ≥10%.[52]

History and exam

Key diagnostic factors

presence of risk factors (common)

Key risk factors include immunosuppression due to chronic illness (e.g., diabetes mellitus, alcohol dependence); cutaneous trauma, surgery, or ulcerative conditions; varicella zoster infections; intravenous drug use; and hospitalisation.[1] [2] [16] [24][30] [43]

Necrotising fasciitis in the context of recent abdominal surgery or in the groin is most likely to be polymicrobial.

anaesthesia or severe pain over site of infection (common)

Anaesthesia or severe pain over the site of infection indicates necrotising fasciitis.[1] [2] [16] [53] The pain experienced with necrotising fasciitis may be disproportionate to the visible skin changes.

fever (common)

Systemic symptom of infection, though present in only 40% of patients with necrotising fasciitis.[1] [2] [4][16][35]

palpitations, tachycardia, tachypnoea, hypotension, and lightheadedness (common)

Systemic symptoms/signs of infection.[1] [2] [4][16][35]

nausea and vomiting (common)

Systemic symptoms of infection.[1][35]

delirium (uncommon)

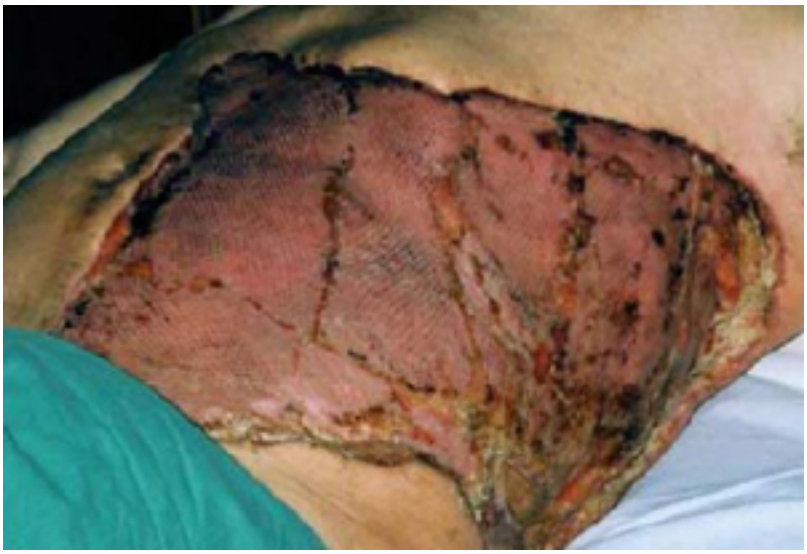
Systemic symptom of infection.[1] [2]

crepitus (uncommon)

Examination of the skin overlying the area of infection may reveal crepitus.[4] [16]

vesicles or bullae (uncommon)

Examination of the skin overlying the area of infection may reveal vesicles or bullae.[4] [16] It should be noted that patients with necrotising fasciitis can present with normal overlying skin and that skin changes overlying group A streptococcal necrotising fasciitis are a late sign.[16] Subtle skin changes such as leakage of fluid and oedema precede the overt skin changes of blistering and redness.



Split thickness skin grafting after surgical debridement

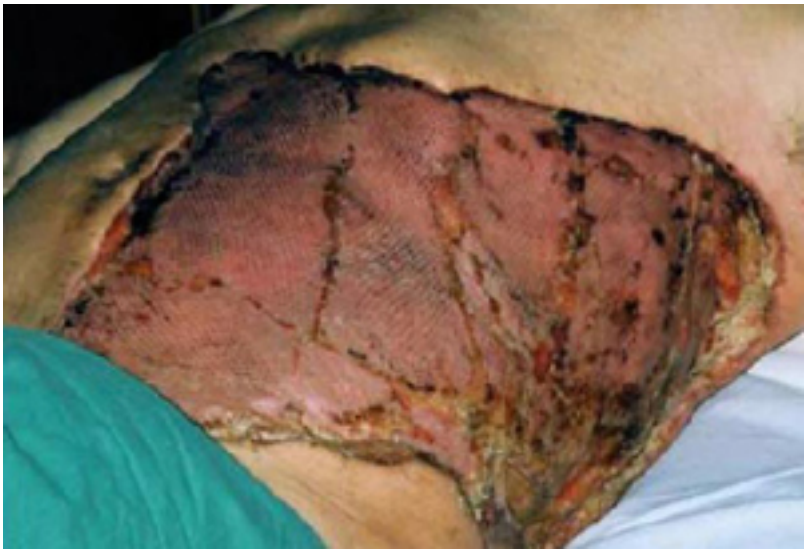
From: Hasham S, Matteucci P, Stanley PRW, et al. Necrotising fasciitis. BMJ. 2005 Apr 9;330(7495):830-3

grey discoloration of skin (uncommon)

Examination of the skin overlying the area of infection may reveal greyish discoloration. It should be noted that patients with necrotising fasciitis can present with normal overlying skin and that skin changes overlying group A streptococcal necrotising fasciitis are a late sign.

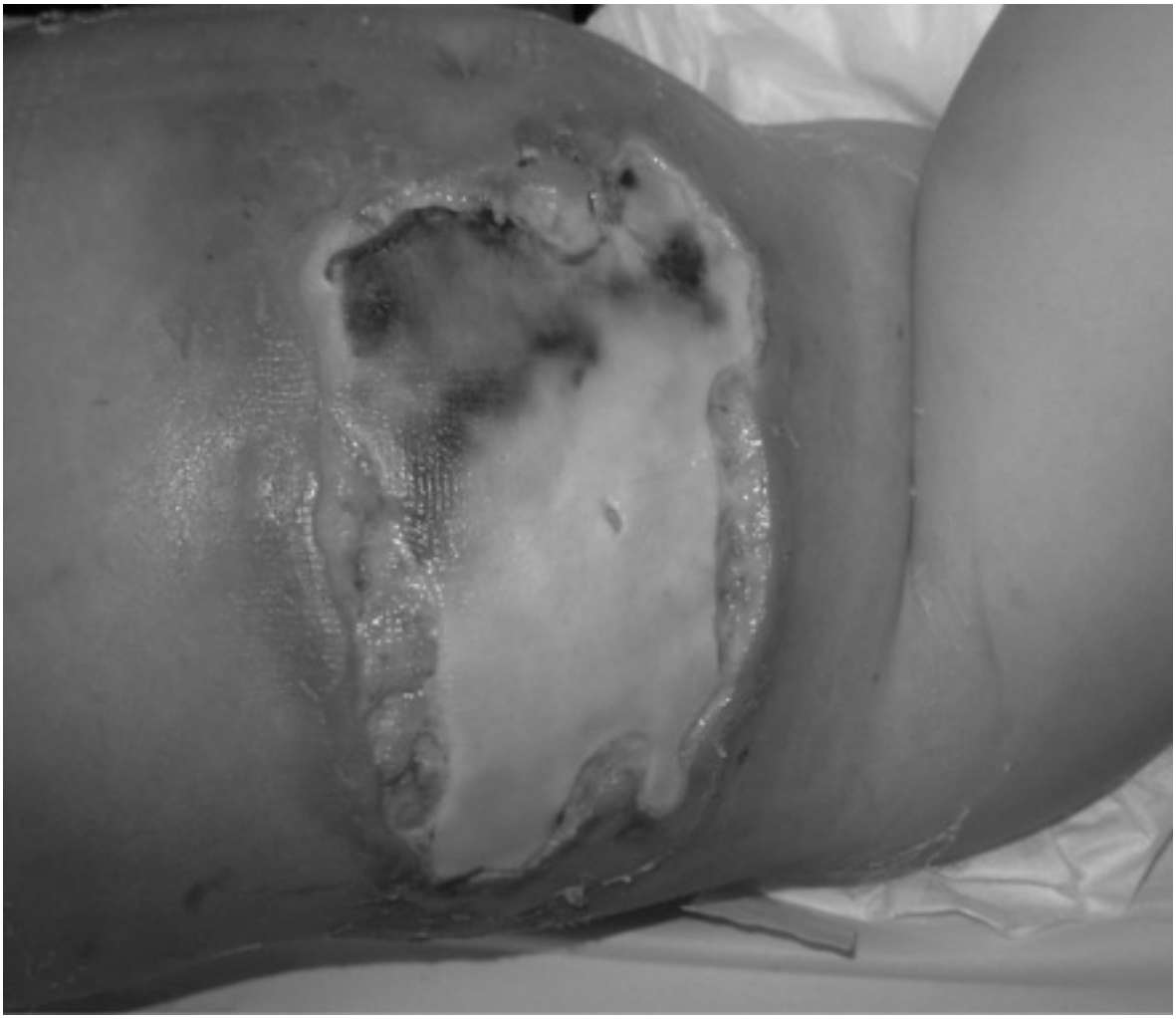
oedema or induration (uncommon)

Examination of the skin overlying the area of infection may reveal oedema.[4] Induration may be noted beyond the area of cellulitis. It should be noted that patients with necrotising fasciitis can present with normal overlying skin and that skin changes overlying group A streptococcal necrotising fasciitis are a late sign. Subtle skin changes such as leakage of fluid and oedema precede the overt skin changes of blistering and redness.



Split thickness skin grafting after surgical debridement

From: Hasham S, Matteucci P, Stanley PRW, et al. Necrotising fasciitis. BMJ. 2005 Apr 9;330(7495):830-3



Necrotising fasciitis on the right abdomen of a 2-year old girl following varicella infection

From: de Benedictis FM, Osimani P. Necrotising fasciitis complicating varicella. BMJ Case Rep. 2009;2009:bcr2008141994

location of lesion (uncommon)

About half of cases occur in the extremities, with the remainder affecting the perineum, trunk, or head and neck.[1] [2] [16][19] [20] The most common site of group A streptococcal necrotising fasciitis is the thigh. Necrotising fasciitis of a limb, especially the arm, is more likely to be due to group A streptococcus than a polymicrobial infection. Some cases of necrotising fasciitis may have associated myositis due to contiguous spread. This is more common in group A streptococcal than polymicrobial infections.

Investigations

1st test to order

Test	Result
surgical exploration If you suspect necrotising fasciitis clinically, refer the patient immediately for inspection, exploration, and debridement of infected tissue. The 'finger test' is a surgical method that can be performed under local anaesthesia at the bedside for the diagnosis of necrotising fasciitis.[3] It involves making a 2 cm incision down to the deep fascia. Findings that suggest necrotising fasciitis following incision include:[3] • Minimal resistance to finger dissection (a 'positive' finger test) • Absence of bleeding • Presence of necrotic tissue • Murky or greyish 'dishwater' fluid.	necrotising soft-tissue infection on surgical exploration positive finger test, absence of bleeding, presence of necrotic tissue, murky or greyish 'dishwater' fluid following incision
blood and tissue cultures Definitive bacteriological diagnosis is best made using tissue specimens obtained from surgical debridement and blood cultures.[2]	positive; may indicate polymicrobial or monomicrobial aetiology
Gram stain Staining of clinically affected tissue may provide early indication of causative organism(s). For example, small chains of gram-positive cocci suggest a streptococcal infection; clumps of large cocci suggest *Staphylococcus aureus* .	variable
full blood count and differential High WBC count is a non-specific finding that may be seen in any systemic infection or circulatory collapse. A low WBC count may be a sign of severe sepsis. If you suspect necrotising fasciitis, immediately refer the patient to the surgical team; do not wait for the results of investigations before referral.[2] [3] Necrotising fasciitis is a clinical diagnosis. However, investigations can support the diagnosis if this is unclear.[3]	abnormally high or low WBC count with or without a left shift (elevated percentage of polymorphonuclear leukocytes and/or bands)
serum electrolytes Hyponatraemia is a non-specific finding that may be seen in any systemic infection or circulatory collapse. If a spreading soft-tissue infection is present, necrotising fasciitis should be suspected.[2] [3] [4] [5] If you suspect necrotising fasciitis, immediately refer the patient to the surgical team; do not wait for the results of investigations before referral.[2] [3] Necrotising fasciitis is a clinical diagnosis. However, investigations can support the diagnosis if this is unclear.[3]	sodium may be decreased

Test	Result
serum urea and creatinine Elevated urea and creatinine may be seen due to intracellular volume depletion, and in any systemic infection or circulatory collapse. If you suspect necrotising fasciitis, immediately refer the patient to the surgical team; do not wait for the results of investigations before referral.[2] [3] [4] [5] Necrotising fasciitis is a clinical diagnosis. However, investigations can support the diagnosis if this is unclear.[3]	serum urea and creatinine may be elevated
serum CRP Elevated CRP is a non-specific finding that may be seen in a range of systemic infections. If you suspect necrotising fasciitis, immediately refer the patient to the surgical team; do not wait for the results of investigations before referral.[2] [3] [4] [5] Necrotising fasciitis is a clinical diagnosis. However, investigations can support the diagnosis if this is unclear.[3]	usually elevated
serum creatine kinase A non-specific finding suggestive of systemic infection or circulatory collapse. If you suspect necrotising fasciitis, immediately refer the patient to the surgical team; do not wait for the results of investigations before referral.[2] [3] [4] [5] Necrotising fasciitis is a clinical diagnosis. However, investigations can support the diagnosis if this is unclear.[3]	may be elevated
liver function tests May be elevated if there is organ dysfunction due to sepsis. If you suspect necrotising fasciitis, immediately refer the patient to the surgical team; do not wait for the results of investigations before referral.[2] [3] [4] [5] Necrotising fasciitis is a clinical diagnosis. However, investigations can support the diagnosis if this is unclear.[3]	may be elevated
serum lactate A non-specific finding suggestive of systemic infection. Elevated serum lactate at admission appears to be associated with the presence of necrotising fasciitis.[15] [54]	usually elevated
clotting screen Use to determine whether the patient has established coagulopathy in the presence of sepsis. This is associated with a worse prognosis.[55] If you suspect necrotising fasciitis, immediately refer the patient to the surgical team; do not wait for the results of investigations before referral.[2] [3] [4] [5] Necrotising fasciitis is a clinical diagnosis. However, investigations can support the diagnosis if this is unclear.[3]	may show coagulopathy
blood gas (venous or arterial) Acidosis may be present in the setting of sepsis. Obtain an arterial blood gas if you are concerned about respiratory compromise in order to determine the patient's respiratory status.	acidosis may be present and lactate is usually elevated

Test	Result
If you suspect necrotising fasciitis, immediately refer the patient to the surgical team; do not wait for the results of investigations before referral.[2] [3] [4] [5] Necrotising fasciitis is a clinical diagnosis. However, investigations can support the diagnosis if this is unclear.[3]	arterial blood gas may show hypoxaemia

Other tests to consider

Test	Result

CT/MRI, x-ray, ultrasound
Necrotising fasciitis is a clinical diagnosis.[4] However, investigations can support the diagnosis if this is unclear.[3]
Imaging may show **soft-tissue gas**, which is highly suggestive of the diagnosis; imaging may also demonstrate abnormalities in the involved soft tissue.[1][3] [16]
Seek advice from a radiologist to determine the most appropriate imaging modality for your patient.
• CT is the imaging of choice.[5]
  • Both CT and MRI offer higher sensitivity than x-ray. However, MRI may be difficult to organise in an emergency and is not recommended as the first-line imaging technique.[3] [4]
• Do not use a plain x-ray to rule out the diagnosis because x-ray is frequently normal during the early stages; subcutaneous gas may only be present as the disease progresses.[15]
• Bedside ultrasound may be performed if the patient is clinically unstable.[3] In practice, however, bedside ultrasound is not widely used in all regions (including in the UK).
  • In one prospective study, ultrasound findings of diffuse thickening of the subcutaneous tissue, accompanied by fluid accumulation greater than 4 mm in depth, had a sensitivity of 88% and a specificity of 93%.[47]
	oedema extending along fascial plane and/or soft-tissue gas

fresh frozen section Early frozen-section soft-tissue biopsy can provide a definitive diagnosis and it may be used if the diagnosis is unclear clinically or radiologically.[3] However, frozen-section soft-tissue biopsy requires specialist pathology expertise, takes time to perform, and is not widely available in all regions, including in the UK.[3]	evidence of bacteria and tissue necrosis

Differentials

Condition	Differentiating signs / symptoms	Differentiating tests
Cellulitis	• Systemic toxicity should be absent or minimal.[2]	• Absence of major abnormalities in full blood count, serum biochemistry, imaging findings.
Impetigo	• Patchy distribution of superficial blistering, with or without bullae, with crusting and erythema. May be asymptomatic or with pruritus.	• Culture of infected tissue identifies *Staphylococcus aureus* or *Streptococcus pyogenes* .[2]
Erysipelas	• Painful bright red, tender plaque with clear margins.[2]	• Culture of infected tissue identifies *S pyogenes* or other streptococci.[2]
Myositis	• No involvement of skin or soft tissue. Swelling over involved area is present but may not be painful. Unusual to see systemic signs/symptoms of toxicity. • Some cases of necrotising fasciitis may have associated myositis due to contiguous spread. This is more common in group A streptococcal than polymicrobial infections.	• Ultrasound or CT/MRI to identify focal involvement of muscle with swelling. • MRI can also identify oedema.
Cutaneous anthrax	• History of intravenous drug use, or contact with animals or their products (e.g. hides, wool). Painless, pruritic papule forms 2 to 5 days after exposure. Lesion becomes vesicular, evolving into a necrotic black eschar with massive surrounding oedema 24 to 36 hours later. Regional lymphadenopathy is common.	• Vesicular fluid/blood Gram stain and culture: gram-positive bacilli in short chains (*Bacillus anthracis*); flat, non-haemolytic mucoid colonies on 5% sheep's blood agar. • Punch biopsy of cutaneous lesion: necrosis of the dermis and epidermis, oedema, and mild inflammatory infiltrate; abundant bacillary fragments (prior to antibiotic therapy); *Bacillus anthracis* (post-antibiotics).

Recommendations

Key Recommendations

Refer the patient for **emergency** surgical debridement as soon as you suspect necrotising fasciitis and get an early review from the critical care team; this is a life-threatening surgical scenario.[3] [5]

- Surgical debridement should be performed as soon as possible, but at least within 12 hours of hospital admission, to reduce the number of subsequent debridements, progression to organ failure, and mortality.[3] [56]
- Repeated surgical debridement is usually required.[3] [37]

Start **empirical antibiotics** as soon as you have obtained blood cultures and ensure the patient has adequate haemodynamic support (intravenous fluids ± vasoactive drugs) and analgesia.[3] [35]

- Urgently discuss choice of antibiotic with an infectious disease or microbiology specialist.
- Once culture results are available and the causative organism is identified, tailor the patient's antibiotics accordingly.[3]

This topic covers the diagnosis and management of necrotising fasciitis in adults only.

Full Recommendations

Immediate surgical referral

Refer the patient immediately to the surgical team; necrotising fasciitis requires rapid debridement of the infected subcutaneous tissues in combination with empirical antibiotic therapy.[2][3] [4] [20]

- The infected subcutaneous tissue is devitalised, so expedited surgical removal of all infected tissue, drainage of infected fluids, and removal of infected devices or foreign bodies is critical for successful treatment.[3] Surgical debridement should be performed as soon as possible, but within 6-12 hours of hospital admission.[3] [15] [46] [57]
    - Delay in surgical debridement (>12 hours after admission) has been associated with the need for a greater number of subsequent debridements, higher incidence of organ failure, and higher mortality.[4] [46][56]
- Surgical specimens including tissue and fluid should be obtained for microbiological culture.[2] [3]
- A multidisciplinary approach (including a surgeon, an infectious disease or microbiology specialist, and critical care) is required.[15] [53]

While the patient is waiting for surgery, monitor them for systemic toxicity (e.g., signs of end-organ damage), as well as local signs and symptoms of extension of the area of necrotising fasciitis.[4]

Consider the need for additional debridement or alterations in antibiotic or antifungal therapy, based on culture results from subcutaneous tissue or blood, the patient's clinical condition, and discussion with the multidisciplinary team.[2][3] [4] See *Surgical re-exploration* below.

More info: Surgical debridement

When surgical debridement is performed, incisions should extend beyond the areas of visible necrosis and the entire necrotic area excised.[4] Further surgical evaluation and debridement is necessary in most cases, and several procedures may be required to ensure that all necrotic tissue is removed. Data to guide optimal timing for surgical re-exploration are lacking; a reasonable approach may be serial debridement every 12 to 24 hours until minimal or no remaining necrotic tissue is encountered.[3]

Supportive care

Get an early review from the **critical care** team. Some patients will need critical care support, depending on the degree of surgical resection and their physiological state.

Give intensive haemodynamic support with intravenous fluids, and vasoactive drugs if needed.[2] [3] [4]

- Check local protocols for specific recommendations on fluid choice. Evidence from critically ill patients in general (not specifically people with necrotising fasciitis) suggests that there is no difference in benefit between normal saline and a balanced crystalloid (such as Hartmann's solution [also known as Ringer's lactate] or Plasma-Lyte®), and therefore either choice of fluid is reasonable.[58] [59]

Ensure the patient has adequate analgesia.[3] Morphine is used in practice.

Practical tip

Be aware that large volumes of normal saline as the sole fluid for resuscitation may lead to hyperchloremic acidosis.
Also note that use of lactate-containing fluid in a patient with impaired liver metabolism may lead to a spuriously elevated lactate level, so results need to be interpreted with other markers of volume status.

Evidence: Choice of fluids

Evidence from two large randomised controlled trials (RCTs) suggests there is no difference between normal saline and a balanced crystalloid in mortality at 90 days, and therefore either option is a reasonable choice for the resuscitation of critically ill patients.

There has been extensive debate over the choice between normal saline (an unbalanced crystalloid) versus a balanced crystalloid (such as Hartmann's solution [also known as Ringer's lactate] or Plasma-Lyte®). Evidence from critically ill patients points to no benefits from using a balanced crystalloid in preference to normal saline. Clinical practice varies widely, so you should check local protocols.

- In 2021 to 2022 two large double-blind RCTs were published assessing intravenous fluid resuscitation in intensive care unit (ICU) patients with a balanced crystalloid solution (Plasma-Lyte) versus normal saline: the Plasma-Lyte 148 versus Saline (PLUS) trial (53 ICUs in Australia and New Zealand; N=5037) and the Balanced Solutions in Intensive Care Study (BaSICS) trial (75 ICUs in Brazil; N=11,052).[58] [59]
    - In the PLUS study 45.2% of patients were admitted to ICU directly from surgery (emergency or elective), 42.3% had sepsis and 79.0% were receiving mechanical ventilation at the time of randomisation.
    - In BaSICS almost half the patients (48.4%) were admitted to ICU after elective surgery and around 68% had some form of fluid resuscitation before being randomised.
    - Both found no difference in 90-day mortality overall or in prespecified subgroups for patients with acute kidney injury (AKI), sepsis, or post-surgery. They also found no difference in the risk of AKI.
    - In BaSICS, for patients with traumatic brain injury, there was a small decrease in 90-day mortality with normal saline - however, the overall number of patients was small (<5% of total included in the study) so there is some uncertainty about this result. Patients with traumatic brain injury were excluded from PLUS as the authors felt these patients should be receiving saline or a solution of similar tonicity.
- One meta-analysis of 13 RCTs (including PLUS and BaSICS) confirmed no overall difference, although the authors did highlight a non-significant trend towards a benefit of balanced solutions for risk of death.[60]
- Previous evidence has been mixed.
    - One 2015 double-blind, cluster-randomised, double-crossover trial conducted in four ICUs in New Zealand (N=2278), the 0.9% Saline vs. Plasma-Lyte for ICU fluid Therapy (SPLIT) trial, found no difference for in-hospital mortality, AKI, or use of renal-replacement therapy.[61]
    - However, one 2018 US multicentre unblinded cluster-randomised trial - the isotonic Solutions and Major Adverse Renal events Trial (SMART), among 15,802 critically ill adults receiving ICU care - found possible small benefits from balanced crystalloid (Ringer's lactate or Plasma-Lyte) compared with normal saline. The 30-day outcomes showed a non-significant reduced mortality in the balanced crystalloid group versus the normal saline group (10.3% vs. 11.1%; odds ratio [OR] 0.90, 95% CI 0.80 to 1.01) and a

major adverse kidney event rate of 14.3% versus 15.4% respectively (OR 0.91, 95% CI 0.84 to 0.99).[62]

- One 2019 Cochrane review included 21 RCTs (N=20,213) assessing balanced crystalloids versus normal saline for resuscitation or maintenance in a critical care setting.[63]
  - The three largest RCTs in the Cochrane review (including SMART and SPLIT) all examined fluid resuscitation in adults and made up 94.2% of participants (N=19,054).
  - There was no difference in in-hospital mortality (OR 0.91, 95% CI 0.83 to 1.01; high-quality evidence as assessed by GRADE), AKI (OR 0.92, 95% CI 0.84 to 1.00; GRADE low), or organ system dysfunction (OR 0.80, 95% CI 0.40 to 1.61; GRADE very low).

Antibiotics

Empirical antibiotic therapy

Start empirical intravenous antibiotic therapy **as soon as you have obtained blood cultures**.[2] [3] [35] Urgently discuss choice of antibiotic with an infectious disease or microbiology specialist.

- Use high-dose broad-spectrum antibiotics that target the most common aetiologies of:
  - Type I infection
    - Anaerobes such as *Bacteroides* or *Peptostreptococcus* with a facultative anaerobe such as certain Enterobacterales (*Escherichia coli* , *Enterobacter* , *Klebsiella* , *Proteus*), MRSA, or non-group A streptococcus
  - Type II infection
    - Group A streptococcus.
- Consider local resistance and epidemiological patterns (including extended-spectrum beta-lactamase or carbapenemase-producing organisms).
- Appropriate antibiotics include piperacillin/tazobactam, a carbapenem (e.g.,meropenem), a cephalosporin (e.g., ceftriaxone), or a fluoroquinolone such as ciprofloxacin (if the patient is allergic to penicillin).
- Always add vancomycin, linezolid, tedizolid, or daptomycin for MRSA cover.[3]
- Add antibiotics that inhibit toxin production until group A streptococcus involvement is excluded.[3] Evidence for clindamycin is strongest.[2] [37]
- Fungal pathogens (Mucorales, *Candida*) are rare causes of necrotising fasciitis; empirical antifungal agents are not recommended.

Continue empirical antibiotics until the causative organism has been determined. Once these results are available, tailor antibiotic therapy accordingly.

Drug safety alert: Fluoroquinolones

Fluoroquinolones have been associated with serious, disabling, and potentially irreversible adverse effects, including tendonitis, tendon rupture, arthralgia, neuropathies, and other musculoskeletal or nervous system effects.[64] Warnings have also been issued about the increased risk of aortic dissection, significant hypoglycaemia, and mental health adverse effects in patients taking fluoroquinolones.[65] [66]

Pathogen-targeted antibiotic therapy

Once culture results are available, tailor the patient's antibiotics to target the causative organism.[4] Always consult an infectious disease or microbiology specialist (this is particularly important if the patient has multi-drug resistant necrotising fasciitis or is allergic to penicillin) and consider local epidemiological patterns.

Continue antibiotics until further debridement (see *Surgical re-exploration* below) is no longer necessary, the patient has clinically improved, and fever has resolved for 48 to 72 hours.[3] [67]

Type I infection (polymicrobial)

Type I infection involves aerobic and anaerobic organisms.[3] It is most commonly seen in older patients and in those with underlying illnesses.[8]

Suitable antibiotic regimens include piperacillin/tazobactam, a carbapenem (e.g., meropenem), a cephalosporin (e.g., ceftriaxone), or a fluoroquinolone such as ciprofloxacin (if the patient is allergic to penicillin).

- Continue vancomycin, linezolid, tedizolid, or daptomycin if MRSA is confirmed. Continue an antibiotic that inhibits toxin production until group A streptococcus involvement is excluded.[3] Evidence for clindamycin is strongest.[2] [37]

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Type II infection

Monomicrobial: group A streptococcus or Staphylococcus aureus

Type II infection is most commonly due to group A streptococcus; clindamycin plus a penicillin is recommended.[2][37]

- If the patient has a penicillin allergy, vancomycin monotherapy may be used.

S aureus may also cause type II infection.[3]

- Use antibiotics that are active against MRSA until cultures confirm susceptibilities; options include vancomycin, linezolid, tedizolid, or daptomycin. Ceftaroline or dalbavancin are also reasonable choices.[2] [3] In practice, tigecycline may also be used.
- Flucloxacillin or cefazolin may be used if methicillin susceptibility is confirmed.[2]

Monomicrobial: gram-negative or clostridium organisms

Monomicrobial infection with gram-negative or clostridium organisms is rare.[5] It includes infection with:

- *Vibrio vulnificus* : predisposing risk factors include hepatic disease, diabetes mellitus, chronic renal insufficiency, and adrenal insufficiency[9]
- *Aeromonas hydrophila* : most common in patients with immunosuppression, burns, and trauma in an aquatic setting[10]
- *Klebsiella pneumoniae* , in South East Asian countries, in particular Taiwan[11]
- *Clostridium* : can also cause gangrenous necrotising fasciitis – see Gangrene .

Doxycycline should be included in the management of necrotising fasciitis due to *V vulnificus* or *A hydrophila* .[2]

Fungal infection

Fungal infection is rare and is the result of infection with pathogens such as Mucorales and *Candida* species. Seek advice from an infectious disease or microbiology specialist. Liposomal amphotericin-B is usually the primary treatment option.

- Although necrotising mucormycosis predominantly affects immunocompromised people, it may also occur in immunocompetent individuals.[12] [13] [14]

Streptococcal toxic shock syndrome

If the patient develops streptococcal toxic shock syndrome (TSS), seek urgent advice from a senior colleague. Consider intravenous immunoglobulin (IVIG) as an adjunctive therapy for these patients.[3] [4] See Toxic shock syndrome . IVIG may also have a role in managing patients with streptococcal infection without TSS.[3]

More info: IVIG

Efficacy data for use of IVIG for necrotising fasciitis are conflicting. Some studies suggest modest benefit; however, one Cochrane review showed no clear benefit on adverse events or mortality.[68] [69] [70] [71] [72] [73]

The World Society of Emergency Surgery consensus recommendations suggest consideration of IVIG in patients with necrotising fasciitis due to group A streptococcus.[3] [4]

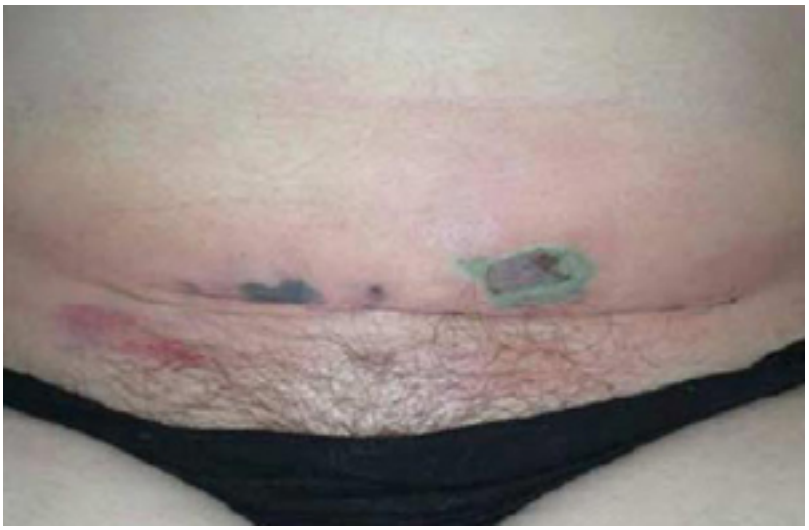
The Infectious Diseases Society of America guidelines do not include a recommendation regarding the use of IVIG in patients with necrotising fasciitis with streptococcal toxic shock syndrome, citing the need for additional efficacy studies.[2]

Surgical re-exploration

Consider the need for additional debridement or alterations in antibiotic or antifungal therapy, based on culture results from subcutaneous tissue or blood, the patient's clinical condition, and discussion with the multidisciplinary team.[2] [3] [4] [53]

- Surgical re-exploration to assess the need for further debridement should be performed at least every 12 to 24 hours after the initial debridement.[3] However, re-exploration may be needed sooner in some patients; inform the surgical team urgently if the patient has clinical signs of worsening infection (local or systemic), or worsening laboratory markers (particularly white blood cell count).[3]
- Re-exploration should be repeated until the patient has no necrotic tissue remaining.[3] [4]

If the patient has functional or cosmetic disability due to extensive surgical debridement, reconstructive surgery may be required. However, this should only be considered when the patient is stable and the infection has been completely eradicated.



Small areas of skin necrosis in a young woman with cellulitis and necrotizing fasciitis of her lower abdomen 5 days after a cesarean section

From: Hasham S, Matteucci P, Stanley PRW, et al. Necrotising fasciitis. BMJ. 2005 Apr 9;330(7495):830-3

Treatment algorithm overview

Please note that formulations/routes and doses may differ between drug names and brands, drug formularies, or locations. Treatment recommendations are specific to patient groups: see disclaimer

Initial		(summary)
suspected necrotising fasciitis, organism unknown		
	1st	**emergency surgical debridement**
	plus	**supportive care**
	plus	**empirical antibiotic therapy**

MANAGEMENT

Acute		(summary)
type I necrotising fasciitis (polymicrobial)		
	1st	surgical re-exploration ± debridement
	plus	supportive care
	plus	pathogen-targeted antibiotic therapy
	consider	MRSA antibiotic cover
	consider	group A streptococcus antibiotic cover
type II necrotising fasciitis due to group A streptococcus		
	1st	surgical re-exploration ± debridement
	plus	supportive care
	plus	pathogen-targeted antibiotic therapy
	consider	intravenous immunoglobulin
type II necrotising fasciitis due to Staphylococcus aureus		
	1st	surgical re-exploration ± debridement
	plus	supportive care
	plus	pathogen-targeted antibiotic therapy
type II necrotising fasciitis due to Vibrio vulnificus		
	1st	surgical re-exploration ± debridement
	plus	supportive care
	plus	pathogen-targeted antibiotic therapy
type II necrotising fasciitis due to Aeromonas hydrophila		
	1st	surgical re-exploration ± debridement
	plus	supportive care
	plus	pathogen-targeted antibiotic therapy
type II necrotising fasciitis due to Clostridium		
	1st	surgical re-exploration ± debridement
	plus	supportive care
	plus	pathogen-targeted antibiotic therapy
fungal infection		

Acute		(summary)
	1st	surgical re-exploration ± debridement
	plus	supportive care
	plus	discussion with infectious disease or microbiology specialist and antifungal therapy

Ongoing		(summary)
persistent cosmetic and functional defects after debridement		
	1st	reconstructive surgery

Treatment algorithm

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Initial

suspected necrotising fasciitis, organism unknown

1st **emergency surgical debridement**

» Refer the patient immediately to the surgical team; necrotising fasciitis requires **rapid debridement** of all infected tissues in combination with empirical antibiotic therapy.[2] [3] [4][20][46]

- The infected subcutaneous tissue is devitalised, so expedited surgical removal of all infected tissue, drainage of infected fluids, and removal of infected devices or foreign bodies is critical for successful treatment.[3] Surgical debridement should be performed as soon as possible, but at least within 6-12 hours of hospital admission.[3] [15] [46] [57]
  - Delay in surgical debridement (>12 hours after admission) has been associated with the need for a greater number of subsequent debridements, higher incidence of organ failure, and higher mortality. [4] [46][56]
- Surgical specimens including tissue and fluid should be obtained for microbiological culture.[2] [3]
- A multidisciplinary approach (including a surgeon, an infectious disease or microbiology specialist, and critical care) is required.[15] [53]

» While the patient is waiting for surgery, monitor them for systemic toxicity (e.g., signs of end-organ damage), as well as local signs and symptoms of extension of the area of necrotising fasciitis.[4]

» Consider the need for additional debridement or alterations in antibiotic or antifungal therapy, based on culture results from subcutaneous tissue or blood, the patient's clinical condition, and discussion with the multidisciplinary team.[2] [3] [4] [53] See *Acute - surgical re-exploration ± debridement*.

Initial

plus **supportive care**

Treatment recommended for ALL patients in selected patient group

Primary options

» morphine sulfate: 5-10 mg orally (immediate-release)/subcutaneously/ intravenously/intramuscularly every 4 hours initially, adjust dose according to response

» Get an early review from the **critical care** team. Some patients will need critical care support depending on the degree of surgical resection and their physiological state.

» Give intensive haemodynamic support with intravenous fluids, and vasoactive drugs if needed.[2] [3] [4]

- Check local protocols for specific recommendations on fluid choice. Evidence from critically ill patients in general (not specifically people with necrotising fasciitis) suggests that there is no difference in benefit between normal saline and a balanced crystalloid (such as Hartmann's solution [also known as Ringer's lactate] or Plasma-Lyte®), and therefore either choice of fluid is reasonable.[58] [59]

Practical tip

Be aware that large volumes of normal saline as the sole fluid for resuscitation may lead to hyperchloremic acidosis. Also note that use of lactate-containing fluid in a patient with impaired liver metabolism may lead to a spuriously elevated lactate level, so results need to be interpreted with other markers of volume status.

Evidence: Choice of fluids

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- Previous evidence has been mixed.
    - One 2015 double-blind, cluster-randomised, double-crossover trial conducted in four ICUs in New Zealand (N=2278), the 0.9% Saline vs. Plasma-Lyte for ICU fluid Therapy (SPLIT) trial, found no difference for in-hospital mortality, AKI, or use of renal-replacement therapy.[61]
    - However, one 2018 US multicentre unblinded cluster-randomised trial - the isotonic Solutions and Major Adverse Renal events Trial (SMART), among 15,802 critically ill adults receiving ICU care - found possible small benefits from balanced crystalloid (Ringer's lactate or Plasma-Lyte) compared with normal saline. The 30-day outcomes showed a non-significant reduced mortality in the balanced crystalloid group versus the normal saline group (10.3% vs. 11.1%; odds ratio [OR] 0.90, 95% CI 0.80 to 1.01) and a major adverse kidney event rate of 14.3% versus 15.4% respectively (OR 0.91, 95% CI 0.84 to 0.99).[62]
- One 2019 Cochrane review included 21 RCTs (N=20,213) assessing balanced crystalloids versus normal saline for resuscitation or maintenance in a critical care setting.[63]

Initial

- The three largest RCTs in the Cochrane review (including SMART and SPLIT) all examined fluid resuscitation in adults and made up 94.2% of participants (N=19,054).
- There was no difference in in-hospital mortality (OR 0.91, 95% CI 0.83 to 1.01; high-quality evidence as assessed by GRADE), AKI (OR 0.92, 95% CI 0.84 to 1.00; GRADE low), or organ system dysfunction (OR 0.80, 95% CI 0.40 to 1.61; GRADE very low).

- Check your local protocols for choice and dose of vasoactive drugs.
- Ensure the patient has adequate analgesia.[3] Morphine is used in practice.

plus **empirical antibiotic therapy**

Treatment recommended for ALL patients in selected patient group

Primary options

» piperacillin/tazobactam: 4.5 g intravenously every 8 hours, may increase to 4.5 g every 6 hours in severe infections
Dose consists of 4 g of piperacillin plus 0.5 g of tazobactam.

-or-
» meropenem: 0.5 to 1 g intravenously every 8 hours
-or-
» ceftriaxone: 2 g intravenously every 24 hours
-or-
» ciprofloxacin: 400 mg intravenously every 8-12 hours

--AND--

» vancomycin: 15-20 mg/kg intravenously every 8-12 hours, maximum 2000 mg/dose

Initial

Adjust dose according to serum vancomycin level. A loading dose of 25-30 mg/kg may be considered in seriously ill patients.

-or-
» linezolid: 600 mg intravenously every 12 hours
-or-
» tedizolid phosphate: 200 mg intravenously every 24 hours
-or-
» daptomycin: 4-6 mg/kg intravenously every 24 hours

--AND--

» clindamycin: 600-2700 mg/day intravenously given in 2-4 divided doses, may increase to 1200 mg every 6 hours in life-threatening infections

» Start empirical intravenous antibiotic therapy **as soon as you have obtained blood cultures**.[2] [3] [35] **Urgently discuss** choice of antibiotic with an infectious disease or microbiology specialist.

- Use high-dose broad-spectrum antibiotics that target the most common aetiologies of:
    - Type I infection
        - Anaerobes such as *Bacteroides* or *Peptostreptococcus* with a facultative anaerobe such as certain Enterobacterales (*Escherichia coli* , *Enterobacter* , *Klebsiella* , *Proteus*), MRSA, or non-group A streptococcus
    - Type II infection
        - Group A streptococcus.
- Consider local resistance and epidemiological patterns (including extended-spectrum beta-lactamase or carbapenemase-producing organisms).
- Appropriate antibiotics include piperacillin/tazobactam, a carbapenem (e.g.,

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meropenem), a cephalosporin (e.g., ceftriaxone), or a fluoroquinolone such as ciprofloxacin (if the patient is allergic to penicillin).

- Always add vancomycin, linezolid, tedizolid, or daptomycin for MRSA cover.[3]
- Add antibiotics that inhibit toxin production until group A streptococcus involvement is excluded.[3] Evidence for clindamycin is strongest.[2] [37]
- Fungal pathogens (e.g., Mucorales, *Candida*) are rare causes of necrotising fasciitis; empirical antifungal agents are not recommended.

» Continue empirical antibiotics until the causative organism has been determined. Once these results are available, tailor antibiotic therapy accordingly.

Drug safety alert: Fluoroquinolones

Fluoroquinolones have been associated with serious, disabling, and potentially irreversible adverse effects, including tendonitis, tendon rupture, arthralgia, neuropathies, and other musculoskeletal or nervous system effects.[64] Warnings have also been issued about the increased risk of aortic dissection, significant hypoglycaemia, and mental health adverse effects in patients taking fluoroquinolones.[65] [66]

Acute

type I necrotising fasciitis (polymicrobial)

1st **surgical re-exploration ± debridement**

» Consider the need for additional debridement (following the initial debridement), based on culture results from subcutaneous tissue or blood, the patient's clinical condition, and discussion with the multidisciplinary team.[2] [3] [4] [53]

- Surgical re-exploration to assess the need for further debridement should be performed at least every 12 to 24 hours after the initial debridement.[3] However, re-exploration may be needed sooner in some patients; inform the surgical team urgently if the patient has clinical signs of worsening infection (local or systemic), or worsening laboratory markers (particularly white blood cell count).[3]
- Re-exploration should be repeated until the patient has no necrotic tissue remaining.[3]

plus **supportive care**

Treatment recommended for ALL patients in selected patient group

Primary options

» morphine sulfate: 5-10 mg orally (immediate-release)/subcutaneously/intravenously/intramuscularly every 4 hours initially, adjust dose according to response

» Get an early review from the **critical care** team. Some patients will need critical care support depending on the degree of surgical resection and their physiological state.

» Give intensive haemodynamic support with intravenous fluids, and vasoactive drugs if needed.[2] [3] [4]

» Check local protocols for specific recommendations on fluid choice. Evidence from critically ill patients in general (not specifically people with necrotising fasciitis) suggests that there is no difference in benefit between normal saline and a balanced crystalloid (such as Hartmann's solution [also known as Ringer's lactate] or Plasma-Lyte®), and therefore either choice of fluid is reasonable.[58] [59]

Acute

Practical tip

Be aware that large volumes of normal saline as the sole fluid for resuscitation may lead to hyperchloremic acidosis. Also note that use of lactate-containing fluid in a patient with impaired liver metabolism may lead to a spuriously elevated lactate level, so results need to be interpreted with other markers of volume status.

Evidence: Choice of fluids

Evidence from two large randomised controlled trials (RCTs) suggests there is no difference between normal saline and a balanced crystalloid in mortality at 90 days, and therefore either option is a reasonable choice for the resuscitation of critically ill patients.

There has been extensive debate over the choice between normal saline (an unbalanced crystalloid) versus a balanced crystalloid (such as Hartmann's solution [also known as Ringer's lactate] or Plasma-Lyte®). Evidence from critically ill patients points to no benefits from using a balanced crystalloid in preference to normal saline. Clinical practice varies widely, so you should check local protocols.

- In 2021 to 2022 two large double-blind RCTs were published assessing intravenous fluid resuscitation in intensive care unit (ICU) patients with a balanced crystalloid solution (Plasma-Lyte) versus normal saline: the Plasma-Lyte 148 versus Saline (PLUS) trial (53 ICUs in Australia and New Zealand; N=5037) and the Balanced Solutions in Intensive Care Study (BaSICS) trial (75 ICUs in Brazil; N=11,052).[58] [59]
    - In the PLUS study 45.2% of patients were admitted to ICU directly from surgery (emergency or elective), 42.3% had sepsis and 79.0% were receiving mechanical ventilation at the time of randomisation.

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- In BaSICS almost half the patients (48.4%) were admitted to ICU after elective surgery and around 68% had some form of fluid resuscitation before being randomised.
- Both found no difference in 90-day mortality overall or in prespecified subgroups for patients with acute kidney injury (AKI), sepsis, or post-surgery. They also found no difference in the risk of AKI.
- In BaSICS, for patients with traumatic brain injury, there was a small decrease in 90-day mortality with normal saline - however, the overall number of patients was small (<5% of total included in the study) so there is some uncertainty about this result. Patients with traumatic brain injury were excluded from PLUS as the authors felt these patients should be receiving saline or a solution of similar tonicity.

- One meta-analysis of 13 RCTs (including PLUS and BaSICS) confirmed no overall difference, although the authors did highlight a non-significant trend towards a benefit of balanced solutions for risk of death.[60]
- Previous evidence has been mixed.
  - One 2015 double-blind, cluster-randomised, double-crossover trial conducted in four ICUs in New Zealand (N=2278), the 0.9% Saline vs. Plasma-Lyte for ICU fluid Therapy (SPLIT) trial, found no difference for in-hospital mortality, AKI, or use of renal-replacement therapy.[61]
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Renal events Trial (SMART), among 15,802 critically ill adults receiving ICU care - found possible small benefits from balanced crystalloid (Ringer's lactate or Plasma-Lyte) compared with normal saline. The 30-day outcomes showed a non-significant reduced mortality in the balanced crystalloid group versus the normal saline group (10.3% vs. 11.1%; odds ratio [OR] 0.90, 95% CI 0.80 to 1.01) and a major adverse kidney event rate of 14.3% versus 15.4% respectively (OR 0.91, 95% CI 0.84 to 0.99).[62]

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  - There was no difference in in-hospital mortality (OR 0.91, 95% CI 0.83 to 1.01; high-quality evidence as assessed by GRADE), AKI (OR 0.92, 95% CI 0.84 to 1.00; GRADE low), or organ system dysfunction (OR 0.80, 95% CI 0.40 to 1.61; GRADE very low).

- Check your local protocols for choice and dose of vasoactive drugs.
- Ensure the patient has adequate analgesia.[3] Morphine is used in practice.

plus **pathogen-targeted antibiotic therapy**

Treatment recommended for ALL patients in selected patient group

Acute

Primary options

» piperacillin/tazobactam: 4.5 g intravenously every 8 hours, may increase to 4.5 g every 6 hours in severe infections
Dose consists of 4 g of piperacillin plus 0.5 g of tazobactam.

OR

» meropenem: 0.5 to 1 g intravenously every 8 hours

OR

» ceftriaxone: 2 g intravenously every 24 hours

OR

» ciprofloxacin: 400 mg intravenously every 8-12 hours

» Continue empirical antibiotics started in the initial phase (see *Suspected necrotising fasciitis*) until culture results are known.

» Once culture results are available, tailor the patient's antibiotics to target the causative organism. Always consult an infectious disease or microbiology specialist (this is particularly important if the patient has multi-drug resistant necrotising fasciitis or is allergic to penicillin) and consider local epidemiological patterns.

- Necrotising fasciitis is **classified according to the underlying pathogen** as type I or II.[5]
- Type I infection involves aerobic and anaerobic organisms.[3] It is usually seen in older patients or in those with underlying illnesses.[8]

» Suitable antibiotic regimens include piperacillin/tazobactam, a carbapenem (e.g., meropenem), a cephalosporin (e.g., ceftriaxone), or a fluoroquinolone such as ciprofloxacin (if the patient is allergic to penicillin).

» Stop antibiotic cover for MRSA and group A streptococcus if these are ruled out.

» Continue antibiotics until further debridement is no longer necessary, the patient has clinically

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improved, and fever has resolved for 48 to 72 hours.[3] [67]

Drug safety alert: Fluoroquinolones

Fluoroquinolones have been associated with serious, disabling, and potentially irreversible adverse effects, including tendonitis, tendon rupture, arthralgia, neuropathies, and other musculoskeletal or nervous system effects.[64] Warnings have also been issued about the increased risk of aortic dissection, significant hypoglycaemia, and mental health adverse effects in patients taking fluoroquinolones.[65] [66]

consider **MRSA antibiotic cover**

Treatment recommended for SOME patients in selected patient group

Primary options

» vancomycin: 15-20 mg/kg intravenously every 8-12 hours, maximum 2000 mg/dose Adjust dose according to serum vancomycin level. A loading dose of 25-30 mg/kg may be considered in seriously ill patients.

OR

» linezolid: 600 mg intravenously every 12 hours

OR

» tedizolid phosphate: 200 mg intravenously every 24 hours

OR

» daptomycin: 4-6 mg/kg intravenously every 24 hours

» Continue vancomycin, linezolid, tedizolid, or daptomycin if MRSA is confirmed.

» Continue antibiotics until further debridement is no longer necessary, the patient has clinically improved, and fever has resolved for 48 to 72 hours.[3] [67]

consider **group A streptococcus antibiotic cover**

Treatment recommended for SOME patients in selected patient group

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Primary options

» clindamycin: 600-2700 mg/day intravenously given in 2-4 divided doses, may increase to 1200 mg every 6 hours in life-threatening infections

» Continue an antibiotic that inhibits toxin production until group A streptococcus involvement is excluded.[3] Evidence for clindamycin is strongest.[2] [37]

» Continue antibiotics until further debridement is no longer necessary, the patient has clinically improved, and fever has resolved for 48 to 72 hours.[3] [67]

type II necrotising fasciitis due to group A streptococcus

1st **surgical re-exploration ± debridement**

» Consider the need for additional debridement (following the initial debridement), based on culture results from subcutaneous tissue or blood, the patient's clinical condition, and discussion with the multidisciplinary team.[2] [3] [4] [53]

- Surgical re-exploration to assess the need for further debridement should be performed at least every 12 to 24 hours after the initial debridement.[3] However, re-exploration may be needed sooner in some patients; inform the surgical team urgently if the patient has clinical signs of worsening infection (local or systemic), or worsening laboratory markers (particularly white blood cell count).[3]
- Re-exploration should be repeated until the patient has no necrotic tissue remaining.[3] [4]

plus **supportive care**

Treatment recommended for ALL patients in selected patient group

Primary options

» morphine sulfate: 5-10 mg orally (immediate-release)/subcutaneously/intravenously/intramuscularly every 4 hours initially, adjust dose according to response

» Get an early review from the **critical care** team. Some patients will need critical care

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support depending on the degree of surgical resection and their physiological state.

» Give intensive haemodynamic support with intravenous fluids, and vasoactive drugs if needed.[2] [3] [4]

- Check local protocols for specific recommendations on fluid choice. Evidence from critically ill patients in general (not specifically people with necrotising fasciitis) suggests that there is no difference in benefit between normal saline and a balanced crystalloid (such as Hartmann's solution [also known as Ringer's lactate] or Plasma-Lyte®), and therefore either choice of fluid is reasonable.[58] [59]

Practical tip

Be aware that large volumes of normal saline as the sole fluid for resuscitation may lead to hyperchloremic acidosis. Also note that use of lactate-containing fluid in a patient with impaired liver metabolism may lead to a spuriously elevated lactate level, so results need to be interpreted with other markers of volume status.

Evidence: Choice of fluids

Evidence from two large randomised controlled trials (RCTs) suggests there is no difference between normal saline and a balanced crystalloid in mortality at 90 days, and therefore either option is a reasonable choice for the resuscitation of critically ill patients.

There has been extensive debate over the choice between normal saline (an unbalanced crystalloid) versus a balanced crystalloid (such as Hartmann's solution [also known as Ringer's lactate] or Plasma-Lyte®). Evidence from critically ill patients points to no benefits from using a balanced crystalloid in preference to normal saline. Clinical practice varies widely, so you should check local protocols.

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intensive care unit (ICU) patients with a balanced crystalloid solution (Plasma-Lyte) versus normal saline: the Plasma-Lyte 148 versus Saline (PLUS) trial (53 ICUs in Australia and New Zealand; N=5037) and the Balanced Solutions in Intensive Care Study (BaSICS) trial (75 ICUs in Brazil; N=11,052).[58] [59]

- In the PLUS study 45.2% of patients were admitted to ICU directly from surgery (emergency or elective), 42.3% had sepsis and 79.0% were receiving mechanical ventilation at the time of randomisation.
- In BaSICS almost half the patients (48.4%) were admitted to ICU after elective surgery and around 68% had some form of fluid resuscitation before being randomised.
- Both found no difference in 90-day mortality overall or in prespecified subgroups for patients with acute kidney injury (AKI), sepsis, or post-surgery. They also found no difference in the risk of AKI.
- In BaSICS, for patients with traumatic brain injury, there was a small decrease in 90-day mortality with normal saline - however, the overall number of patients was small (<5% of total included in the study) so there is some uncertainty about this result. Patients with traumatic brain injury were excluded from PLUS as the authors felt these patients should be receiving saline or a solution of similar tonicity.

- One meta-analysis of 13 RCTs (including PLUS and BaSICS) confirmed no overall difference,

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although the authors did highlight a non-significant trend towards a benefit of balanced solutions for risk of death.[60]

- Previous evidence has been mixed.
    - One 2015 double-blind, cluster-randomised, double-crossover trial conducted in four ICUs in New Zealand (N=2278), the 0.9% Saline vs. Plasma-Lyte for ICU fluid Therapy (SPLIT) trial, found no difference for in-hospital mortality, AKI, or use of renal-replacement therapy.[61]
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    - There was no difference in in-hospital mortality (OR 0.91, 95% CI 0.83 to 1.01; high-quality evidence as assessed by GRADE), AKI (OR 0.92, 95%

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CI 0.84 to 1.00; GRADE low), or organ system dysfunction (OR 0.80, 95% CI 0.40 to 1.61; GRADE very low).

- Check your local protocols for choice and dose of vasoactive drugs.
- Ensure the patient has adequate analgesia.[3] Morphine is used in practice.

plus **pathogen-targeted antibiotic therapy**

Treatment recommended for ALL patients in selected patient group

Primary options

» clindamycin: 600-2700 mg/day intravenously given in 2-4 divided doses, may increase to 1200 mg every 6 hours in life-threatening infections
-and-
» benzylpenicillin sodium: 0.6 to 1.2 g intravenously every 6 hours, may increase dose in serious infections, maximum 14.4 g/day

OR

» vancomycin: 15-20 mg/kg intravenously every 8-12 hours, maximum 2000 mg/dose
Adjust dose according to serum vancomycin level. A loading dose of 25-30 mg/kg may be considered in seriously ill patients.

» Continue empirical antibiotics started in the initial phase (see *Suspected necrotising fasciitis*) until culture results are known.

» Once culture results are available, tailor the patient's antibiotics to target the causative organism. Always consult an infectious disease or microbiology specialist (this is particularly important if the patient has multi-drug resistant necrotising fasciitis or is allergic to penicillin) and consider local epidemiological patterns.

- Necrotising fasciitis is **classified according to the underlying pathogen** as type I or II.[5]

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- Type II infection is most commonly due to group A streptococcus, but can also be due to *Staphylococcus aureus* .[3]

» Clindamycin plus a penicillin is recommended for type II infection due to group A streptococcus.[2]

- If the patient has a penicillin allergy, vancomycin monotherapy may be used.

» Continue antibiotics until further debridement is no longer necessary, the patient has clinically improved, and fever has resolved for 48 to 72 hours.[3] [67]

consider **intravenous immunoglobulin**

Treatment recommended for SOME patients in selected patient group

Primary options

» normal immunoglobulin human: consult specialist for guidance on dose

» If the patient develops streptococcal toxic shock syndrome (TSS), seek urgent advice from a senior colleague. Consider intravenous immunoglobulin (IVIG) as an adjunctive therapy for these patients.[3] See Toxic shock syndrome .

» IVIG may also have a role in managing patients with streptococcal infection without TSS.[3]

type II necrotising fasciitis due to Staphylococcus aureus

1st **surgical re-exploration ± debridement**

» Consider the need for additional debridement (following the initial debridement), based on culture results from subcutaneous tissue or blood, the patient's clinical condition, and discussion with the multidisciplinary team.[2] [3] [4] [53]

- Surgical re-exploration to assess the need for further debridement should be performed at least every 12 to 24 hours after the initial debridement.[3] However, re-exploration may be needed sooner in some patients; inform the surgical team urgently if the patient has clinical signs of worsening infection (local or systemic), or worsening laboratory markers (particularly white blood cell count).[3]

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- Re-exploration should be repeated until the patient has no necrotic tissue remaining.[3] [4]

plus **supportive care**

Treatment recommended for ALL patients in selected patient group

Primary options

» morphine sulfate: 5-10 mg orally (immediate-release)/subcutaneously/intravenously/intramuscularly every 4 hours initially, adjust dose according to response

» Get an early review from the **critical care** team. Some patients will need critical care support depending on the degree of surgical resection and their physiological state.

» Give intensive haemodynamic support with intravenous fluids, and vasoactive drugs if needed.[2] [3]

- Check local protocols for specific recommendations on fluid choice. Evidence from critically ill patients in general (not specifically people with necrotising fasciitis) suggests that there is no difference in benefit between normal saline and a balanced crystalloid (such as Hartmann's solution [also known as Ringer's lactate] or Plasma-Lyte®), and therefore either choice of fluid is reasonable.[58] [59]

Practical tip

Be aware that large volumes of normal saline as the sole fluid for resuscitation may lead to hyperchloremic acidosis. Also note that use of lactate-containing fluid in a patient with impaired liver metabolism may lead to a spuriously elevated lactate level, so results need to be interpreted with other markers of volume status.

Evidence: Choice of fluids

Evidence from two large randomised controlled trials (RCTs) suggests there is no difference between normal saline and a balanced crystalloid in mortality at 90 days, and therefore either option is a

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There has been extensive debate over the choice between normal saline (an unbalanced crystalloid) versus a balanced crystalloid (such as Hartmann's solution [also known as Ringer's lactate] or Plasma-Lyte®). Evidence from critically ill patients points to no benefits from using a balanced crystalloid in preference to normal saline. Clinical practice varies widely, so you should check local protocols.

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- Check your local protocols for choice and dose of vasoactive drugs.
- Ensure the patient has adequate analgesia.[3] Morphine is used in practice.

plus **pathogen-targeted antibiotic therapy**

Treatment recommended for ALL patients in selected patient group

Primary options

MRSA

» vancomycin: 15-20 mg/kg intravenously every 8-12 hours, maximum 2000 mg/dose Adjust dose according to serum vancomycin level. A loading dose of 25-30 mg/kg may be considered in seriously ill patients.

OR

MRSA

» linezolid: 600 mg intravenously every 12 hours

OR

MRSA

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» tedizolid phosphate: 200 mg intravenously every 24 hours

OR

MRSA

» daptomycin: 4-6 mg/kg intravenously every 24 hours

OR

MSSA

» flucloxacillin: 1-2 g intravenously every 6 hours

OR

MSSA

» cefazolin: 1-2 g/day intravenously given in 2-3 divided doses, may increase to 6 g/day given in 3-4 divided doses in severe infections

Secondary options

MRSA

» ceftaroline: 600 mg intravenously every 12 hours
May increase to 600 mg every 8 hours if infection is confirmed or suspected to be caused by *S aureus* with a minimum inhibitory concentration (MIC) of 2 mg/L or 4 mg/L to ceftaroline.

OR

MRSA

» dalbavancin: 1500 mg intravenously as a single dose; or 1000 mg intravenously as a single dose, followed by 500 mg after 1 week

OR

MRSA

» tigecycline: 100 mg intravenously initially, followed by 50 mg every 12 hours

» Continue empirical antibiotics started in the initial phase (see *Suspected necrotising fasciitis*) until culture results are known.

» Once culture results are available, tailor the patient's antibiotics to target the causative organism. Always consult an infectious disease or microbiology specialist (this is particularly important if the patient has multi-drug resistant

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necrotising fasciitis or is allergic to penicillin) and consider local epidemiological patterns.

- Necrotising fasciitis is **classified according to the underlying pathogen** as type I or II.[5]
- Type II infection is most commonly due to group A streptococcus, but can also be due to *Staphylococcus aureus* .[3]

» Use antibiotics that are active against MRSA until cultures confirm susceptibilities; options include vancomycin, linezolid, tedizolid, or daptomycin. Ceftaroline or dalbavancin are also reasonable choices.[2] [3] In practice, tigecycline may also be used.

» Flucloxacillin or cefazolin may be used if methicillin susceptibility is confirmed.[2]

» Continue antibiotics until further debridement is no longer necessary, the patient has clinically improved, and fever has resolved for 48 to 72 hours.[3] [67]

type II necrotising fasciitis due to Vibrio vulnificus

1st **surgical re-exploration ± debridement**

» C onsider the need for additional debridement (following the initial debridement), based on culture results from subcutaneous tissue or blood, the patient's clinical condition, and discussion with the multidisciplinary team.[2] [3] [4] [53]

- Surgical re-exploration to assess the need for further debridement should be performed at least every 12 to 24 hours after the initial debridement.[3] However, re-exploration may be needed sooner in some patients; inform the surgical team urgently if the patient has clinical signs of worsening infection (local or systemic), or worsening laboratory markers (particularly white blood cell count).[3]
- Re-exploration should be repeated until the patient has no necrotic tissue remaining.[3] [4]

plus **supportive care**

Treatment recommended for ALL patients in selected patient group

Primary options

Acute

» morphine sulfate: 5-10 mg orally (immediate-release)/subcutaneously/intravenously/intramuscularly every 4 hours initially, adjust dose according to response

» Get an early review from the **critical care** team. Some patients will need critical care support depending on the degree of surgical resection and their physiological state.

» Give intensive haemodynamic support with intravenous fluids, and vasoactive drugs if needed.[2] [3] [4]

- Check local protocols for specific recommendations on fluid choice. Evidence from critically ill patients in general (not specifically people with necrotising fasciitis) suggests that there is no difference in benefit between normal saline and a balanced crystalloid (such as Hartmann's solution [also known as Ringer's lactate] or Plasma-Lyte®), and therefore either choice of fluid is reasonable.[58] [59]

Practical tip

Be aware that large volumes of normal saline as the sole fluid for resuscitation may lead to hyperchloremic acidosis. Also note that use of lactate-containing fluid in a patient with impaired liver metabolism may lead to a spuriously elevated lactate level, so results need to be interpreted with other markers of volume status.

Evidence: Choice of fluids

Evidence from two large randomised controlled trials (RCTs) suggests there is no difference between normal saline and a balanced crystalloid in mortality at 90 days, and therefore either option is a reasonable choice for the resuscitation of critically ill patients.

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  - In the PLUS study 45.2% of patients were admitted to ICU directly from surgery (emergency or elective), 42.3% had sepsis and 79.0% were receiving mechanical ventilation at the time of randomisation.
  - In BaSICS almost half the patients (48.4%) were admitted to ICU after elective surgery and around 68% had some form of fluid resuscitation before being randomised.
  - Both found no difference in 90-day mortality overall or in prespecified subgroups for patients with acute kidney injury (AKI), sepsis, or post-surgery. They also found no difference in the risk of AKI.
  - In BaSICS, for patients with traumatic brain injury, there was a small decrease in 90-day mortality with normal saline - however, the overall number of patients was small (<5% of total included in the study) so there is some uncertainty about this result. Patients with traumatic brain injury were excluded from PLUS as the authors felt these patients should be receiving

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saline or a solution of similar tonicity.

- One meta-analysis of 13 RCTs (including PLUS and BaSICS) confirmed no overall difference, although the authors did highlight a non-significant trend towards a benefit of balanced solutions for risk of death.[60]
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    - One 2015 double-blind, cluster-randomised, double-crossover trial conducted in four ICUs in New Zealand (N=2278), the 0.9% Saline vs. Plasma-Lyte for ICU fluid Therapy (SPLIT) trial, found no difference for in-hospital mortality, AKI, or use of renal-replacement therapy.[61]
    - However, one 2018 US multicentre unblinded cluster-randomised trial - the isotonic Solutions and Major Adverse Renal events Trial (SMART), among 15,802 critically ill adults receiving ICU care - found possible small benefits from balanced crystalloid (Ringer's lactate or Plasma-Lyte) compared with normal saline. The 30-day outcomes showed a non-significant reduced mortality in the balanced crystalloid group versus the normal saline group (10.3% vs. 11.1%; odds ratio [OR] 0.90, 95% CI 0.80 to 1.01) and a major adverse kidney event rate of 14.3% versus 15.4% respectively (OR 0.91, 95% CI 0.84 to 0.99).[62]

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- Check your local protocols for choice and dose of vasoactive drugs.
- Ensure the patient has adequate analgesia.[3] Morphine is used in practice.

plus **pathogen-targeted antibiotic therapy**

Treatment recommended for ALL patients in selected patient group

Primary options

» doxycycline: 100 mg intravenously every 12 hours
-and-
» ceftriaxone: 2 g intravenously every 24 hours

» Continue empirical antibiotics started in the initial phase (see *Suspected necrotising fasciitis*) until culture results are known.

» Once culture results are available, tailor the patient's antibiotics to target the causative organism. Always consult an infectious disease or microbiology specialist (this is particularly important if the patient has multi-drug resistant necrotising fasciitis or is allergic to penicillin) and consider local epidemiological patterns.

- Type II infection with *Vibrio vulnificus* is associated with predisposing risk factors including hepatic disease, diabetes mellitus, chronic renal insufficiency, and adrenal insufficiency.[5] [9]

» Doxycycline plus a cephalosporin (e.g., ceftriaxone) is recommended for the management of necrotising fasciitis due to *V vulnificus*.[2]

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» Continue antibiotics until further debridement is no longer necessary, the patient has clinically improved, and fever has resolved for 48 to 72 hours.[3] [67]

type II necrotising fasciitis due to Aeromonas hydrophila

1st **surgical re-exploration ± debridement**

» Consider the need for additional debridement (following the initial debridement), based on culture results from subcutaneous tissue or blood, the patient's clinical condition, and discussion with the multidisciplinary team.[2] [3] [4] [53]

- Surgical re-exploration to assess the need for further debridement should be performed at least every 12 to 24 hours after the initial debridement.[3] However, re-exploration may be needed sooner in some patients; inform the surgical team urgently if the patient has clinical signs of worsening infection (local or systemic), or worsening laboratory markers (particularly white blood cell count).[3]
- Re-exploration should be repeated until the patient has no necrotic tissue remaining.[3] [4]

plus **supportive care**

Treatment recommended for ALL patients in selected patient group

Primary options

» morphine sulfate: 5-10 mg orally (immediate-release)/subcutaneously/intravenously/intramuscularly every 4 hours initially, adjust dose according to response

» Get an early review from the **critical care** team. Some patients will need critical care support depending on the degree of surgical resection and their physiological state.

» Give intensive haemodynamic support with intravenous fluids, and vasoactive drugs if needed.[2] [3] [4]

- Check local protocols for specific recommendations on fluid choice. Evidence from critically ill patients in general (not specifically people with necrotising fasciitis) suggests that there is no difference in benefit between normal

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saline and a balanced crystalloid (such as Hartmann's solution [also known as Ringer's lactate] or Plasma-Lyte®), and therefore either choice of fluid is reasonable.[58] [59]

Practical tip

Be aware that large volumes of normal saline as the sole fluid for resuscitation may lead to hyperchloremic acidosis. Also note that use of lactate-containing fluid in a patient with impaired liver metabolism may lead to a spuriously elevated lactate level, so results need to be interpreted with other markers of volume status.

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- Check your local protocols for choice and dose of vasoactive drugs.

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- Ensure the patient has adequate analgesia.[3] Morphine is used in practice.

plus **pathogen-targeted antibiotic therapy**

Treatment recommended for ALL patients in selected patient group

Primary options

» doxycycline: 100 mg intravenously every 12 hours

--AND--

» ciprofloxacin: 400 mg intravenously every 8-12 hours

-or-

» ceftriaxone: 2 g intravenously every 24 hours

» Continue empirical antibiotics started in the initial phase (see *Suspected necrotising fasciitis*) until culture results are known.

» Tailor the patient's antibiotics according to the culture results, and discussion with an infectious disease or microbiology specialist (particularly if the patient has multi-drug resistant necrotising fasciitis or is allergic to penicillin) and consider local epidemiological patterns.

- Necrotising fasciitis is **classified according to the underlying pathogen** as type I or II.[5]
- Type II infection infection with *Aeromonas hydrophila* is more common in patients with immunosuppression, burns, and trauma in an aquatic setting.[5] [10]

» Doxycycline plus ciprofloxacin or ceftriaxone are recommended for the management of necrotising fasciitis due to *A hydrophila* .[2]

» Continue antibiotics until further debridement is no longer necessary, the patient has clinically improved, and fever has resolved for 48 to 72 hours.[3] [67]

type II necrotising fasciitis due to Clostridium

1st **surgical re-exploration ± debridement**

» Consider the need for additional debridement (following the initial debridement), based on culture results from subcutaneous tissue or blood, the patient's clinical condition, and

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discussion with the multidisciplinary team.[2] [3] [4] [53]

- Surgical re-exploration to assess the need for further debridement should be performed at least every 12 to 24 hours after the initial debridement.[3] However, re-exploration may be needed sooner in some patients; inform the surgical team urgently if the patient has clinical signs of worsening infection (local or systemic), or worsening laboratory markers (particularly white blood cell count).[3]
- Re-exploration should be repeated until the patient has no necrotic tissue remaining.[3] [4]

plus **supportive care**

Treatment recommended for ALL patients in selected patient group

Primary options

» morphine sulfate: 5-10 mg orally (immediate-release)/subcutaneously/intravenously/intramuscularly every 4 hours initially, adjust dose according to response

» Get an early review from the **critical care** team. Some patients will need critical care support depending on the degree of surgical resection and their physiological state.

» Give intensive haemodynamic support with intravenous fluids, and vasoactive drugs if needed.[2] [3] [4]

- Check local protocols for specific recommendations on fluid choice. Evidence from critically ill patients in general (not specifically people with necrotising fasciitis) suggests that there is no difference in benefit between normal saline and a balanced crystalloid (such as Hartmann's solution [also known as Ringer's lactate] or Plasma-Lyte®), and therefore either choice of fluid is reasonable.[58] [59]

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Be aware that large volumes of normal saline as the sole fluid for resuscitation may lead to hyperchloremic acidosis. Also note that use of lactate-containing fluid in a patient with impaired liver metabolism may lead to a spuriously elevated lactate level, so results need to be interpreted with other markers of volume status.

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- Check your local protocols for choice and dose of vasoactive drugs.
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plus **pathogen-targeted antibiotic therapy**

Treatment recommended for ALL patients in selected patient group

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Primary options

» clindamycin: 600-2700 mg/day intravenously given in 2-4 divided doses, may increase to 1200 mg every 6 hours in life-threatening infections
-and-
» benzylpenicillin sodium: 0.6 to 1.2 g intravenously every 6 hours, may increase dose in serious infections, maximum 14.4 g/day

» Continue empirical antibiotics started in the initial phase (see *Suspected necrotising fasciitis*) until culture results are known.

» Once culture results are available, tailor the patient's antibiotics to target the causative organism. Always consult an infectious disease or microbiology specialist (this is particularly important if the patient has multi-drug resistant necrotising fasciitis or is allergic to penicillin) and consider local epidemiological patterns.

- Necrotising fasciitis is **classified according to the underlying pathogen** as type I or II.[5]
- Type II infection with *Clostridium* can also cause gangrenous necrotising fasciitis.[5] See Gangrene .

fungal infection

1st **surgical re-exploration ± debridement**

» Consider the need for additional debridement (following the initial debridement), based on culture results from subcutaneous tissue or blood, the patient's clinical condition, and discussion with the multidisciplinary team.[2] [3] [4] [53]

- Surgical re-exploration to assess the need for further debridement should be performed at least every 12 to 24 hours after the initial debridement.[3] However, re-exploration may be needed sooner in some patients; inform the surgical team urgently if the patient has clinical signs of worsening infection (local or systemic), or worsening laboratory markers (particularly white blood cell count).[3]
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Treatment recommended for ALL patients in selected patient group

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- Check your local protocols for choice and dose of vasoactive drugs.
- Ensure the patient has adequate analgesia.[3] Morphine is used in practice.

plus **discussion with infectious disease or microbiology specialist and antifungal therapy**

Treatment recommended for ALL patients in selected patient group

Primary options

» amphotericin B liposomal: consult specialist for guidance on dose

» Continue empirical antibiotics started in the initial phase (see *Suspected necrotising fasciitis*) until culture results are known.

» Seek advice from an infectious disease or microbiology specialist to determine further treatment, which should be tailored according to the culture results.

- Fungal infection is rare and is the result of infection with pathogens such as Mucorales and *Candida* species. Although necrotising mucormycosis predominantly affects immunocompromised people, it may also occur in immunocompetent individuals.[12] [13] [14]

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- Liposomal amphotericin-B is usually the primary treatment option.

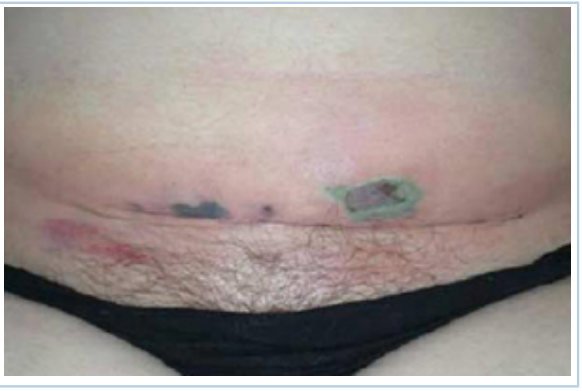
Ongoing

persistent cosmetic and functional defects after debridement

1st **reconstructive surgery**

» If functional and cosmetic disability results from extensive surgical debridement for necrotising fasciitis, reconstructive surgery may be required.[4] However, this should only be considered when the patient is stable and the infection has been completely eradicated.

»



Small areas of skin necrosis in a young woman with cellulitis and necrotizing fasciitis of her lower abdomen 5 days after a cesarean section
From: Hasham S, Matteucci P, Stanley PRW, et al. Necrotising fasciitis. BMJ. 2005 Apr 9;330(7495):830-3

Emerging

Hyperbaric oxygen (HBO) therapy

HBO therapy has been advocated by some physicians based upon its beneficial effects on cutaneous wound healing, but there is a lack of prospective controlled studies to demonstrate its efficacy.[1] [74] HBO therapy is not recommended in England for necrotising fasciitis.[1] [74] [75] It may be readily available and recommended for this indication in other regions; HBO therapy must never delay surgical debridement or appropriate antibiotic treatment.

Newer antibiotics

Antibiotics with activity against gram-positive organisms (including MRSA), such as the second-generation glycopeptide antibiotic oritavancin and the fluoroquinolone delafloxacin, may be considered for inclusion in an antimicrobial regimen for necrotising fasciitis, where available.[76] [77] [78] However, clinical data supporting their use are limited, the efficacy of these agents has not been rigorously demonstrated in necrotising fasciitis, and there are significant adverse effects associated with each of these agents. With the exception of use in uncommon and specific antibiotic resistance patterns in an isolated causative organism, there is thus far no compelling evidence to recommend their use in necrotising fasciitis.

Reltecimod

Reltecimod is an immune-modulating peptide derived from the T-cell receptor CD28, which targets the co-stimulatory pathway that induces pro-inflammatory cytokines. It has been shown to be of benefit in bacterial infections including necrotising fasciitis.[4] In one study in necrotising fasciitis, reltecimod was associated with faster resolution of organ dysfunction and hospital discharge; however, further studies are needed.[4] It is not currently licensed or available in the UK.

Primary prevention

Measures to prevent the development of necrotising fasciitis include:

- Prevention of trauma or breaking of skin integrity (that may constitute portals of entry for the infection)
- Treatment of cellulitis to prevent extension into subcutaneous tissue[2]
- Immunisation against varicella zoster virus (in adults and children). This may prevent necrotising fasciitis as a complication of skin breaks due to chickenpox or zoster.[33]

Secondary prevention

Infection control practices should be in place in the hospital to prevent patient to patient spread of group A streptococcus in all patients, including those with type II necrotising fasciitis.

Patient discussions

Advise the patient that necrotising fasciitis is a life-threatening infection and that surgical excision and drainage of infected tissue as necessary, combined with intravenous antibiotic therapy, is essential.

Recurrence of necrotising fasciitis is rare. However, significant functional and cosmetic morbidity may remain following initial surgical therapies, which may require subsequent reconstruction.

FOLLOW UP

Monitoring

Monitoring

While the patient is waiting for initial emergency debridement, monitor them for systemic toxicity (e.g., signs of end-organ damage), as well as local signs and symptoms of extension of the area of necrotising fasciitis.

Following the initial debridement, monitor the patient closely for decline in respiratory/haemodynamic function. Watch for the progression to toxic shock syndrome in patients with group A streptococcal infection. See Toxic shock syndrome .

Consider the need for additional debridement or alteration in antibiotic or antifungal therapy, based on culture results from subcutaneous tissue or blood, the patient's clinical condition, and discussion with the multidisciplinary team.[53]

- Surgical re-exploration to assess the need for further debridement should be performed at least every 12 to 24 hours after the initial debridement.[3]
- However, re-exploration may be needed sooner in some patients; inform the surgical team urgently if the patient has clinical signs of worsening infection (local or systemic), or worsening laboratory markers (particularly white blood cell count).[3]

Complications

Complications	Timeframe	Likelihood
mortality	**short term**	**high**
Mortality from necrotising fasciitis properly treated with surgery plus antibiotics has been estimated at between 10% and 40%. Mortality is higher in patients who develop shock and end-organ damage, approaching 50% to 70%.[2]		
skin loss and scarring	**long term**	**high**
Functional and cosmetic disability may result from extensive surgical debridement for necrotising fasciitis. Reconstructive surgery may be required.[4]		

Prognosis

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Recurrence of necrotising fasciitis is rare.[1] [2] However, significant functional and cosmetic morbidity may remain following initial surgical therapies, which may require subsequent reconstruction.

Diagnostic guidelines

United Kingdom

Necrotising fasciitis

Published by: Public Health England **Last published:** 2013

International

WSES/GAIS/WSIS/SIS-E/AAST global clinical pathways for patients with skin and soft tissue infections

Published by: World Society of Emergency Surgery; Global Alliance for Infections in Surgery; World Surgical Infection Society; Surgical Infection Society Europe; American Association for the Surgery of Trauma **Last published:** 2022

2018 WSES/SIS-E consensus conference: recommendations for the management of skin and soft-tissue infections

Published by: World Society of Emergency Surgery; Surgical Infection Society Europe **Last published:** 2018

North America

Practice guidelines for the diagnosis and management of skin and soft-tissue infections

Published by: Infectious Diseases Society of America **Last published:** 2014

Treatment guidelines

United Kingdom

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International

WSES/GAIS/WSIS/SIS-E/AAST global clinical pathways for patients with skin and soft tissue infections

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2018 WSES/SIS-E consensus conference: recommendations for the management of skin and soft-tissue infections

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North America

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Key articles

- Sartelli M, Guirao X, Hardcastle TC, et al. 2018 WSES/SIS-E consensus conference: recommendations for the management of skin and soft-tissue infections. World J Emerg Surg. 2018 Dec 14;13:58. Full text Abstract
- Public Health England. Necrotising fasciitis (NF). April 2013 [internet publication]. Full text

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Images

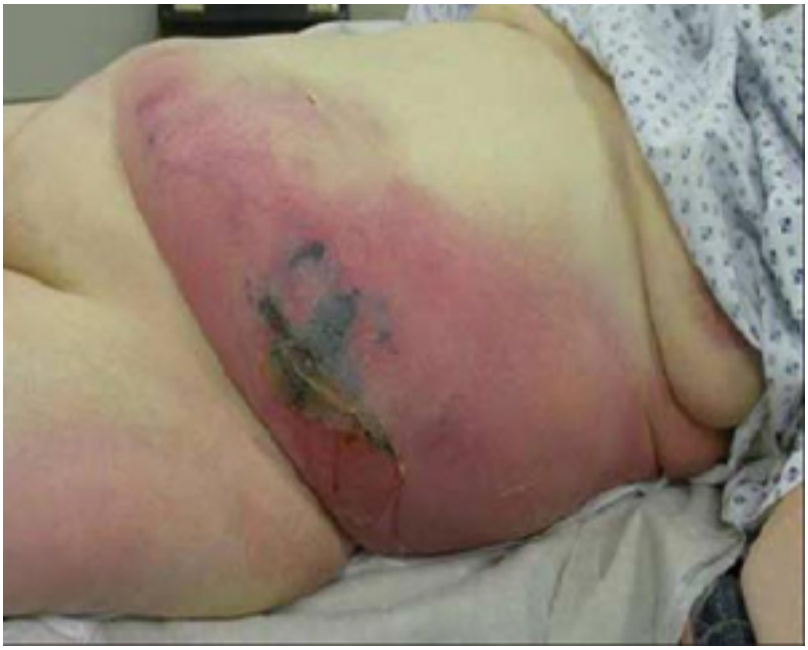

Figure 1: Late signs of necrotising fasciitis with extensive cellulitis, induration, skin necrosis, and formation of haemorrhagic bullae

From: Hasham S, Matteucci P, Stanley PRW, et al. Necrotising fasciitis. BMJ. 2005 Apr 9;330(7495):830-3

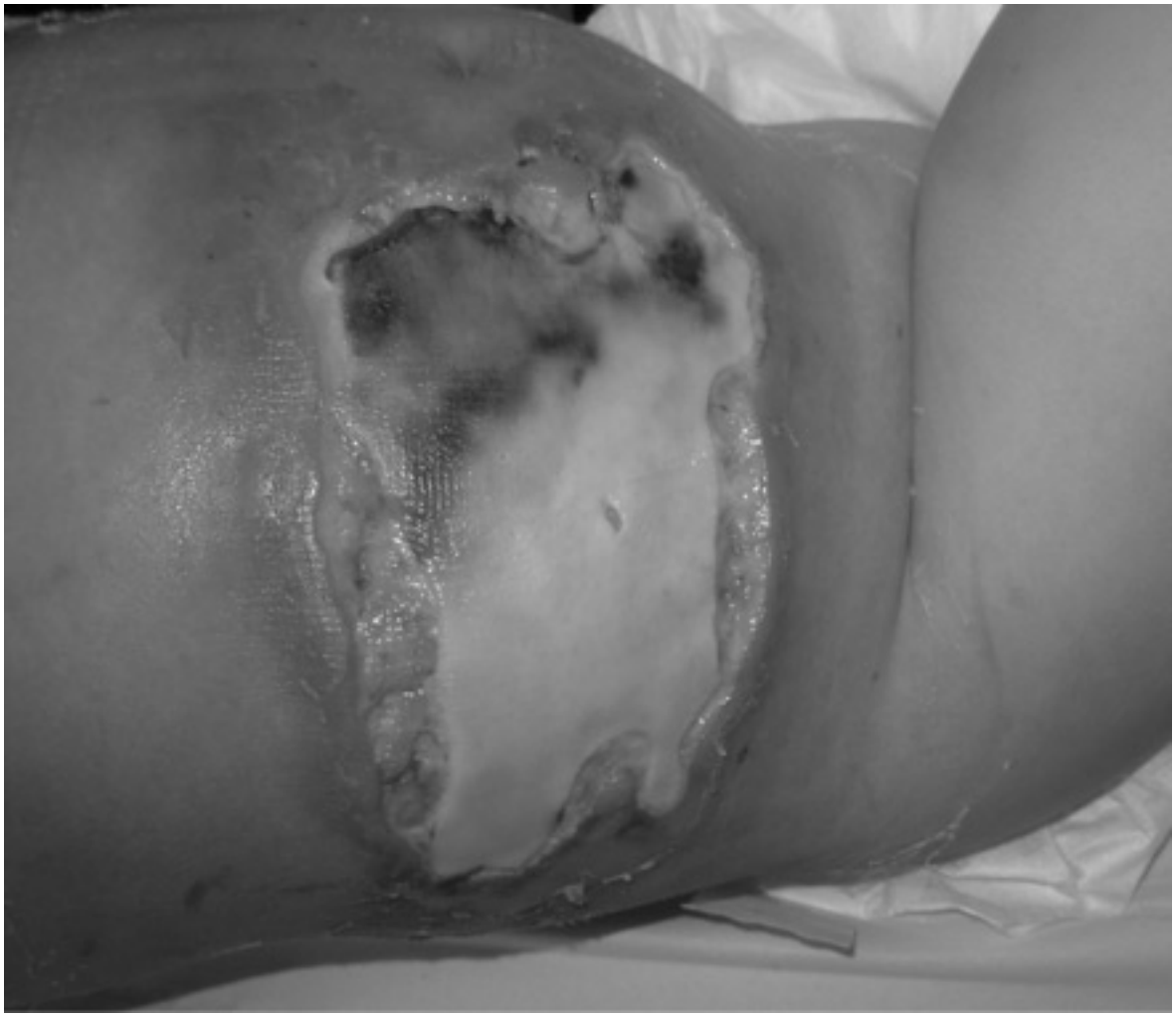

Figure 2: Necrotising fasciitis on the right abdomen of a 2-year old girl following varicella infection

From: de Benedictis FM, Osimani P. Necrotising fasciitis complicating varicella. BMJ Case Rep. 2009;2009:bcr2008141994

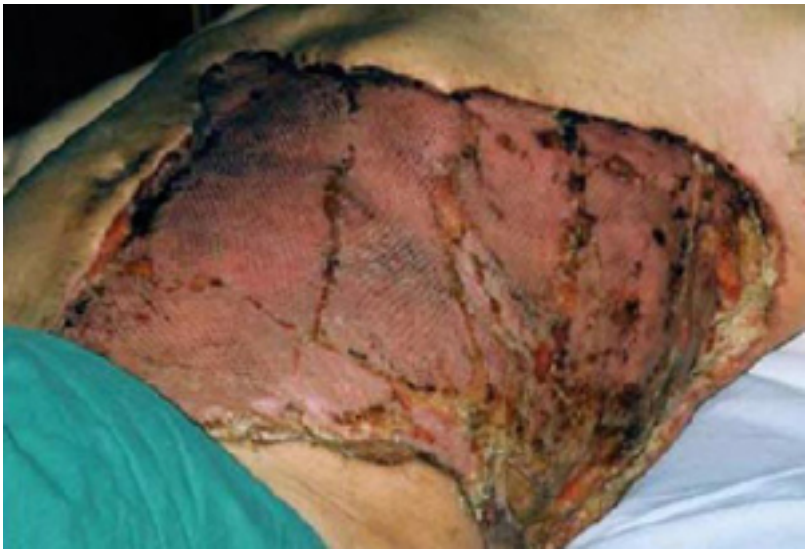

Figure 3: Split thickness skin grafting after surgical debridement

From: Hasham S, Matteucci P, Stanley PRW, et al. Necrotising fasciitis. BMJ. 2005 Apr 9;330(7495):830-3

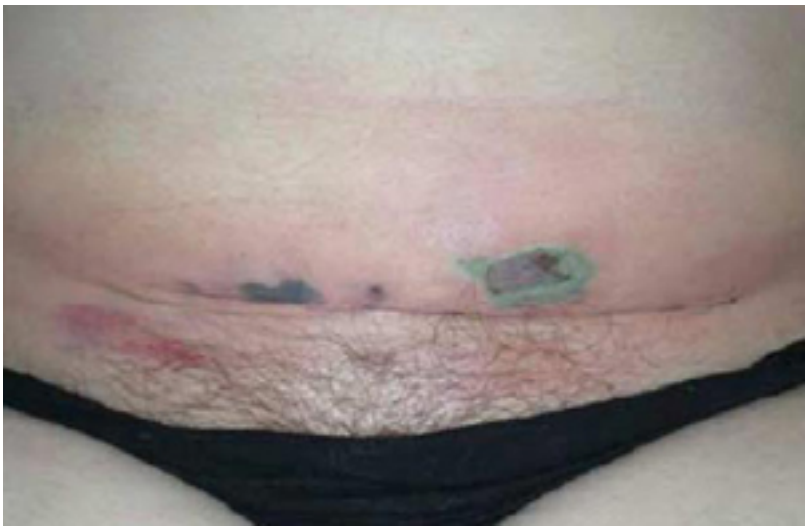

Figure 4: Small areas of skin necrosis in a young woman with cellulitis and necrotizing fasciitis of her lower abdomen 5 days after a cesarean section

From: Hasham S, Matteucci P, Stanley PRW, et al. Necrotising fasciitis. BMJ. 2005 Apr 9;330(7495):830-3

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