BMJ Best Practice

Necrotizing fasciitis

Straight to the point of care

Last updated: Jan 05, 2024

Table of Contents

Summary

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

Infection may be polymicrobial in etiology (type I) due to mixed anaerobic/facultative anaerobic organisms, or due to a single organism (type II), most commonly *Streptococcus pyogenes* , also called group A streptococcus.

Necrotizing fasciitis should be suspected in any patient with a rapidly progressing soft-tissue infection and any of the following: severe pain (disproportionate to the clinical findings) or anesthesia over the site of infection; edema and erythema (edema will typically extend beyond the erythema); systemic signs of infection. However, necrotizing fasciitis can be easily missed because the patient may present earlier in the disease process with nonspecific signs and symptoms.

No laboratory or imaging studies, alone or in combination, are sufficiently sensitive and specific to definitively diagnose or rule out necrotizing fasciitis.

An urgent surgical consultation should be obtained as soon as the diagnosis is suspected. Treatment should not be delayed while awaiting microbiologic and imaging investigations.

Definitive treatment is surgical debridement, repeated as necessary. Antibiotic therapy is crucial, but is considered adjunctive to surgical management. Empiric antibiotics should cover major bacterial etiologic agents, and group A streptococcal toxin production that can accompany type II necrotizing fasciitis.

Definition

Necrotizing fasciitis is a life-threatening subcutaneous soft-tissue infection progressively extending 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. Two main clinical forms exist. Type I necrotizing fasciitis is a polymicrobial infection with an anaerobe 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 necrotizing fasciitis is most commonly a monomicrobial infection with typically *Streptococcus pyogenes* (group A streptococci) or occasionally *Staphylococcus aureus* .[1][2] [3] [4]

Other infectious etiologies may rarely cause a monomicrobial necrotizing infection that may be associated with specific exposures or risk factors (e.g., freshwater exposure associated with *Aeromonas hydrophila* , saltwater exposure or consumption of raw oysters associated with *Vibrio vulnificus* , recent travel to [or living in] Taiwan, where *Klebsiella pneumoniae* is a common cause of monomicrobial infection).[6]

 © BMJ Publishing Group Ltd 2024. All rights reserved.

Epidemiology

Absolute data for the incidence and prevalence of necrotizing fasciitis are lacking and may vary by geographic location. The incidence is higher in adults compared to children (estimated at 0.4 per 100,000 per year versus 0.08 to 0.13 per 100,000 per year).[21] Type I (due to mixed anaerobic-facultative anaerobic infections) is more common than type II necrotizing fasciitis overall, whereas group A streptococcal type II necrotizing fasciitis is the most common in children.[21] [22]

US-based multisite surveillance data from 2021 shows that necrotizing fasciitis complicated about 4.5% of invasive group A streptococcal infections, with approximately 100 cases per year.[23] The overall prevalence, incidence, and epidemiology remain stable.

Etiology

Type I necrotizing fasciitis is a polymicrobial infection of subcutaneous tissue with 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 necrotizing fasciitis is a monomicrobial infection of subcutaneous tissue most commonly caused by *Streptococcus pyogenes* (group A streptococci) and occasionally *Staphylococcus aureus* . Panton-Valentine leukocidin (PVL)-positive *Staphylococcus aureus,* and MRSA are also potentially causative organisms.[1][2]

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

- *Aeromonas hydrophila* , associated with freshwater exposure[3] [9] [10]
- *Vibrio vulnificus* , from saltwater exposure or consumption of contaminated raw oysters[3] [9] [10]
- *Klebsiella pneumoniae* , in South East Asian countries, in particular Taiwan[11]
- *Clostridium* , can cause gangrenous necrotizing fasciitis (see Gangrene).
- *Pseudomonas aeruginosa* , *Enterobacterales* , or *Clostridium septicum* in neutropenic children.[21]

Very rarely necrotizing fasciitis is a monomicrobial infection caused by fungal pathogens such as mucormycosis.[4] Mucormycosis has been reported as a cause in immunocompromised and immunocompetent patients.[12] [13] [14] Few cases of candida necrotizing fasciitis have been reported following surgery.[24]

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][25] [26]

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 necrotizing fasciitis, such as fever, tachycardia, and hypotension, are primarily due to the action of bacterial toxins.[27] [28]

This PDF of the BMJ Best Practice topic is based on the web version that was last updated: Jan 05, 2024. BMJ Best Practice topics are regularly updated and the most recent version of the topics can be found on bestpractice.bmj.com . Use of this content is subject to our disclaimer (. Use of this content is subject to our) . © BMJ Publishing Group Ltd 2024. All rights reserved.

Classification

Clinical presentation

Necrotizing 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 necrotizing 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

Nonspecific or variable symptoms with an insidious onset.[7] Localized pain at the site of the skin lesion may be mild or absent.[7]

Causative organism

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

Type I

Polymicrobial infection with an anaerobe such as *Bacteroides* or *Peptostreptococcus* plus a facultative anaerobe such as certain Enterobacterales (*Escherichia coli* , *Enterobacter* , *Klebsiella* , *Proteus*) or non-group A streptococcus.[1][2] [3] [4] 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 such as Panton-Valentine leukocidin (PVL)-positive *Staphylococcus aureus* and MRSA.[1][2] [3] [4] Other infectious etiologies may rarely cause a monomicrobial necrotizing infection associated with specific exposures or risk factors:

- *Aeromonas hydrophila* , associated with freshwater exposure.[3] [9] [10]
- *Vibrio vulnificus* , from saltwater exposure or consumption of contaminated raw oysters.[3] [9] [10]
- *Klebsiella pneumoniae* , in South East Asian countries, in particular Taiwan.[11]
- *Clostridium* , can cause gangrenous necrotizing fasciitis. Usually follows severe penetrating trauma or crush injury with interruption of blood supply to the affected area. Can be difficult to differentiate clinically.[5] See Gangrene .

Very rarely monomicrobial infection is caused by fungal pathogens such as mucormycosis.[4] Mucormycosis has been reported as a cause in immunocompromised and immunocompetent patients.[12] [13] [14]

 © BMJ Publishing Group Ltd 2024. All rights reserved.

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.[3] Some references, including other publications from WSES, further subclassify monomicrobial gram-negative infections including *Aeromonas* and *Vibrio* infections as type III and fungal infections as type IV.[2][15]

Anatomical location

Fournier gangrene is type I necrotizing fasciitis of the scrotum or male perineum.[1][2] [3][4] [5] [16]

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

Cervicofacial necrotizing 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 necrotizing fasciitis in the context of surgical site infections or rare organisms.

Case history

Case history #1

A 35-year-old woman is admitted to the hospital because of pain and swelling of the right thigh. The patient has been in excellent health until the morning before admission, when she observed a pimple 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 99.5°F (37.5°C), pulse is 128 bpm, and respirations are 20 breaths/minute. BP is 85/60 mmHg. On physical examination, the patient appears ill 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 because of pain and reports pain on passive extension of the right ankle. The temperature soon rises to 101°F (38.4°C), and the BP drops to 70/40 mmHg. Hematocrit is 42, WBC count 5900/mm^3 (with 64% neutrophils, 19% band forms), serum creatinine 1.9 mg/dL, and BUN 22 mg/dL. Contrast-enhanced computed tomography shows a diffuse, nonenhancing, 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

Necrotizing fasciitis should be considered in a patient with cellulitis who also has systemic symptoms and signs such as hypotension, tachycardia, tachypnea, nausea, vomiting, or delirium. The area of cellulitis may be either severely and constantly painful (disproportionate to skin findings) or, conversely, anesthetic. Examination of the skin overlying the area of cellulitis may reveal underlying induration extending beyond the area of cellulitis, ecchymoses, vesicles, bullae, grayish discoloration, or edema extending beyond erythema.[3] Crepitus may be noted on exam. Rapid extension of cellulitis despite the use of appropriate antibiotics should also raise suspicion for a necrotizing process. About half of cases occur in the extremities, with the remainder concentrated in the perineum, trunk, and head and neck areas.[1][2] [3] [4] [16][19][20]

This PDF of the BMJ Best Practice topic is based on the web version that was last updated: Jan 05, 2024. BMJ Best Practice topics are regularly updated and the most recent version of the topics can be found on bestpractice.bmj.com . Use of this content is subject to our disclaimer (. Use of this content is subject to our) . © BMJ Publishing Group Ltd 2024. All rights reserved.

Atypical presentations include necrotizing fasciitis that occurs without an obvious overlying skin lesion (approximately 20% of cases), or that arise from a Bartholin gland or perianal abscess. Fournier gangrene is a form of type I necrotizing fasciitis that occurs in the perineum.[1][5][16]

This PDF of the BMJ Best Practice topic is based on the web version that was last updated: Jan 05, 2024.
BMJ Best Practice topics are regularly updated and the most recent version of the topics
can be found on bestpractice.bmj.com . Use of this content is subject to our disclaimer (.
Use of this content is subject to our) . © BMJ Publishing Group Ltd 2024. All rights reserved.

Approach

Early recognition is important as disease progression is typically rapid. Necrotizing fasciitis is a life-threatening and time-critical surgical emergency.[2] [4] Sepsis and multi-organ failure may be present.[2] [34] Prompt referral to the surgical team and discussion with critical care is essential. Despite the need for laboratory testing including microbiologic testing and imaging when possible, necrotizing fasciitis remains primarily a clinical diagnosis with signs and symptoms that change rapidly over time.[2] [4]

History

The speed of symptom progression should be determined. Inquire for specific risk factors such as:

- Preceding skin lesions or breakdown
- Trauma, surgery
    - Necrotizing 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

It is important to remember that the inciting insult may be minor (e.g., an insect bite) or not recalled by the patient.[1] [25] 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 empiric antibiotic selection should be broad and not guided solely by historical exposures.[9] [15] [25]

Other possible symptoms or signs of necrotizing fasciitis in a patient with cellulitis include lightheadedness, palpitations, nausea or vomiting, or delirium.

Physical exam

The diagnosis should be considered in a patient with cellulitis and clinical warning signs such as a positive quick Sepsis-Related Organ Failure Assessment (qSOFA; not specifically validated for necrotizing fasciitis). Altered mental status (Glasgow Coma Scale ≤15), systolic hypotension (systolic bp ≤100 mmHg), and elevated respiratory rate (≥ 22 bpm) suggest that a patient with cellulitis may have a necrotizing process requiring expedited surgical evaluation.[2] However, the patient may present with nonspecific or nonlocalized symptoms (e.g., acutely unwell with a normal temperature) or there may be severe signs with evidence of multi-organ dysfunction and shock.[2]

Anesthesia or severe pain over the site of cellulitis may be clues to an underlying subcutaneous infection. [1][16] [19] [20] [35] The pain experienced with necrotizing fasciitis may be disproportionate to the visible skin changes. It should be noted that patients with necrotizing fasciitis can present with normal overlying skin, and that skin changes overlying group A streptococcal necrotizing fasciitis are a late sign.

Exam of the skin overlying the area of cellulitis may reveal crepitus, vesicles, bullae, grayish discoloration, or edema extending beyond erythema.[3] Subtle skin changes such as leakage of fluid and edema precede the overt skin changes of blistering and redness.

This PDF of the BMJ Best Practice topic is based on the web version that was last updated: Jan 05, 2024. BMJ Best Practice topics are regularly updated and the most recent version of the topics can be found on bestpractice.bmj.com . Use of this content is subject to our disclaimer (. Use of this content is subject to our) . © BMJ Publishing Group Ltd 2024. All rights reserved.

About half of cases occur in the extremities, with the remainder concentrated in the perineum, trunk, and head and neck areas.[1][2] [3] [4] [16] [19] [20] The most common site of group A streptococcal necrotizing fasciitis is the thigh and necrotizing fasciitis of a limb, especially the arm, is more likely to be due to group A streptococci than a polymicrobial infection. Some cases of necrotizing fasciitis may have associated myositis due to contiguous spread. This is more common in group A streptococcal than polymicrobial infections.

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

Laboratory evaluation

All patients admitted with suspected necrotizing fasciitis should have a complete blood count with white cell differential, blood urea nitrogen (BUN), electrolytes, creatinine, and C-reactive protein (CRP) measured urgently. Some of these biomarkers are used for predictive scores including the Laboratory Risk Indicator for Necrotizing Fasciitis (LRINEC). Raised lactate and procalcitonin may also be associated with increased likelihood of morbidity and mortality.[15] Arterial blood gases may be obtained if there is concern for respiratory compromise.

Necrotizing fasciitis is frequently associated with a range of nonspecific laboratory abnormalities including:

- Abnormally high or low white blood cell (WBC) count with or without a left shift (elevated percentage of polymorphonuclear leukocytes and/or bands). A low WBC count may be a sign of severe sepsis
- Elevated BUN and creatinine due to intracellular volume depletion
- Decreased serum sodium
- Elevated CRP
- Elevated serum creatine kinase
- Elevated plasma lactate.

Blood cultures should be obtained before starting antibiotics, and may help identify the causative organism.[15] Gram stain and cultures from needle aspiration or deep tissue cultures obtained during surgical debridement or exploration may also yield a bacteriologic diagnosis.[5] [15] Superficial skin swabs should be avoided.[15]

Molecular testing and next generation sequencing when available can help identify additional pathogens.[15]

Imaging

Imaging studies should not delay surgical intervention when diagnosis is clinically suspected.[2] [3] [5][15]

In clinically stable patients, radiography may provide supportive evidence for a necrotizing process.

Plain radiography is frequently normal during the early stages; subcutaneous gas may be present as the disease progresses. The diagnosis should be strongly suspected if soft-tissue gas is visualized on radiologic examination, which may also demonstrate abnormalities in the involved soft tissue.[1][2] [16] [36] However, soft-tissue gas is a late sign, and plain radiography has poor sensitivity for detecting signs of necrotizing infection.[15]

 © BMJ Publishing Group Ltd 2024. All rights reserved.

Ultrasound may help to differentiate simple cellulitis from necrotizing fasciitis and has the advantage that it can be rapidly performed at bedside.[2] 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 specificity of 93%.[37]

Computed tomography (CT) and magnetic resonance imaging (MRI) offer higher sensitivity.[3] [36] CT is indicated in abdominoperineal and cervicofacial infections to show the portal of entry of infection and to guide surgical intervention; for limb or peripheral necrotizing soft-tissue CT is of limited value.[15] [36] MRI is more sensitive for assessing necrotizing soft-tissue infections of the limbs, and may show thickening of the fascia, deep fascial fluid or edema. However, these signs are not specific to necrotizing infection and may be seen in other soft-tissue infections such as cellulitis, and MRI may be difficult to organize in an emergency and is not recommended as the first-line imaging technique.[2][3][15][38]

In a meta-analysis, CT had sensitivity of 88.5% and specificity of 93.3%, while plain radiography had sensitivity of 48.9% and specificity of 94.0% for diagnosing necrotizing soft tissue infections.[39]

Early frozen-section soft-tissue biopsy can provide a definitive diagnosis and may be used if the diagnosis is unclear clinically or radiologically.[2] 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.[2]

Diagnostic criteria

The use of severity assessment scores may be helpful in identifying patients at high risk of systemic sepsis and, therefore, those who need urgent critical care and surgical assessment.[2]

Necrotizing fasciitis should be considered in a patient with cellulitis and clinical warning signs such as a positive quick Sepsis-Related Organ Failure Assessment (qSOFA; for use in settings other than the intensive care unit). However, no studies have specifically addressed the use of qSOFA, or the Sequential Organ Failure Assessment (SOFA; for use in the intensive care unit), in necrotizing fasciitis.

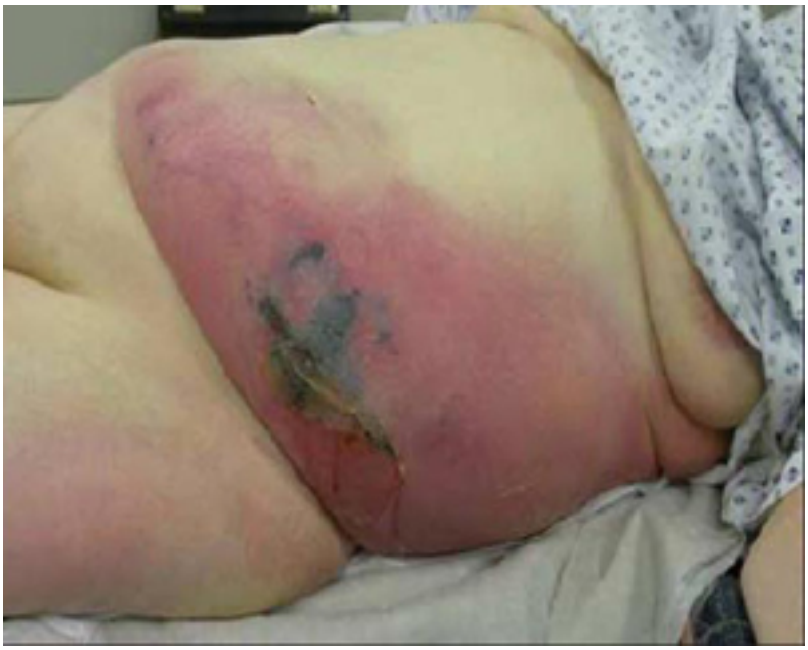
The LRINEC scoring system, based upon laboratory abnormalities (CRP, hemoglobin sodium, creatinine, glucose), has been developed in an attempt to assist with early discrimination of necrotizing fasciitis from less severe skin and soft tissue infections.[3] [40] In a 2019 meta-analysis, a LRINEC 6 was associated with a sensitivity of 68.2% (95% CI 51.4% to 81.3%) and specificity of 84.8% (95% CI 75.8% to 90.9%) for diagnosis of necrotizing soft tissue infections.[39] While validation studies have failed to demonstrate sufficient sensitivity or specificity to either diagnose or exclude necrotizing fasciitis, a higher LRINEC score on presentation has been associated with a higher risk of mortality in necrotizing fasciitis.[39] [41] [42]

Consultation

Involving a range of experts with a multidisciplinary approach may improve management and prognosis, along with a high index of suspicion for the condition.[15] Senior decision makers should be involved early when necrotizing fasciitis is suspected, given that there is a low incidence and poor sensitivity of early clinical signs and in the absence of optimal noninvasive diagnostic tools.[15] An urgent surgical consultation for inspection, exploration, and debridement of infected tissue should be obtained as soon as the diagnosis is suspected.[3] Additional early consultations include involving an intensive care specialist.[15] Definitive bacteriologic diagnosis is best made from tissue specimens obtained from surgical debridement.[5] Staining of clinically affected tissue may provide an early indication of

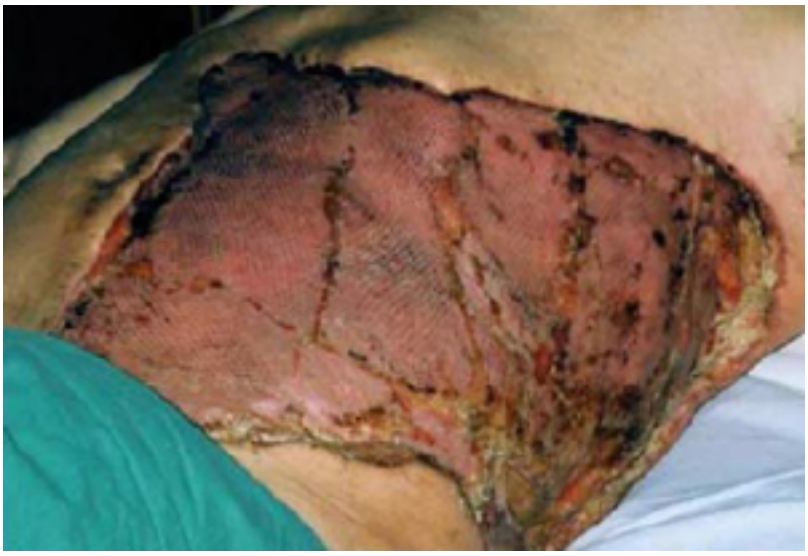
This PDF of the BMJ Best Practice topic is based on the web version that was last updated: Jan 05, 2024. BMJ Best Practice topics are regularly updated and the most recent version of the topics can be found on bestpractice.bmj.com . Use of this content is subject to our disclaimer (. Use of this content is subject to our) . © BMJ Publishing Group Ltd 2024. All rights reserved.

the causative organism(s). For example, small chains of gram-positive cocci suggest a streptococcal infection, whereas clumps of large cocci suggest *Staphylococcus aureus* . Culture growth of blood or tissue specimens identifies a bacterial cause. The infection may be monomicrobial or polymicrobial.[1] [5] [16][19] [20]



Late signs of necrotizing fasciitis with extensive cellulitis, induration, skin necrosis, and formation of hemorrhagic bullae

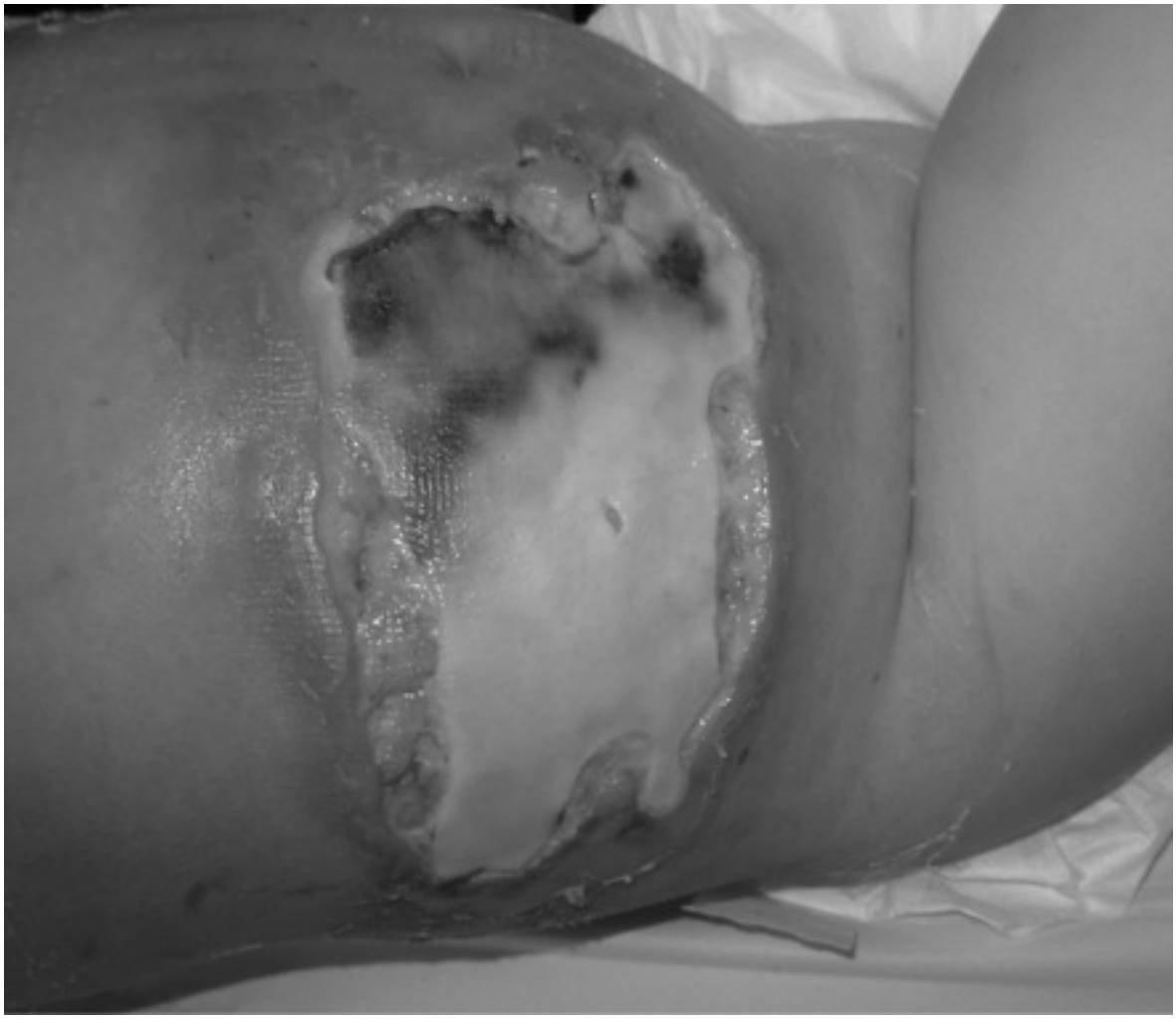
From: Hasham S, Matteucci P, Stanley PRW, et al. Necrotising fasciitis. BMJ. 2005 Apr 9;330(7495):830-3



Split thickness skin grafting after surgical debridement

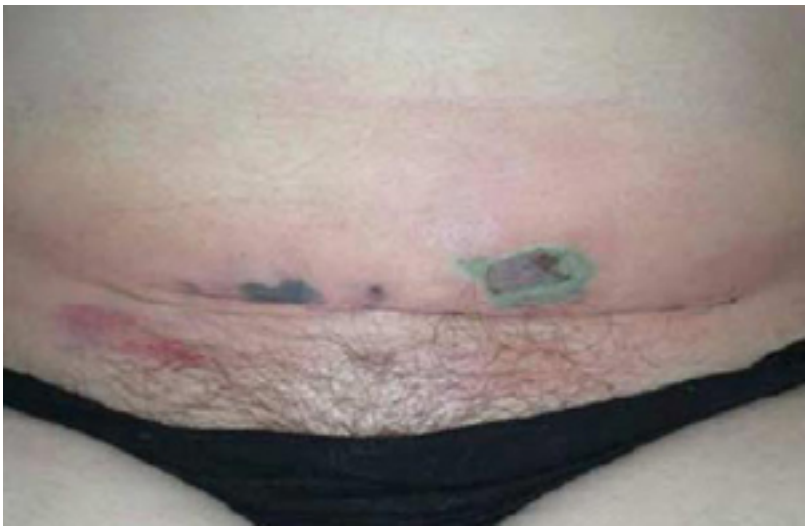
From: Hasham S, Matteucci P, Stanley PRW, et al. Necrotising fasciitis. BMJ. 2005 Apr 9;330(7495):830-3

This PDF of the BMJ Best Practice topic is based on the web version that was last updated: Jan 05, 2024. BMJ Best Practice topics are regularly updated and the most recent version of the topics can be found on bestpractice.bmj.com . Use of this content is subject to our disclaimer (. Use of this content is subject to our) . © BMJ Publishing Group Ltd 2024. All rights reserved.



Necrotizing 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



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

This PDF of the BMJ Best Practice topic is based on the web version that was last updated: Jan 05, 2024. BMJ Best Practice topics are regularly updated and the most recent version of the topics can be found on bestpractice.bmj.com . Use of this content is subject to our disclaimer (. Use of this content is subject to our) . © BMJ Publishing Group Ltd 2024. All rights reserved.

Consultation with an infectious disease expert to assist with empiric antibiotic regimen, as well as subsequent appropriate de-escalation of therapy, is highly recommended.[15]

History and exam

Key diagnostic factors

history of traumatic or nontraumatic cutaneous lesion (common)

- Cutaneous injury, surgery, trauma, intravenous drug use, or chronic or acute skin conditions (e.g., eczema, psoriasis, cutaneous ulcers, and burns) may serve as a cutaneous portal of entry for infective organisms.[1][5] [16][29]

anesthesia or severe pain over site of cellulitis (common)

- Anesthesia or severe pain over the site of cellulitis indicates necrotizing fasciitis.[1] [5] [16][43] The pain experienced with necrotizing fasciitis may be disproportionate to the visible skin changes.

fever (common)

- Systemic symptom of infection, though present in only 40% of patients with necrotizing fasciitis.[1] [3] [5][16]

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

- Systemic symptoms/signs of infection.[1] [3][5][16]

nausea and vomiting (common)

- Systemic symptoms of infection.[1][5]

delirium (uncommon)

- Systemic symptom of infection.[1][5]

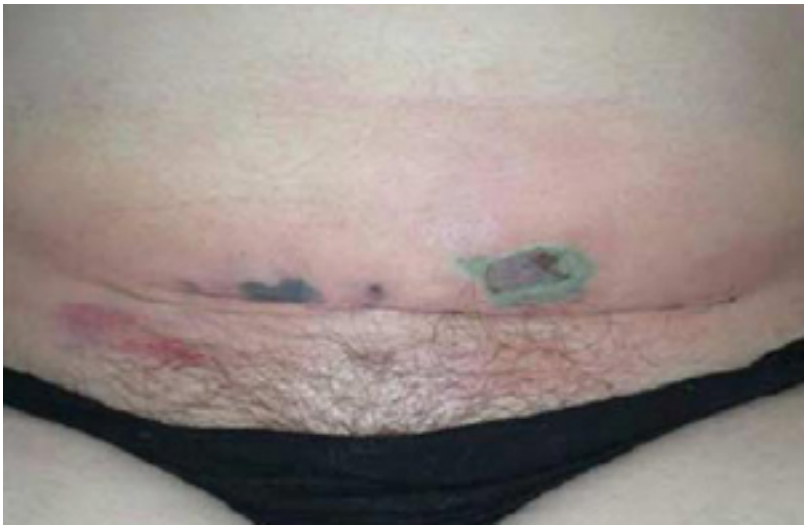
crepitus (uncommon)

- Examination of the skin overlying the area of cellulitis may reveal crepitus.[3] [16]

vesicles or bullae (uncommon)

- Examination of the skin overlying the area of cellulitis may reveal vesicles or bullae.[3] [16] It should be noted that patients with necrotizing fasciitis can present with normal overlying skin and that skin changes overlying group A streptococcal necrotizing fasciitis are a late sign.[16] Subtle skin changes such as leakage of fluid and edema precede the overt skin changes of blistering and redness.

 © BMJ Publishing Group Ltd 2024. All rights reserved.



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

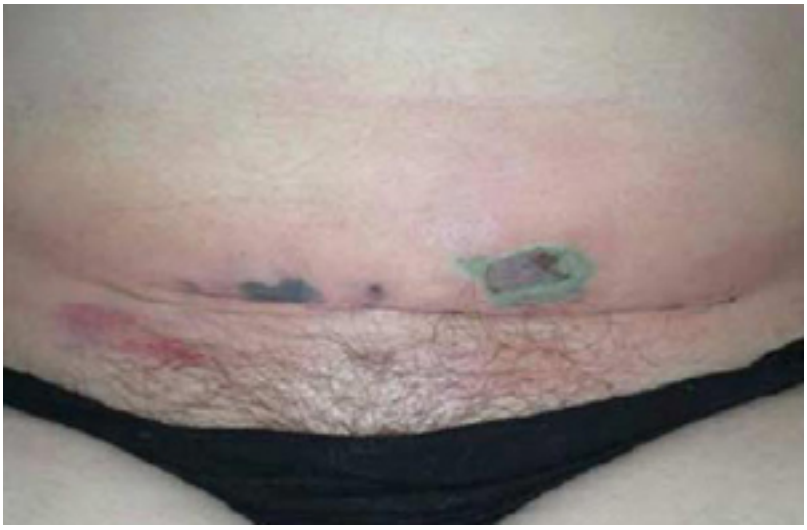
From: Hasham S, Matteucci P, Stanley PRW, et al. Necrotising fasciitis. BMJ. 2005 Apr 9;330(7495):830-3

gray discoloration of skin (uncommon)

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

edema or induration (uncommon)

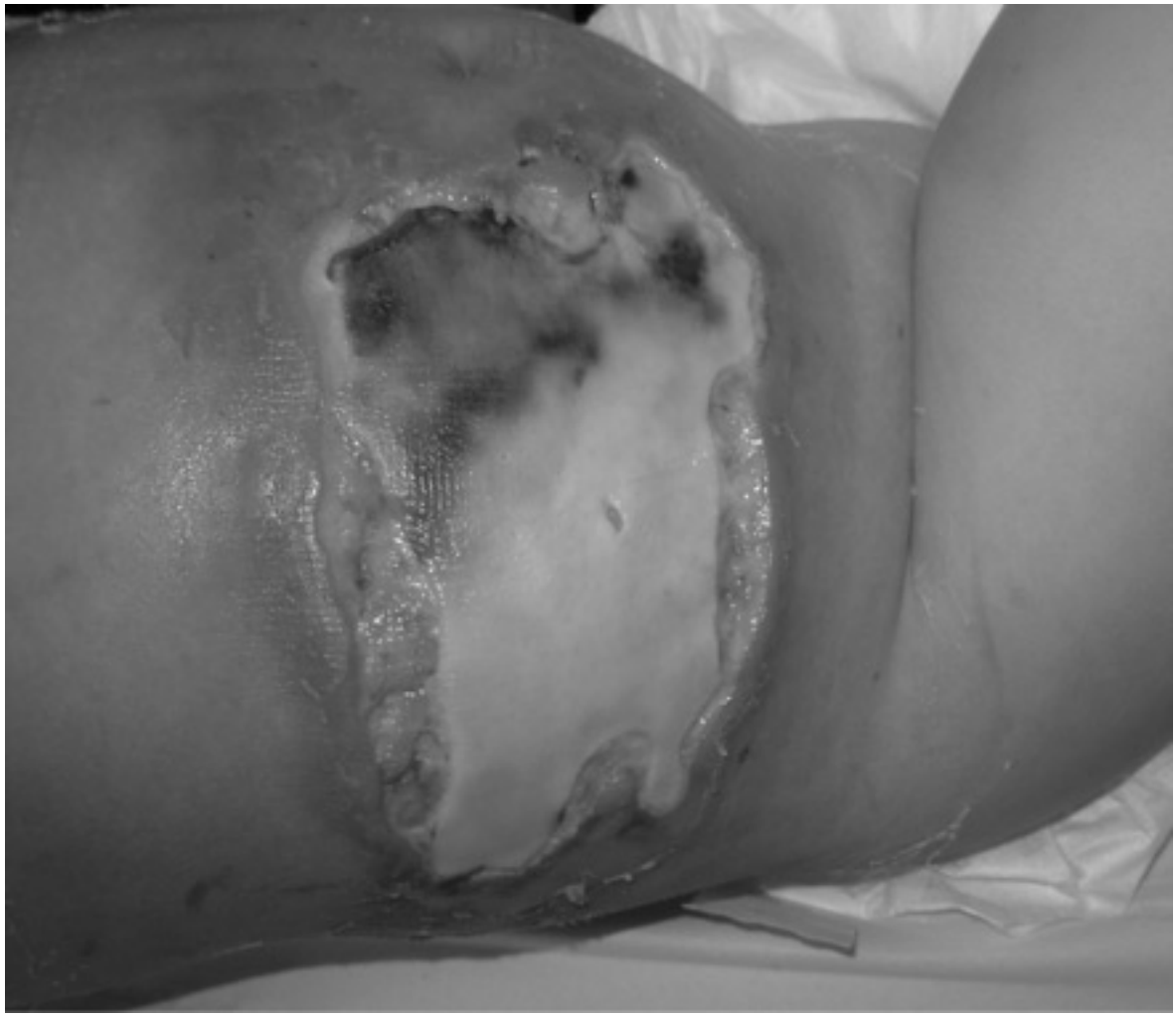
- Examination of the skin overlying the area of cellulitis may reveal edema.[5] Induration may be noted beyond the area of cellulitis.[5] It should be noted that patients with necrotizing fasciitis can present with normal overlying skin and that skin changes overlying group A streptococcal necrotizing fasciitis are a late sign. Subtle skin changes such as leakage of fluid and edema precede the overt skin changes of blistering and redness.



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

This PDF of the BMJ Best Practice topic is based on the web version that was last updated: Jan 05, 2024. BMJ Best Practice topics are regularly updated and the most recent version of the topics can be found on bestpractice.bmj.com . Use of this content is subject to our disclaimer (. Use of this content is subject to our) . © BMJ Publishing Group Ltd 2024. All rights reserved.



Necrotizing 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 concentrated in the perineum, trunk, and head and neck areas.[1] [5] [16][19] [20] The most common site of group A streptococcal necrotizing fasciitis is the thigh and necrotizing fasciitis of a limb, especially the arm, is more likely to be due to group A streptococcus than a polymicrobial infection. Some cases of necrotizing fasciitis may have associated myositis due to contiguous spread. This is more common in group A streptococcal than polymicrobial infections.
- Necrotizing fasciitis in the context of recent abdominal surgery or in the groin is most likely to be polymicrobial.

Risk factors

Strong

 © BMJ Publishing Group Ltd 2024. All rights reserved.

inpatient contact with index case

- Patient to patient spread of group A streptococcal infection, with median interval of 4 days to spread from index case to second case.[1][29]

Varicella zoster infection

- Serves as a cutaneous portal of entry for infective organisms.[1][29]

cutaneous injury, surgery, trauma

- Serve as a cutaneous portal of entry for infective organisms.[1] [5][16]

nontraumatic 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] [5][16][29]

intravenous drug use

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

Weak

chronic illness

- A generalized immunosuppressed state as a consequence of longstanding disease (alcoholism, diabetes mellitus, cardiac or pulmonary disease, peripheral vascular disease, renal failure) may predispose to soft-tissue infections.[1] [3][5] [16][29]

immunosuppression

- Immunosuppression due to malignancy and/or chemotherapy or radiation therapy, medications (especially chronic corticosteroid use), or infection (HIV) may predispose to soft-tissue infections.[3] Immunosuppressed status may lead to a delay in diagnosis and surgical management leading to greater risk of death.[16] [25] [30]

nonsteroidal anti-inflammatory drugs (NSAIDs)

- It has been postulated that use of NSAIDs may mask symptoms of necrotizing fasciitis, delaying diagnosis and that suppression 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.[31] [32] However, good evidence for the association of NSAIDs and necrotizing fasciitis in humans is not available.

This PDF of the BMJ Best Practice topic is based on the web version that was last updated: Jan 05, 2024. BMJ Best Practice topics are regularly updated and the most recent version of the topics can be found on bestpractice.bmj.com . Use of this content is subject to our disclaimer (. Use of this content is subject to our) . © BMJ Publishing Group Ltd 2024. All rights reserved.

Investigations

1st test to order

Test	Result
surgical exploration • If necrotizing fasciitis is suspected clinically, immediate surgical consultation for inspection, exploration, and debridement of infected tissue should be obtained.[3] [5] • The "finger test" is a surgical method that can be performed under local anesthesia at the bedside for the diagnosis of necrotizing fasciitis.[2] It involves making a 2 cm incision down to the deep fascia. Findings that suggest necrotizing fasciitis following incision include:[2] • Minimal resistance to finger dissection (a "positive" finger test) • Absence of bleeding • Presence of necrotic tissue • Murky or greyish "dishwater" fluid.	**necrotizing 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 bacteriologic diagnosis is best made using tissue specimens obtained from surgical debridement and blood cultures.[5]	**positive; may indicate polymicrobial or monomicrobial etiology**
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**
complete blood count and differential • High WBC is a nonspecific finding that may be seen in any systemic infection or circulatory collapse. A low WBC count may be a sign of severe sepsis. If a spreading soft-tissue infection is present, necrotizing fasciitis should be suspected.[2] [3] [4] [5] • If necrotizing fasciitis is suspected, immediately refer the patient to the surgical team; do not wait for the results of investigations before referral.[2] [5] Necrotizing fasciitis is a clinical diagnosis. However, investigations can support the diagnosis if this is unclear.[2]	**abnormally high or low WBC count with or without a left shift (elevated percentage of polymorphonuclear leukocytes and/or bands)**
serum electrolytes • Hyponatremia is a nonspecific finding that may be seen in any systemic infection or circulatory collapse. If a spreading soft-tissue infection is present, necrotizing fasciitis should be suspected.[2] [3] [4] [5] • If necrotizing fasciitis is suspected, immediately refer the patient to the surgical team; do not wait for the results of investigations before referral.[2] [5] Necrotizing fasciitis is a clinical diagnosis. However, investigations can support the diagnosis if this is unclear.[2]	**sodium may be decreased**
serum BUN and creatinine • A nonspecific finding that may be seen in any systemic infection or circulatory collapse. If a spreading soft-tissue infection is present, necrotizing fasciitis should be suspected.[2] [3] [4] [5] • If necrotizing fasciitis is suspected, immediately refer the patient to the surgical team; do not wait for the results of investigations before	**serum BUN and creatinine may be elevated**

This PDF of the BMJ Best Practice topic is based on the web version that was last updated: Jan 05, 2024. BMJ Best Practice topics are regularly updated and the most recent version of the topics can be found on bestpractice.bmj.com . Use of this content is subject to our disclaimer (. Use of this content is subject to our) . © BMJ Publishing Group Ltd 2024. All rights reserved.

Test	Result
referral.[2] [5] Necrotizing fasciitis is a clinical diagnosis. However, investigations can support the diagnosis if this is unclear.[2]	
serum CRP • Elevated CRP is a nonspecific finding that may be seen in a range of systemic infections. If a spreading soft-tissue infection is present, necrotizing fasciitis should be suspected.[2] [3] [4][5]	**usually elevated**
serum creatine kinase (CK) • A nonspecific finding suggestive of systemic infection or circulatory collapse. If a spreading soft-tissue infection is present, necrotizing fasciitis should be suspected.[2] [3] [4] [5]	**may be elevated**
serum lactate • A nonspecific finding suggestive of systemic infection. Elevated serum lactate at admission appears to be associated with the presence of necrotizing fasciitis.[44] High lactate may be associated with worse outcome and need for amputation.[15] [44]	**usually elevated**
clotting screen • Used to determine whether the patient has established coagulopathy in the presence of sepsis. This is associated with a worse prognosis.[45] • If you suspect necrotizing fasciitis, immediately refer the patient to the surgical team; do not wait for the results of investigations before referral.[2] [3] [4] [5] Necrotizing fasciitis is a clinical diagnosis. However, investigations can support the diagnosis if this is unclear.[2]	**may show coagulopathy**
arterial blood gas • Acidosis may be present in the setting of sepsis. May be obtained if there is concern for respiratory compromise. • Helps determine patient's respiratory status.	**hypoxemia, acidosis**

This PDF of the BMJ Best Practice topic is based on the web version that was last updated: Jan 05, 2024. BMJ Best Practice topics are regularly updated and the most recent version of the topics can be found on bestpractice.bmj.com . Use of this content is subject to our disclaimer (. Use of this content is subject to our) . © BMJ Publishing Group Ltd 2024. All rights reserved.

Other tests to consider

Test	Result
radiography, CT/MRI, ultrasound • Imaging studies should not delay surgical intervention when diagnosis is clinically suspected.[2] [3][5] • Plain radiography, ultrasound, or CT/MRI (if available) may be obtained in all patients with suspected necrotizing fasciitis, if clinically appropriate.[3] [36] Plain radiography is frequently normal during the early stages; subcutaneous gas may be present as the disease progresses. The diagnosis should be strongly suspected if soft-tissue gas is visualized on radiologic exam, which may also demonstrate abnormalities in the involved soft tissue.[1] [2] [16][36] However, soft-tissue gas is a late sign, and plain radiography has poor sensitivity for detecting signs of necrotizing infection.[15] • Ultrasound may help to differentiate simple cellulitis from necrotizing fasciitis and has the advantage that it can be rapidly performed at bedside.[2] 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 specificity of 93%.[37] • CT and MRI offer higher sensitivity.[3] [36] CT is indicated in abdominoperineal and cervicofacial infections to show the portal of entry of infection and to guide surgical intervention; for limb or peripheral necrotizing soft-tissue CT is of limited value.[15] [36] MRI is more sensitive for assessing necrotizing soft-tissue infections of the limbs, and may show thickening of the fascia, deep fascial fluid or edema. However, these signs are not specific to necrotizing infection and may be seen in other soft-tissue infections such as cellulitis, and MRI may be difficult to organize in an emergency and is not recommended as the first-line imaging technique.[2][3] [15][38] [46] • In a meta-analysis, CT had sensitivity of 88.5% and specificity of 93.3%, while plain radiography had sensitivity of 48.9% and specificity of 94.0% for diagnosing necrotizing soft tissue infections.[39]	**edema 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.[2] However, frozen-section soft-tissue biopsy requires specialist pathology expertise, takes time to perform, and is not widely available in all regions.[2]	**evidence of bacteria and tissue necrosis**

 © BMJ Publishing Group Ltd 2024. All rights reserved.

Differentials

Condition	Differentiating signs / symptoms	Differentiating tests
Cellulitis	• Systemic toxicity should be absent or minimal.[5]	• Absence of major abnormalities in complete 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* .[5]
Erysipelas	• Painful bright red, tender plaque with clear margins.[5]	• Culture of infected tissue identifies *S pyogenes* or other streptococci.[5]
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 necrotizing 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 edema.
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 edema 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, nonhemolytic mucoid colonies on 5% sheep's blood agar. • Punch biopsy of cutaneous lesion: necrosis of the dermis and epidermis, edema, and mild inflammatory infiltrate; abundant bacillary fragments (prior to antibiotic therapy); *Bacillus anthracis* (post-antibiotics).

This PDF of the BMJ Best Practice topic is based on the web version that was last updated: Jan 05, 2024. BMJ Best Practice topics are regularly updated and the most recent version of the topics can be found on bestpractice.bmj.com . Use of this content is subject to our disclaimer (. Use of this content is subject to our) . © BMJ Publishing Group Ltd 2024. All rights reserved.

Approach

Necrotizing fasciitis is a surgical emergency, requiring rapid debridement of the infected subcutaneous tissues, in combination with empiric antibiotic therapy directed broadly at the likely etiologic agents.[2] [3][5] [20][38]

Tailoring of antimicrobial therapy, as appropriate, is recommended when causative microbial organism(s) are identified via culture.

A combined team approach (surgeon, infectious disease specialist, microbiologist) provides the basis for optimal management.[15] [43]

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

Initial management

A surgical consultation should be obtained as soon as the diagnosis is suspected.[2] [5][43] Surgical debridement should be performed as soon as possible, but at least within 12 hours of hospital admission.[2] [38] The infected subcutaneous tissue is devitalized, so expedited surgical removal of infected tissue is critical for successful treatment. 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.[3] [38] [47]

While surgery is awaited, patients should be monitored for systemic toxicity (e.g., signs of end-organ damage), as well as local signs and symptoms of extension of the area of necrotizing fasciitis.[3] Empiric antibiotic therapy should be started immediately, then tailored based on culture results from subcutaneous tissue or blood, the patient's clinical condition, and discussion with the multidisciplinary team.[2][3][5] An early review from the critical care team should be sought; intensive hemodynamic support with intravenous fluids, and possibly vasoactive drugs, will be needed.[2] [5][43]

When debridement is performed, surgical incisions should extend beyond the areas of visible necrosis and the entire necrotic area excised.[3] Surgical specimens including tissue and fluid should be obtained for pathology and microbiological culture.[2] [5] 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 is lacking; a reasonable approach may be serial debridement every 12 to 24 hours until minimal or no remaining necrotic tissue is encountered.[2]

Streptococcal toxic shock syndrome

For patients who develop streptococcal toxic shock syndrome, the addition of intravenous immunoglobulin (IVIG) may be considered, but efficacy data are conflicting. Some studies suggest modest benefit; however, one Cochrane review showed no clear benefit on adverse events or mortality.[48] [49] [50] [51] [52] [53]

Infectious Diseases Society of America (IDSA) guidelines do not include a recommendation regarding the use of IVIG in patients with necrotizing fasciitis with streptococcal toxic shock syndrome, citing the need for additional efficacy studies.[5] World Society of Emergency Surgery consensus recommendations suggest consideration of IVIG in patients with necrotizing fasciitis due to group A streptococcus.[2] [3]

This PDF of the BMJ Best Practice topic is based on the web version that was last updated: Jan 05, 2024. BMJ Best Practice topics are regularly updated and the most recent version of the topics can be found on bestpractice.bmj.com . Use of this content is subject to our disclaimer (. Use of this content is subject to our) . © BMJ Publishing Group Ltd 2024. All rights reserved.

Choice of antibiotics

Until microbial etiology and antimicrobial susceptibilities are known, empiric broad-spectrum antibiotics should be administered targeting the most common etiologies of type I infection (mixed infections with 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), and also type II infection due to group A streptococcus. Consider local resistance and epidemiologic patterns (including extended-spectrum beta-lactamase or carbapenemase-producing organisms).

When further information is available and the etiologic agent has been determined, antibiotic therapy should be tailored to target the specific agent. As there are currently no definitive clinical trials, the IDSA recommends continuing antibiotics until no further surgical debridement is needed, the patient has improved clinically, and fever has been absent for 48 to 72 hours.[5] [54]

Recommended empiric regimens

Include vancomycin, linezolid, tedizolid, or daptomycin combined with either: piperacillin/tazobactam or a carbapenem (e.g., meropenem, imipenem/cilastatin, ertapenem). Local resistance patterns (including extended-spectrum beta-lactamase or carbapenemase-producing organisms) should be considered. Vancomycin should be used with caution in patients with renal impairment.

Until group A streptococcus involvement is excluded, antimicrobial agents that inhibit toxin production should be included empirically. Clindamycin should be added to empiric treatment until group A streptococcus involvement has been excluded if linezolid is not already being used as part of the empiric regimen.[5][34]

Fungal pathogens (especially mucorales) are rare causes of necrotizing fasciitis; empiric inclusion of antifungal agents is not recommended.

Recommendations for type I mixed infections

Include vancomycin, linezolid, tedizolid, or daptomycin combined with either: piperacillin/tazobactam or a carbapenem (e.g., meropenem, imipenem/cilastatin, ertapenem). The IDSA supports some of these regimens.[5]

Recommendations for type II infection

Type II infection is most commonly due to group A streptococcus; clindamycin plus penicillin is recommended.[5] For patients with a penicillin allergy, vancomycin monotherapy may be used. When monomicrobial *Staphylococcus aureus* is the etiologic agent, antibiotics active against MRSA should be used until cultures confirm susceptibilities; options include vancomycin, linezolid, tedizolid, or daptomycin. Ceftaroline, telavancin, or dalbavancin are also reasonable choices, although clinical data are sparse.[2] [5]

Nafcillin, oxacillin, or cefazolin may be used if methicillin susceptibility is confirmed.

Doxycycline is used in the management of type II necrotizing fasciitis attributable to *Vibrio vulnificus* and *Aeromonas hydrophila* .

Fungal pathogens are rare causes of necrotizing fasciitis; lipid amphotericin B is the primary treatment option for patients with mucorales infections.

This PDF of the BMJ Best Practice topic is based on the web version that was last updated: Jan 05, 2024. BMJ Best Practice topics are regularly updated and the most recent version of the topics can be found on bestpractice.bmj.com . Use of this content is subject to our disclaimer (. Use of this content is subject to our) . © BMJ Publishing Group Ltd 2024. All rights reserved.

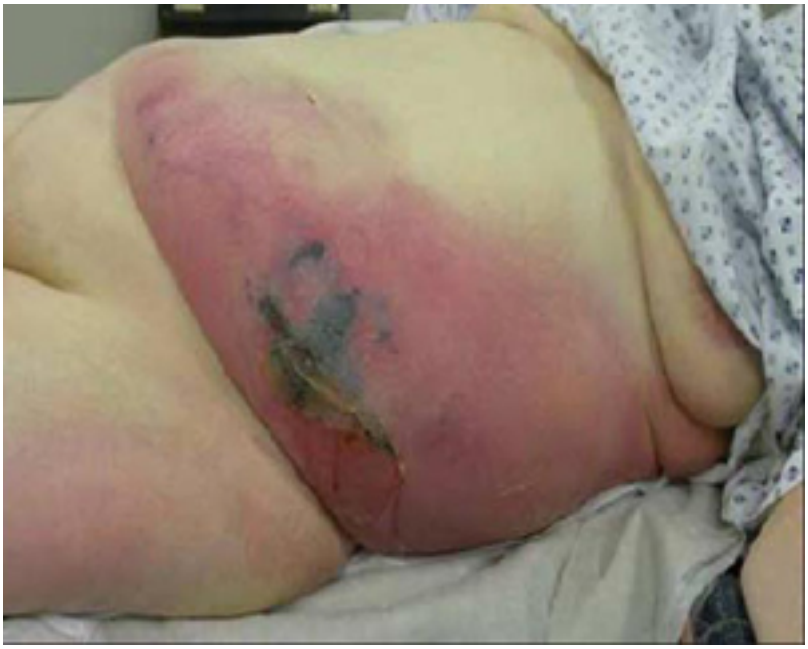
Subsequent management

In treatment-resistant patients, the need for additional debridement or alteration in antibiotic therapy (based on culture results from subcutaneous tissue or blood) should be considered.[3] [5]

After complete removal of necrotic tissue and final debridement, negative pressure wound therapy may be considered to help with granulation and healing of the wound.[15]

Supportive surgical interventions such as fecal diversion for colostomy in cases of Fournier gangrene with fecal contamination, or tracheostomy for patients with cervicofacial necrotizing fasciitis may be warranted. [3] [18]

Where functional and cosmetic disability results from extensive surgical debridement for necrotizing fasciitis, reconstructive surgery with skin grafting may be required.[3]



Late signs of necrotizing fasciitis with extensive cellulitis, induration, skin necrosis, and formation of hemorrhagic bullae

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

After prolonged hospitalization and recurrent surgical interventions, physical therapy and rehabilitation may be needed for certain patients.

 © BMJ Publishing Group Ltd 2024. All rights reserved.

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)		
organism unknown		
	1st	**surgical debridement and hemodynamic support**
	plus	**empiric broad-spectrum antibiotics**

This PDF of the BMJ Best Practice topic is based on the web version that was last updated: Jan 05, 2024. BMJ Best Practice topics are regularly updated and the most recent version of the topics can be found on bestpractice.bmj.com . Use of this content is subject to our disclaimer (. Use of this content is subject to our) . © BMJ Publishing Group Ltd 2024. All rights reserved.

Acute (summary)		
type I necrotizing fasciitis (polymicrobial)		
	1st	**surgical debridement and hemodynamic support**
	plus	**intravenous antibiotics**
type II necrotizing fasciitis due to group A streptococcus		
	1st	**surgical debridement and hemodynamic support**
	plus	**intravenous antibiotics**
■ **streptococcal toxic shock**	**adjunct**	**intravenous immune globulin (IVIG)**
type II necrotizing fasciitis due to Staphylococcus aureus		
	1st	**surgical debridement and hemodynamic support**
	plus	**intravenous antibiotics**
type II necrotizing fasciitis due to Vibrio vulnificus		
	1st	**surgical debridement and hemodynamic support**
	plus	**intravenous antibiotics**
type II necrotizing fasciitis due to Aeromonas hydrophila		
	1st	**surgical debridement and hemodynamic support**
	plus	**intravenous antibiotics**
type II necrotizing fasciitis due to mucorales		
	1st	**surgical debridement and hemodynamic support**
	plus	**antifungal therapy**

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

This PDF of the BMJ Best Practice topic is based on the web version that was last updated: Jan 05, 2024. BMJ Best Practice topics are regularly updated and the most recent version of the topics can be found on bestpractice.bmj.com . Use of this content is subject to our disclaimer (. Use of this content is subject to our) . © BMJ Publishing Group Ltd 2024. All rights reserved.

Treatment algorithm

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

organism unknown

1st **surgical debridement and hemodynamic support**

» Necrotizing fasciitis is a surgical emergency and the patient should be urgently taken to the operating room for debridement of all infected, devitalized tissues.[3]

» When debridement is performed, surgical incisions should extend beyond the areas of visible necrosis and the entire necrotic area excised.[3]

» Intraoperative tissue and fluid should be obtained for microbiological culture.

» Intensive hemodynamic support with intravenous infusion is an important aspect of surgical management.[2] [3] [5]

» Further surgical evaluation and debridement is necessary in most cases, and several procedures may be required to ensure that all necrotic tissue is removed.[5] Data to guide optimal timing for surgical re-exploration is lacking; a reasonable approach may be serial debridement every 12-24 hours until minimal or no remaining necrotic tissue is encountered.[2] [3]

» After complete removal of necrotic tissue and final debridement, negative pressure wound therapy may be considered to help with granulation and healing of the wound.[15]

plus **empiric broad-spectrum antibiotics**

Treatment recommended for ALL patients in selected patient group

Primary options

» vancomycin: 30 mg/kg/day intravenously given in divided doses every 12 hours
-or-
» linezolid: 600 mg intravenously every 12 hours
-or-
» tedizolid phosphate: 200 mg intravenously every 24 hours
-or-

MANAGEMENT

This PDF of the BMJ Best Practice topic is based on the web version that was last updated: Jan 05, 2024. BMJ Best Practice topics are regularly updated and the most recent version of the topics can be found on bestpractice.bmj.com . Use of this content is subject to our disclaimer (. Use of this content is subject to our) . © BMJ Publishing Group Ltd 2024. All rights reserved.

Initial

» daptomycin: 4 mg/kg intravenously every 24 hours

--AND--

» piperacillin/tazobactam: 3.375 g intravenously every 6 hours
Dose consists of 3 g piperacillin plus 0.375 g tazobactam.

-or-

» imipenem/cilastatin: 1 g intravenously every 6-8 hours
Dose refers to imipenem component.

-or-

» meropenem: 1 g intravenously every 8 hours

-or-

» ertapenem: 1 g intravenously every 24 hours

--AND--

» clindamycin: 600-900 mg intravenously every 8 hours
Only add clindamycin to the empiric regimen if linezolid is not already being used, until group A streptococcus involvement is excluded.

» Until microbial etiology and antimicrobial susceptibilities are known, empiric broad-spectrum antibiotics should be administered targeting the most common etiologies for necrotizing fasciitis. These include type I infections (mixed infections with 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), and type II infections (group A streptococcus [i.e., *Streptococcus pyogenes*]). Consider local resistance and epidemiologic patterns (including extended-spectrum beta-lactamase or carbapenemase-producing organisms).

» Recommended empiric regimens include vancomycin, linezolid, tedizolid, or daptomycin combined with either: piperacillin/tazobactam or a carbapenem (e.g., meropenem, imipenem/cilastatin, ertapenem).

» Until group A streptococcus involvement is excluded, antimicrobial agents that inhibit toxin production should be included empirically. If linezolid is not used as part of the empiric regimen, clindamycin should be added, until

This PDF of the BMJ Best Practice topic is based on the web version that was last updated: Jan 05, 2024. BMJ Best Practice topics are regularly updated and the most recent version of the topics can be found on bestpractice.bmj.com . Use of this content is subject to our disclaimer (. Use of this content is subject to our) . © BMJ Publishing Group Ltd 2024. All rights reserved.

Initial

group A streptococcus treatment has been excluded.[5][34]

» When further information is available and the etiologic agent has been determined, antibiotic therapy should be tailored to target the specific agent.

» As there are currently no definitive clinical trials, the IDSA recommends continuing antibiotics until no further surgical debridement is needed, the patient has improved clinically, and fever has been absent for 48 to 72 hours.[5][54]

This PDF of the BMJ Best Practice topic is based on the web version that was last updated: Jan 05, 2024. BMJ Best Practice topics are regularly updated and the most recent version of the topics can be found on bestpractice.bmj.com . Use of this content is subject to our disclaimer (. Use of this content is subject to our) . © BMJ Publishing Group Ltd 2024. All rights reserved.

Acute

type I necrotizing fasciitis (polymicrobial)

1st **surgical debridement and hemodynamic support**

» Necrotizing fasciitis is a surgical emergency and the patient should be urgently taken to the operating room for debridement of all infected, devitalized tissues.[3]

» When debridement is performed, surgical incisions should extend beyond the areas of visible necrosis and the entire necrotic area excised.[3]

» Intensive hemodynamic support with intravenous infusion is an important aspect of surgical management.[2] [3] [5]

» Further surgical evaluation and debridement is necessary in most cases, and several procedures may be required to ensure that all necrotic tissue is removed.[5] Data to guide optimal timing for surgical re-exploration is lacking; a reasonable approach may be serial debridement every 12-24 hours until minimal or no remaining necrotic tissue is encountered.[2] [3]

plus **intravenous antibiotics**

Treatment recommended for ALL patients in selected patient group

Primary options

» vancomycin: 30 mg/kg/day intravenously given in divided doses every 12 hours
-or-
» linezolid: 600 mg intravenously every 12 hours
-or-
» tedizolid phosphate: 200 mg intravenously every 24 hours
-or-
» daptomycin: 4 mg/kg intravenously every 24 hours

--AND--

» piperacillin/tazobactam: 3.375 g intravenously every 6 hours
Dose consists of 3 g piperacillin plus 0.375 g tazobactam.

-or-
» imipenem/cilastatin: 1 g intravenously every 6-8 hours
Dose refers to imipenem component.

This PDF of the BMJ Best Practice topic is based on the web version that was last updated: Jan 05, 2024. BMJ Best Practice topics are regularly updated and the most recent version of the topics can be found on bestpractice.bmj.com . Use of this content is subject to our disclaimer (. Use of this content is subject to our) . © BMJ Publishing Group Ltd 2024. All rights reserved.

Acute

-or-
» meropenem: 1 g intravenously every 8 hours
-or-
» ertapenem: 1 g intravenously every 24 hours

» In addition to urgent surgical debridement, antibiotics should be administered that cover the most common etiologies of type I necrotizing fasciitis (a mixed infection with anaerobes such as *Bacteroides* or *Peptostreptococcus* with a facultative anaerobe such as *Escherichia coli*, *Enterobacter*, *Klebsiella*, *Proteus*, MRSA, or non-group A streptococcus).

» Recommendations for type I mixed infections include vancomycin, linezolid, tedizolid, or daptomycin combined with either: piperacillin/tazobactam or a carbapenem (e.g., meropenem, imipenem/cilastatin, ertapenem).[2] [5]

» When further information is available and the etiologic agent has been determined, antibiotic therapy should be tailored to target the specific agent.

» As there are currently no definitive clinical trials, the IDSA recommends continuing antibiotics until no further surgical debridement is needed, the patient has improved clinically, and fever has been absent for 48 to 72 hours.[5] [54]

type II necrotizing fasciitis due to group A streptococcus

1st **surgical debridement and hemodynamic support**

» Necrotizing fasciitis is a surgical emergency and the patient should be urgently taken to the operating room for debridement of all infected, devitalized tissues.[3]

» When debridement is performed, surgical incisions should extend beyond the areas of visible necrosis and the entire necrotic area excised.[3]

» Intensive hemodynamic support with intravenous infusion is an important aspect of surgical management.[2] [3] [5]

» Further surgical evaluation and debridement is necessary in most cases, and several procedures may be required to ensure that all necrotic tissue is removed.[5] Data to guide optimal timing for surgical re-exploration is

This PDF of the BMJ Best Practice topic is based on the web version that was last updated: Jan 05, 2024. BMJ Best Practice topics are regularly updated and the most recent version of the topics can be found on bestpractice.bmj.com . Use of this content is subject to our disclaimer (. Use of this content is subject to our) . © BMJ Publishing Group Ltd 2024. All rights reserved.

Acute

lacking; a reasonable approach may be serial debridement every 12-24 hours until minimal or no remaining necrotic tissue is encountered.[2] [3]

plus **intravenous antibiotics**

Treatment recommended for ALL patients in selected patient group

Primary options

» penicillin G sodium: 2-4 million units intravenously every 4-6 hours
-and-
» clindamycin: 600-900 mg intravenously every 8 hours

Secondary options

» vancomycin: 30 mg/kg/day intravenously given in divided doses every 12 hours

» Type II infection is most commonly due to group A streptococcus; clindamycin; plus penicillin is recommended.

» For patients with a penicillin allergy, vancomycin monotherapy may be used.

streptococcal toxic shock **adjunct** **intravenous immune globulin (IVIG)**

Treatment recommended for SOME patients in selected patient group

Primary options

» immune globulin (human): 1 g/kg intravenously on day 1, followed by 0.5 g/kg on days 2 and 3; or 2 g/kg intravenously as a single dose
Dose regimens vary; consult specialist for further guidance on dose.

» The addition of intravenous gamma globulin may be considered for treatment of streptococcal toxic shock syndrome, although data on efficacy are conflicting.[2] [3] [5]

type II necrotizing fasciitis due to Staphylococcus aureus

1st **surgical debridement and hemodynamic support**

» Necrotizing fasciitis is a surgical emergency and the patient should be urgently taken to the operating room for debridement of all infected, devitalized tissues.[3]

This PDF of the BMJ Best Practice topic is based on the web version that was last updated: Jan 05, 2024. BMJ Best Practice topics are regularly updated and the most recent version of the topics can be found on bestpractice.bmj.com . Use of this content is subject to our disclaimer (. Use of this content is subject to our) . © BMJ Publishing Group Ltd 2024. All rights reserved.

Acute

» When debridement is performed, surgical incisions should extend beyond the areas of visible necrosis and the entire necrotic area excised.[3]

» Intensive hemodynamic support with intravenous infusion is an important aspect of surgical management.[2] [3] [5]

» Further surgical evaluation and debridement is necessary in most cases, and several procedures may be required to ensure that all necrotic tissue is removed.[5] Data to guide optimal timing for surgical re-exploration is lacking; a reasonable approach may be serial debridement every 12-24 hours until minimal or no remaining necrotic tissue is encountered.[2] [3]

» After complete removal of necrotic tissue and final debridement, negative pressure wound therapy may be considered to help with granulation and healing of the wound.[15]

plus **intravenous antibiotics**

Treatment recommended for ALL patients in selected patient group

Primary options

MRSA

» vancomycin: 30 mg/kg/day intravenously given in divided doses every 12 hours

OR

MRSA

» linezolid: 600 mg intravenously every 12 hours

OR

MRSA

» tedizolid phosphate: 200 mg intravenously every 24 hours

OR

MRSA

» daptomycin: 4 mg/kg intravenously every 24 hours

Secondary options

MRSA

» ceftaroline fosamil: 600 mg intravenously every 12 hours

This PDF of the BMJ Best Practice topic is based on the web version that was last updated: Jan 05, 2024. BMJ Best Practice topics are regularly updated and the most recent version of the topics can be found on bestpractice.bmj.com . Use of this content is subject to our disclaimer (. Use of this content is subject to our) . © BMJ Publishing Group Ltd 2024. All rights reserved.

Acute

OR

MRSA

» dalbavancin: 1500 mg intravenously as a single dose; or 1000 mg intravenously as a single dose followed by 500 mg one week later

OR

MRSA

» telavancin: 10 mg/kg intravenously every 24 hours

Tertiary options

MSSA

» nafcillin: 1-2 g intravenously every 4 hours

OR

MSSA

» oxacillin: 1-2 g intravenously every 4 hours

OR

MSSA

» cefazolin: 1 g intravenously every 6-8 hours

» In addition to urgent surgical debridement, antistaphylococcal antibiotics should be administered.

» Antibiotics active against MRSA should be used until cultures confirm susceptibilities. Options include vancomycin, linezolid, tedizolid, or daptomycin; ceftaroline, telavancin, or dalbavancin are also reasonable choices, although clinical data are sparse.[2] [5]

» Nafcillin, oxacillin, or cefazolin may be used if methicillin susceptibility is confirmed.

type II necrotizing fasciitis due to Vibrio vulnificus

1st **surgical debridement and hemodynamic support**

» Necrotizing fasciitis is a surgical emergency and the patient should be urgently taken to the operating room for debridement of all infected, devitalized tissues.[3]

» When debridement is performed, surgical incisions should extend beyond the areas of

This PDF of the BMJ Best Practice topic is based on the web version that was last updated: Jan 05, 2024. BMJ Best Practice topics are regularly updated and the most recent version of the topics can be found on bestpractice.bmj.com . Use of this content is subject to our disclaimer (. Use of this content is subject to our) . © BMJ Publishing Group Ltd 2024. All rights reserved.

Acute

visible necrosis and the entire necrotic area excised.[3]

» Intensive hemodynamic support with intravenous infusion is an important aspect of surgical management.[2] [3] [5]

» Further surgical evaluation and debridement is necessary in most cases, and several procedures may be required to ensure that all necrotic tissue is removed.[5] Data to guide optimal timing for surgical re-exploration is lacking; a reasonable approach may be serial debridement every 12-24 hours until minimal or no remaining necrotic tissue is encountered.[2] [3]

plus **intravenous antibiotics**

Treatment recommended for ALL patients in selected patient group

Primary options

» doxycycline: 100 mg intravenously every 12 hours

--AND--

» ceftazidime sodium: 2 g intravenously every 8 hours
-or-
» ceftriaxone: 2 g intravenously every 12-24 hours
-or-
» ciprofloxacin: 400 mg intravenously every 8-12 hours

» Predisposing risk factors include hepatic disease, diabetes mellitus, chronic renal insufficiency, and adrenal insufficiency.[9]

type II necrotizing fasciitis due to Aeromonas hydrophila

1st **surgical debridement and hemodynamic support**

» Necrotizing fasciitis is a surgical emergency and the patient should be urgently taken to the operating room for debridement of all infected, devitalized tissues.[3]

» When debridement is performed, surgical incisions should extend beyond the areas of visible necrosis and the entire necrotic area excised.[3]

» Intensive hemodynamic support with intravenous infusion is an important aspect of surgical management.[2] [3] [5]

This PDF of the BMJ Best Practice topic is based on the web version that was last updated: Jan 05, 2024. BMJ Best Practice topics are regularly updated and the most recent version of the topics can be found on bestpractice.bmj.com . Use of this content is subject to our disclaimer (. Use of this content is subject to our) . © BMJ Publishing Group Ltd 2024. All rights reserved.

Acute

» Further surgical evaluation and debridement is necessary in most cases, and several procedures may be required to ensure that all necrotic tissue is removed.[5] Data to guide optimal timing for surgical re-exploration is lacking; a reasonable approach may be serial debridement every 12-24 hours until minimal or no remaining necrotic tissue is encountered.[2] [3]

plus **intravenous antibiotics**

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

Secondary options

» doxycycline: 100 mg intravenously every 12 hours
--AND--
» ceftriaxone: 2 g intravenously every 12-24 hours
-or-
» cefepime: 2 g intravenously every 8-12 hours

» *Aeromonas hydrophila* infection may cause necrotizing fasciitis in patients with suppressed immune systems, burns, and trauma in an aquatic setting.[10]

type II necrotizing fasciitis due to mucorales

1st **surgical debridement and hemodynamic support**

» Necrotizing fasciitis is a surgical emergency and the patient should be urgently taken to the operating room for debridement of all infected, devitalized tissues.[3]

» When debridement is performed, surgical incisions should extend beyond the areas of visible necrosis and the entire necrotic area excised.[3]

» Intensive hemodynamic support with intravenous infusion is an important aspect of surgical management.[2] [3] [5]

This PDF of the BMJ Best Practice topic is based on the web version that was last updated: Jan 05, 2024. BMJ Best Practice topics are regularly updated and the most recent version of the topics can be found on bestpractice.bmj.com . Use of this content is subject to our disclaimer (. Use of this content is subject to our) . © BMJ Publishing Group Ltd 2024. All rights reserved.

Acute

» Further surgical evaluation and debridement is necessary in most cases, and several procedures may be required to ensure that all necrotic tissue is removed.[5] Data to guide optimal timing for surgical re-exploration is lacking; a reasonable approach may be serial debridement every 12-24 hours until minimal or no remaining necrotic tissue is encountered.[2] [3]

» After complete removal of necrotic tissue and final debridement, negative pressure wound therapy may be considered to help with granulation and healing of the wound.[15]

plus **antifungal therapy**

Treatment recommended for ALL patients in selected patient group

Primary options

» amphotericin B lipid complex: 5 mg/kg intravenously every 24 hours

Secondary options

» isavuconazonium sulfate: 372 mg intravenously/orally every 8 hours for 6 doses as a loading dose, followed by 372 mg every 24 hours (initiate maintenance dose 12-24 hours after last loading dose)
372 mg isavuconazonium is equivalent to 200 mg isavuconazole.

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

This PDF of the BMJ Best Practice topic is based on the web version that was last updated: Jan 05, 2024. BMJ Best Practice topics are regularly updated and the most recent version of the topics can be found on bestpractice.bmj.com . Use of this content is subject to our disclaimer (. Use of this content is subject to our) . © BMJ Publishing Group Ltd 2024. All rights reserved.

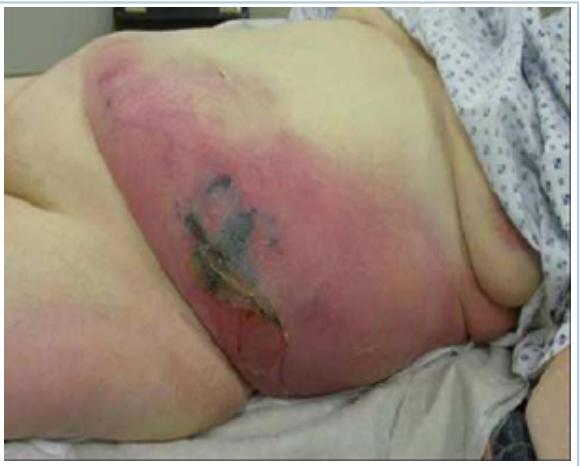
Ongoing

persistent cosmetic and functional defects after debridement

1st **reconstructive surgery**

» Supportive surgical interventions, such as fecal diversion for colostomy in cases of Fournier gangrene with fecal contamination, or tracheostomy for patients with cervicofacial necrotizing fasciitis may be warranted.[3] [18]

» Where functional and cosmetic disability results from extensive surgical debridement for necrotizing fasciitis, reconstructive surgery may be required.[3]



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

» After prolonged hospitalization and recurrent surgical interventions, physical therapy and rehabilitation may be needed for certain patients.

This PDF of the BMJ Best Practice topic is based on the web version that was last updated: Jan 05, 2024. BMJ Best Practice topics are regularly updated and the most recent version of the topics can be found on bestpractice.bmj.com . Use of this content is subject to our disclaimer (. Use of this content is subject to our) . © BMJ Publishing Group Ltd 2024. All rights reserved.

Emerging

Hyperbaric oxygen (HBO)

HBO 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] [55] HBO may be considered if readily available; however, its use must not delay surgical debridement or appropriate antibiotic treatment.

Newer antibiotics

Guidance by an infectious disease expert should be sought if any of these agents are being considered. Antibiotics with activity against gram-positive organisms (including MRSA) - such as the lipoglycopeptide oritavancin, the aminomethylcycline omadacycline (spectrum includes gram-positive, gram-negative aerobes, anaerobes, and atypical bacteria), and the fluoroquinolone delafloxacin - may be considered for inclusion in an antimicrobial regimen for necrotizing fasciitis.[56] [57] [58] However, clinical data supporting their use is limited. Although not specifically approved by the Food and Drug Administration for acute bacterial skin and skin-structure infections, ceftazidime/avibactam and meropenem/vaborbactam offer a spectrum of activity against resistant gram-negative organisms, including those with extended-spectrum beta-lactamases and some carbapenamases. The efficacy of these agents has not been rigorously demonstrated in necrotizing fasciitis. 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 necrotizing fasciitis.

Reltecimod

Reltecimod is a T-lymphocyte CD28 receptor antagonist that acts early in the host immune response by inhibiting T-cell activation, thereby modulating acute inflammation that leads to systemic organ failure. It has been evaluated for suspected organ dysfunction or failure in patients ages ≥12 years with necrotizing soft tissue infection, in conjunction with surgical debridement, antibiotic therapy, and supportive care. In a phase 3 randomized controlled trial, reltecimod did not demonstrate significant improvement in a composite endpoint for patients with necrotizing soft-tissue infections; however, it was associated with improved resolution of organ dysfunction and hospital discharge status.[59] Other immunomodulating agents may have a role in the treatment of necrotizing fasciitis in the future.

Primary prevention

Includes:

- 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[5]
- Immunization against varicella zoster virus (in adults and children), that may prevent necrotizing 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 necrotizing fasciitis.

Chemoprophylaxis may be considered for close contacts of patients with invasive group A streptococcus disease including necrotizing fasciitis, especially if those contacts are at high-risk for invasive group A streptococcus disease, though it is not routinely recommended.[60]

This PDF of the BMJ Best Practice topic is based on the web version that was last updated: Jan 05, 2024. BMJ Best Practice topics are regularly updated and the most recent version of the topics can be found on bestpractice.bmj.com . Use of this content is subject to our disclaimer (. Use of this content is subject to our) . © BMJ Publishing Group Ltd 2024. All rights reserved.

Patient discussions

Patients should be instructed that this 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 necrotizing fasciitis is rare. However, significant functional and cosmetic morbidity may remain following initial surgical therapies, which may require subsequent reconstruction.

Psychologic support should be offered to all patients.

This PDF of the BMJ Best Practice topic is based on the web version that was last updated: Jan 05, 2024. BMJ Best Practice topics are regularly updated and the most recent version of the topics can be found on bestpractice.bmj.com . Use of this content is subject to our disclaimer (. Use of this content is subject to our) . © BMJ Publishing Group Ltd 2024. All rights reserved.

FOLLOW UP

Monitoring

Monitoring

Patients need to be monitored for lack of response to therapy. This most often occurs when there is a need for additional, more extensive debridement. Less commonly, lack of response may be due to either antimicrobial resistance or incorrect antibiotic agent selection.

Monitoring for decline in respiratory/hemodynamic function is essential, with consideration of possible toxic shock in patients with group A streptococcal infection.

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.[43]

- Surgical re-exploration to assess the need for further debridement should be performed at least every 12-24 hours after the initial debridement.[2]
- 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 imaging markers (particularly white blood cell count).[2]

Complications

Complications	Timeframe	Likelihood
mortality	**short term**	**high**
Mortality from necrotizing 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%.[5]		
skin loss and scarring	**long term**	**high**
Functional and cosmetic disability may result from extensive surgical debridement for necrotizing fasciitis. Reconstructive surgery may be required.[3]		

Prognosis

Mortality from necrotizing 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%.[5]

Recurrence of necrotizing fasciitis is rare.[1] [5] However, significant functional and cosmetic morbidity may remain following initial surgical therapies, which may require subsequent reconstruction.

This PDF of the BMJ Best Practice topic is based on the web version that was last updated: Jan 05, 2024. BMJ Best Practice topics are regularly updated and the most recent version of the topics can be found on bestpractice.bmj.com . Use of this content is subject to our disclaimer (. Use of this content is subject to our) . © BMJ Publishing Group Ltd 2024. All rights reserved.

Diagnostic guidelines

International

Practice guidelines for the diagnosis and management of skin and soft-tissue infections (https://www.idsociety.org/practice-guideline/practice-guidelines) [5]

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

Global clinical pathways for patients with skin and soft tissue infections (https://wjes.biomedcentral.com/articles/10.1186/s13017-022-00406-2#Sec17) [3]

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

WSES/SIS-E consensus conference: recommendations for the management of skin and soft-tissue infections (https://wjes.biomedcentral.com/articles/10.1186/s13017-018-0219-9) [2]

Published by: World Society of Emergency Surgery **Last published:** 2018

Treatment guidelines

International

Practice guidelines for the diagnosis and management of skin and soft-tissue infections (http://www.idsociety.org/PracticeGuidelines) [5]

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

Global clinical pathways for patients with skin and soft tissue infections (https://wjes.biomedcentral.com/articles/10.1186/s13017-022-00406-2#Sec17) [3]

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

WSES/SIS-E consensus conference: recommendations for the management of skin and soft-tissue infections (https://wjes.biomedcentral.com/articles/10.1186/s13017-018-0219-9) [2]

Published by: World Society of Emergency Surgery **Last published:** 2018

This PDF of the BMJ Best Practice topic is based on the web version that was last updated: Jan 05, 2024. BMJ Best Practice topics are regularly updated and the most recent version of the topics can be found on bestpractice.bmj.com . Use of this content is subject to our disclaimer (. Use of this content is subject to our) . © BMJ Publishing Group Ltd 2024. All rights reserved.

REFERENCES

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 (https://wjes.biomedcentral.com/articles/10.1186/s13017-018-0219-9) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/30564282?tool=bestpractice.bmj.com)
- Stevens DL, Bisno AL, Chambers HF, et al. Practice guidelines for the diagnosis and management of skin and soft tissue infections: 2014 update by the Infectious Diseases Society of America. Clin Infect Dis. 2014 Jul 15;59(2):e10-52. Full text (https://academic.oup.com/cid/article/59/2/e10/2895845) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/24973422?tool=bestpractice.bmj.com)

References

1. Pasternack MS, Swartz MN. Cellulitis, necrotizing fasciitis, and subcutaneous tissue infections. In: Bennett JE, Dolin R, Blaser MJ, eds. Mandell, Douglas, and Bennett's principles and practice of infectious diseases. Philadelphia, PA: Elsevier; 2015:1194-215.
2. 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 (https://wjes.biomedcentral.com/articles/10.1186/s13017-018-0219-9) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/30564282?tool=bestpractice.bmj.com)
3. Sartelli M, Coccolini F, Kluger Y, et al. WSES/GAIS/WSIS/SIS-E/AAST global clinical pathways for patients with skin and soft tissue infections. World J Emerg Surg. 2022 Jan 15;17(1):3. Full text (https://wjes.biomedcentral.com/articles/10.1186/s13017-022-00406-2) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/35033131?tool=bestpractice.bmj.com)
4. Diab J, Bannan A, Pollitt T. Necrotising fasciitis. BMJ. 2020 Apr 27;369:m1428.
5. Stevens DL, Bisno AL, Chambers HF, et al. Practice guidelines for the diagnosis and management of skin and soft tissue infections: 2014 update by the Infectious Diseases Society of America. Clin Infect Dis. 2014 Jul 15;59(2):e10-52. Full text (https://academic.oup.com/cid/article/59/2/e10/2895845) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/24973422?tool=bestpractice.bmj.com)
6. Cheng NC, Yu YC, Tai HC, et al. Recent trend of necrotizing fasciitis in Taiwan: focus on monomicrobial Klebsiella pneumoniae necrotizing fasciitis. Clin Infect Dis. 2012 Oct;55(7):930-9. Full text (https://academic.oup.com/cid/article/55/7/930/427026) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/22715175?tool=bestpractice.bmj.com)
7. Carter PS, Banwell PE. Necrotising fasciitis: a new management algorithm based on clinical classification. Int Wound J. 2004 Sep;1(3):189-98. Full text (https://onlinelibrary.wiley.com/doi/10.1111/j.1742-4801.2004.00054.x) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/16722877?tool=bestpractice.bmj.com)

This PDF of the BMJ Best Practice topic is based on the web version that was last updated: Jan 05, 2024. BMJ Best Practice topics are regularly updated and the most recent version of the topics can be found on bestpractice.bmj.com . Use of this content is subject to our disclaimer (. Use of this content is subject to our) . © BMJ Publishing Group Ltd 2024. All rights reserved.

8. Stevens DL, Bryant AE. Necrotizing soft-tissue infections. N Engl J Med. 2017 Dec 7;377(23):2253-65.

9. Kuo YL, Shieh SJ, Chiu HY, et al. Necrotizing fasciitis caused by Vibrio vulnificus: epidemiology, clinical findings, treatment and prevention. Eur J Clin Microbiol Infect Dis. 2007 Nov;26(11):785-92. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/17674061?tool=bestpractice.bmj.com)

10. Markov G, Kirov G, Lyutskanov V, et al. Necrotizing fasciitis and myonecrosis due to Aeromonas hydrophila. Wounds. 2007 Aug;19(8):223-6. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/26110366?tool=bestpractice.bmj.com)

11. Rahim GR, Gupta N, Maheshwari P, et al. Monomicrobial Klebsiella pneumoniae necrotizing fasciitis: an emerging life-threatening entity. Clin Microbiol Infect. 2019 Mar;25(3):316-323. Full text (https://www.clinicalmicrobiologyandinfection.com/article/S1198-743X(18)30420-8/fulltext) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/29787886?tool=bestpractice.bmj.com)

12. Jain D, Kumar Y, Vasishta RK, et al. Zygomycotic necrotizing fasciitis in immunocompetent patients: a series of 18 cases. Mod Pathol. 2006 Sep;19(9):1221-6. Full text (http://www.nature.com/articles/3800639) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/16741524?tool=bestpractice.bmj.com)

13. Neblett Fanfair R, Benedict K, Bos J, et al. Necrotizing cutaneous mucormycosis after a tornado in Joplin, Missouri, in 2011. N Engl J Med. 2012 Dec 6;367(23):2214-25. Full text (http://www.nejm.org/doi/full/10.1056/NEJMoa1204781) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/23215557?tool=bestpractice.bmj.com)

14. Ribes JA, Vanover-Sams CL, Baker DJ. Zygomycetes in human disease. Clin Microbiol Rev. 2000 Apr;13(2):236-301. Full text (http://cmr.asm.org/content/13/2/236.long) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/10756000?tool=bestpractice.bmj.com)

15. Hua C, Urbina T, Bosc R, et al. Necrotising soft-tissue infections. Lancet Infect Dis. 2023 Mar;23(3):e81-94. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/36252579?tool=bestpractice.bmj.com)

16. Hasham S, Matteucci P, Stanley PR, et al. Necrotising fasciitis. BMJ. 2005 Apr 9;330(7495):830-3. [Erratum in: BMJ. 2005 May 14;330(7500):1143]. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/15817551?tool=bestpractice.bmj.com)

17. Pérez-Flecha González M, Muñoz Rodríguez JM, San Miguel Mendez C, et al. Meleney's synergic gangrene. J Gastrointest Surg. 2021 Mar;25(3):849-51.

18. Ord R, Coletti D. Cervico-facial necrotizing fasciitis. Oral Dis. 2009 Mar;15(2):133-41. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/19207484?tool=bestpractice.bmj.com)

19. Cheung JP, Fung B, Tang WM, et al. A review of necrotising fasciitis in the extremities. Hong Kong Med J. 2009 Feb;15(1):44-52. Full text (http://www.hkmj.org/system/files/hkm0902p44.pdf) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/19197096?tool=bestpractice.bmj.com)

This PDF of the BMJ Best Practice topic is based on the web version that was last updated: Jan 05, 2024. BMJ Best Practice topics are regularly updated and the most recent version of the topics can be found on bestpractice.bmj.com . Use of this content is subject to our disclaimer (. Use of this content is subject to our) . © BMJ Publishing Group Ltd 2024. All rights reserved.

REFERENCES

20. Angoules AG, Kontakis G, Drakoulakis E, et al. Necrotising fasciitis of upper and lower limb: a systematic review. Injury. 2007 Dec;38(suppl 5):S19-26. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/18048033?tool=bestpractice.bmj.com)

21. Noor A, Krilov LR. Necrotizing fasciitis. Pediatr Rev. 2021 Oct;42(10):573-5.

22. Sarani B, Strong M, Pascual J, et al. Necrotizing fasciitis: current concepts and review of the literature. J Am Coll Surg. 2009 Feb;208(2):279-88. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/19228540?tool=bestpractice.bmj.com)

23. Centers for Disease Control and Prevention. Active bacterial core surveillance (ABCs) report. Emerging infections program network: group A streptococcus, 2021. Sep 2023 [internet publication]. Full text (https://www.cdc.gov/abcs/reports-findings/surv-reports.html)

24. Atallah NJ, Scherer AK, Alexander NJ, et al. Candida albicans necrotizing fasciitis following elective surgery. Med Mycol Case Rep. 2020 Jun;28:39-41. Full text (https://www.sciencedirect.com/science/article/pii/S2211753920300257?via%3Dihub) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/32420013?tool=bestpractice.bmj.com)

25. Stevens DL, Aldape MJ, Bryant AE. Necrotizing fasciitis, gas gangrene, myositis and myonecrosis. In: Cohen J, Powderly WG, Opal SM, eds. Infectious diseases. Amsterdam, Netherlands: Elsevier; 2017:95-103.e1.

26. Hung TH, Tsai CC, Tsai CC, et al. Liver cirrhosis as a real risk factor for necrotising fasciitis: a three-year population-based follow-up study. Singapore Med J. 2014 Jul;55(7):378-82. Full text (https://sma.org.sg/UploadedImg/files/SMJ/5507/5507a5.pdf) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/25091887?tool=bestpractice.bmj.com)

27. Brown EJ. The molecular basis of streptococcal toxic shock syndrome. N Engl J Med. 2004;350:2093-2094. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/15141050?tool=bestpractice.bmj.com)

28. Llewelyn M, Cohen J. Superantigens: microbial agents that corrupt immunity. Lancet Infect Dis. 2002;2:156-162. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/11944185?tool=bestpractice.bmj.com)

29. Daneman N, Green KA, Low DE, et al. Surveillance for hospital outbreaks of invasive group A streptococcal infections in Ontario, Canada, 1992 to 2000. Ann Intern Med. 2007 Aug 21;147(4):234-41. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/17709757?tool=bestpractice.bmj.com)

30. Keung EZ, Liu X, Nuzhad A, et al. Immunocompromised status in patients with necrotizing soft-tissue infection. JAMA Surg. 2013 May;148(5):419-26. Full text (https://jamanetwork.com/journals/jamasurgery/fullarticle/1686088) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/23677405?tool=bestpractice.bmj.com)

31. Weng TC, Chen CC, Toh HS, et al. Ibuprofen worsens Streptococcus pyogenes soft tissue infections in mice. J Microbiol Immunol Infect. 2011 Dec;44(6):418-23. Full text (https://www.sciencedirect.com/

This PDF of the BMJ Best Practice topic is based on the web version that was last updated: Jan 05, 2024. BMJ Best Practice topics are regularly updated and the most recent version of the topics can be found on bestpractice.bmj.com . Use of this content is subject to our disclaimer (. Use of this content is subject to our) . © BMJ Publishing Group Ltd 2024. All rights reserved.

science/article/pii/S1684118211001332?via%3Dihub) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/21697021?tool=bestpractice.bmj.com)

32. Aronoff DM, Bloch KC. Assessing the relationship between the use of nonsteroidal antiinflammatory drugs and necrotizing fasciitis caused by group A streptococcus. Medicine (Baltimore). 2003 Jul;82(4):225-35. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/12861100?tool=bestpractice.bmj.com)

33. Centers for Disease Control and Prevention. Manual for the surveillance of vaccine-preventable diseases: chapter 17: varicella. May 2018 [internet publication]. Full text (https://www.cdc.gov/vaccines/pubs/surv-manual/chpt17-varicella.html)

34. Bonne SL, Kadri SS. Evaluation and management of necrotizing soft tissue infections. Infect Dis Clin North Am. 2017 Sep;31(3):497-511. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/28779832?tool=bestpractice.bmj.com)

35. Endorf FW, Cancio LC, Klein MB. Necrotizing soft-tissue infections: clinical guidelines. J Burn Care Res. 2009 Sep-Oct;30(5):769-75. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/19692912?tool=bestpractice.bmj.com)

36. American College of Radiology. ACR appropriateness criteria: suspected osteomyelitis, septic arthritis, or soft tissue infection (excluding spine and diabetic foot). 2022 [internet publication]. Full text (https://acsearch.acr.org/docs/3094201/Narrative)

37. Yen ZS, Wang HP, Ma HM, et al. Ultrasonographic screening of clinically-suspected necrotizing fasciitis. Acad Emerg Med. 2002 Dec;9(12):1448-51. Full text (https://onlinelibrary.wiley.com/doi/abs/10.1197/aemj.9.12.1448?sid=nlm%3Apubmed) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/12460854?tool=bestpractice.bmj.com)

38. Gelbard RB, Ferrada P, Yeh DD, et al. Optimal timing of initial debridement for necrotizing soft tissue infection: a practice management guideline from the Eastern Association for the Surgery of Trauma. J Trauma Acute Care Surg. 2018 Jul;85(1):208-14. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/29485428?tool=bestpractice.bmj.com)

39. Fernando SM, Tran A, Cheng W, et al. Necrotizing soft tissue infection: diagnostic accuracy of physical examination, imaging, and LRINEC Score: a systematic review and meta-analysis. Ann Surg. 2019 Jan;269(1):58-65. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/29672405?tool=bestpractice.bmj.com)

40. Wong CH, Khin LW, Heng KS, et al. The LRINEC (Laboratory Risk Indicator for Necrotizing Fasciitis) score: a tool for distinguishing necrotizing fasciitis from other soft tissue infections. Crit Care Med. 2004 Jul;32(7):1535-41. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/15241098?tool=bestpractice.bmj.com)

41. Hsiao CT, Chang CP, Huang TY, et al. Prospective validation of the Laboratory Risk Indicator for Necrotizing Fasciitis (LRINEC) Score for necrotizing fasciitis of the extremities. PLoS One. 2020;15(1):e0227748. Full text (https://journals.plos.org/plosone/article?id=10.1371/journal.pone.0227748) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/31978094?tool=bestpractice.bmj.com)

 © BMJ Publishing Group Ltd 2024. All rights reserved.

REFERENCES

42. Hoesl V, Kempa S, Prantl L, et al. The LRINEC score-an indicator for the course and prognosis of necrotizing fasciitis? J Clin Med. 2022 Jun 22;11(13):3583. Full text (https://www.mdpi.com/2077-0383/11/13/3583) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/35806870?tool=bestpractice.bmj.com)

43. Peetermans M, de Prost N, Eckmann C, et al. Necrotizing skin and soft-tissue infections in the intensive care unit. Clin Microbiol Infect. 2020 Jan;26(1):8-17. Full text (https://www.clinicalmicrobiologyandinfection.com/article/S1198-743X(19)30382-9/fulltext) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/31284035?tool=bestpractice.bmj.com)

44. Murphy G, Markeson D, Choa R, et al. Raised serum lactate: a marker of necrotizing fasciitis? J Plast Reconstr Aesthet Surg. 2013 Dec;66(12):1712-6. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/23911720?tool=bestpractice.bmj.com)

45. Semeraro N, Ammollo CT, Semeraro F, et al. Sepsis-associated disseminated intravascular coagulation and thromboembolic disease. Mediterr J Hematol Infect Dis. 2010 Aug 13;2(3):e2010024. Full text (https://www.mjhid.org/mjhid/article/view/2010.024/155) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/21415977?tool=bestpractice.bmj.com)

46. Konishi H, Foster RG, Follett BK. Evidence for a daily rhythmicity in the acute release of luteinizing hormone in response to electrical stimulation in the Japanese quail. J Comp Physiol A. 1987 Aug;161(2):315-9. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/3625579?tool=bestpractice.bmj.com)

47. Kobayashi L, Konstantinidis A, Shackelford S, et al. Necrotizing soft tissue infections: delayed surgical treatment is associated with increased number of surgical debridements and morbidity. J Trauma. 2011 Nov;71(5):1400-5. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/21768906?tool=bestpractice.bmj.com)

48. Carapetis JR, Jacoby P, Carville K, et al. Effectiveness of clindamycin and intravenous immunoglobulin, and risk of disease in contacts, in invasive group A streptococcal infections. Clin Infect Dis. 2014 Aug 1;59(3):358-65. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/24785239?tool=bestpractice.bmj.com)

49. Kaul R, McGeer A, Norrby-Teglund A, et al. Intravenous immunoglobulin therapy for streptococcal toxic shock syndrome--a comparative observational study. The Canadian Streptococcal Study Group. Clin Infect Dis. 1999 Apr;28(4):800-7. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/10825042?tool=bestpractice.bmj.com)

50. Linnér A, Darenberg J, Sjölin J, et al. Clinical efficacy of polyspecific intravenous immunoglobulin therapy in patients with streptococcal toxic shock syndrome: a comparative observational study. Clin Infect Dis. 2014 Sep 15;59(6):851-7. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/24928291?tool=bestpractice.bmj.com)

51. Darenberg J, Ihendyane N, Sjölin J, et al. Intravenous immunoglobulin G therapy in streptococcal toxic shock syndrome: a European randomized, double-blind, placebo-controlled trial. Clin Infect Dis. 2003 Aug 1;37(3):333-40. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/12884156?tool=bestpractice.bmj.com)

This PDF of the BMJ Best Practice topic is based on the web version that was last updated: Jan 05, 2024. BMJ Best Practice topics are regularly updated and the most recent version of the topics can be found on bestpractice.bmj.com . Use of this content is subject to our disclaimer (. Use of this content is subject to our) . © BMJ Publishing Group Ltd 2024. All rights reserved.

52. Kadri SS, Swihart BJ, Bonne SL, et al. Impact of intravenous immunoglobulin on survival in necrotizing fasciitis with vasopressor-dependent shock: a propensity score-matched analysis from 130 US hospitals. Clin Infect Dis. 2017 Apr 1;64(7):877-85. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/28034881?tool=bestpractice.bmj.com)

53. Hua C, Bosc R, Sbidian E, et al. Interventions for necrotizing soft tissue infections in adults. Cochrane Database Syst Rev. 2018 May 31;(5):CD011680. Full text (https://www.cochranelibrary.com/cdsr/doi/10.1002/14651858.CD011680.pub2/full) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/29851032?tool=bestpractice.bmj.com)

54. Terzian WTH, Nunn AM, Call EB, et al. Duration of antibiotic therapy in necrotizing soft tissue infections: shorter is safe. Surg Infect (Larchmt). 2022 Jun;23(5):430-5. Full text (https://www.liebertpub.com/doi/10.1089/sur.2022.011) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/35451883?tool=bestpractice.bmj.com)

55. Vilar DG, Fadrique GG, Martín IJ, et al. Hypebaric oxygen treatment in urology. Arch Esp Urol. 2011 Jul;64(6):507-16. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/21791717?tool=bestpractice.bmj.com)

56. Menichetti F, Giuliano S, Fortunato S. Are there any reasons to change our behavior in necrotizing fasciitis with the advent of new antibiotics? Curr Opin Infect Dis. 2017 Apr;30(2):172-9. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/28134677?tool=bestpractice.bmj.com)

57. Abrahamian FM, Sakoulas G, Tzanis E, et al. Omadacycline for acute bacterial skin and skin structure infections. Clin Infect Dis. 2019 Aug 1;69(suppl 1):S23-32. Full text (https://academic.oup.com/cid/article/69/Supplement_1/S23/5541769) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/31367742?tool=bestpractice.bmj.com)

58. Lee YR, Burton CE, Bevel KR. Delafloxacin for the treatment of acute bacterial skin and skin structure infections. J Pharm Technol. 2019 Jun;35(3):110-8. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/34861007?tool=bestpractice.bmj.com)

59. Bulger EM, May AK, Robinson BRH, et al. A novel immune modulator for patients with necrotizing soft tissue infections (NSTI): results of a multicenter, phase 3 randomized controlled trial of reltecimod (AB 103). Ann Surg. 2020 Sep 1;272(3):469-78. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/32657946?tool=bestpractice.bmj.com)

60. Centers for Disease Control and Prevention. Group A streptococcal (GAS) disease: type II necrotizing fasciitis. Jun 2022 [internet publication]. Full text (https://www.cdc.gov/groupastrep/diseases-hcp/necrotizing-fasciitis.html)

 © BMJ Publishing Group Ltd 2024. All rights reserved.

Images

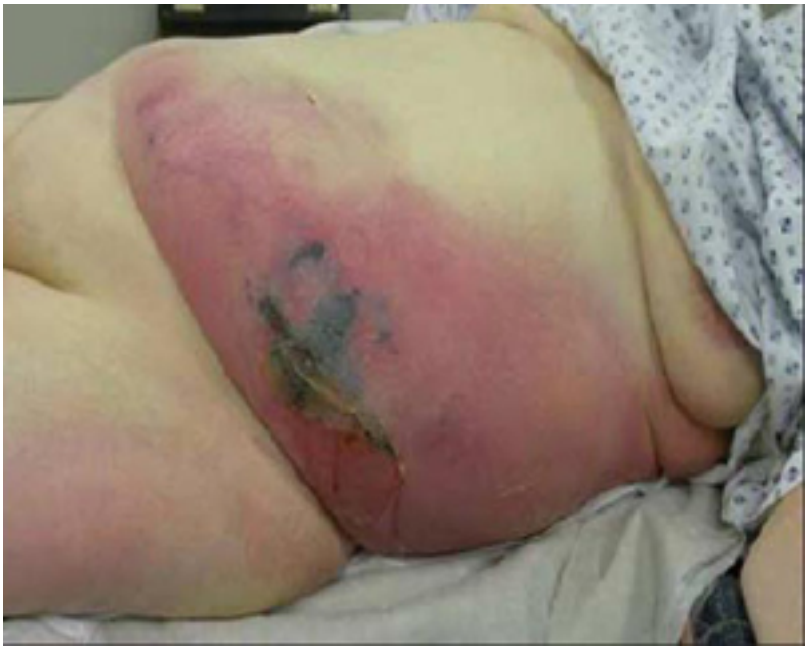


Figure 1: Late signs of necrotizing fasciitis with extensive cellulitis, induration, skin necrosis, and formation of hemorrhagic bullae

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

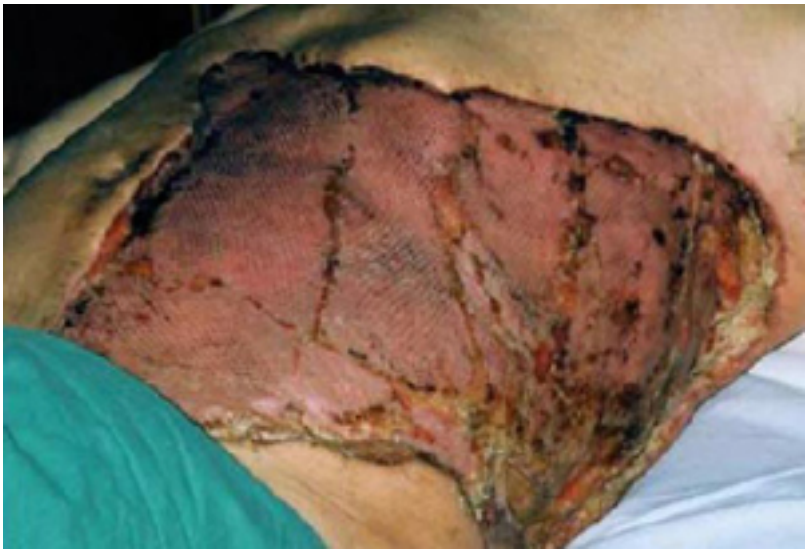


Figure 2: Split thickness skin grafting after surgical debridement

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

This PDF of the BMJ Best Practice topic is based on the web version that was last updated: Jan 05, 2024.
BMJ Best Practice topics are regularly updated and the most recent version of the topics
can be found on bestpractice.bmj.com . Use of this content is subject to our disclaimer (.
Use of this content is subject to our) . © BMJ Publishing Group Ltd 2024. All rights reserved.

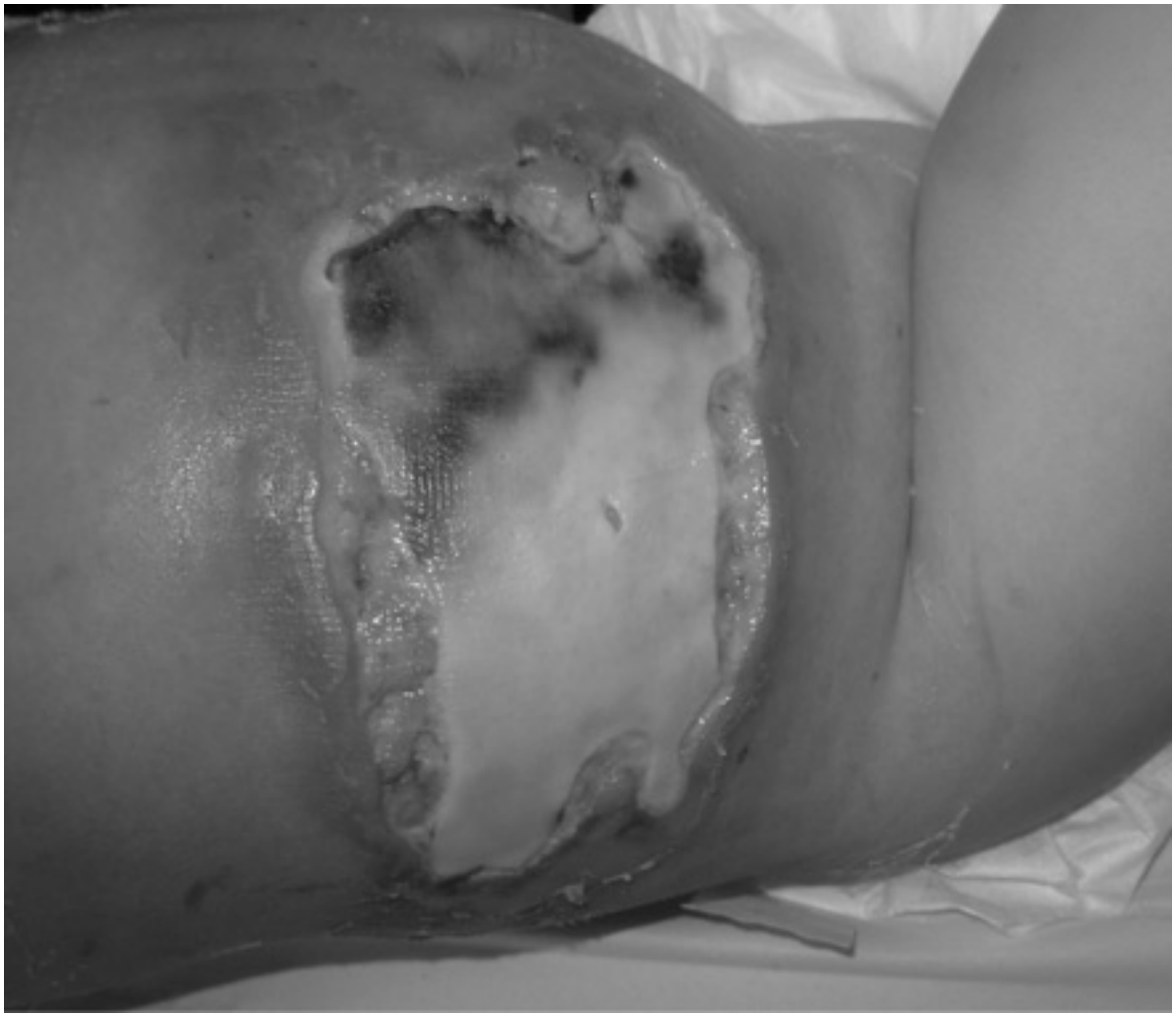


Figure 3: Necrotizing fasciitis on the right abdomen of a 2-year old girl following varicella infection

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

 © BMJ Publishing Group Ltd 2024. All rights reserved.

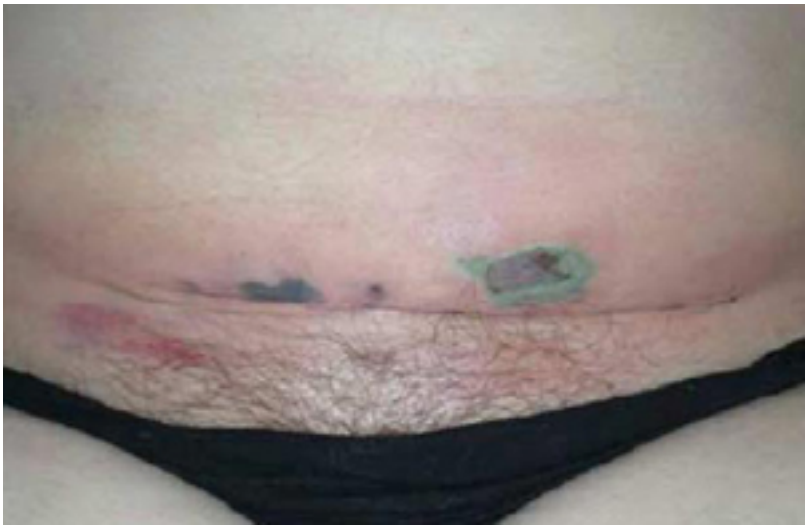


Figure 4: Small areas of skin necrosis in a young woman with cellulitis and necrotizing fasciitis of her lower abdomen 5 days after a cesarean section

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

This PDF of the BMJ Best Practice topic is based on the web version that was last updated: Jan 05, 2024.
BMJ Best Practice topics are regularly updated and the most recent version of the topics
can be found on bestpractice.bmj.com . Use of this content is subject to our disclaimer (.
Use of this content is subject to our) . © BMJ Publishing Group Ltd 2024. All rights reserved.

Disclaimer

BMJ Best Practice is intended for licensed medical professionals. BMJ Publishing Group Ltd (BMJ) does not advocate or endorse the use of any drug or therapy contained within this publication nor does it diagnose patients. As a medical professional you retain full responsibility for the care and treatment of your patients and you should use your own clinical judgement and expertise when using this product.

This content is not intended to cover all possible diagnosis methods, treatments, follow up, drugs and any contraindications or side effects. In addition, since such standards and practices in medicine change as new data become available, you should consult a variety of sources. We strongly recommend that you independently verify specified diagnosis, treatments and follow-up and ensure it is appropriate for your patient within your region. In addition, with respect to prescription medication, you are advised to check the product information sheet accompanying each drug to verify conditions of use and identify any changes in dosage schedule or contraindications, particularly if the drug to be administered is new, infrequently used, or has a narrow therapeutic range. You must always check that drugs referenced are licensed for the specified use and at the specified doses in your region.

Information included in BMJ Best Practice is provided on an “as is” basis without any representations, conditions or warranties that it is accurate and up to date. BMJ and its licensors and licensees assume no responsibility for any aspect of treatment administered to any patients with the aid of this information. To the fullest extent permitted by law, BMJ and its licensors and licensees shall not incur any liability, including without limitation, liability for damages, arising from the content. All conditions, warranties and other terms which might otherwise be implied by the law including, without limitation, the warranties of satisfactory quality, fitness for a particular purpose, use of reasonable care and skill and non-infringement of proprietary rights are excluded.

Where BMJ Best Practice has been translated into a language other than English, BMJ does not warrant the accuracy and reliability of the translations or the content provided by third parties (including but not limited to local regulations, clinical guidelines, terminology, drug names and drug dosages). BMJ is not responsible for any errors and omissions arising from translation and adaptation or otherwise.Where BMJ Best Practice lists drug names, it does so by recommended International Nonproprietary Names (rINNs) only. It is possible that certain drug formularies might refer to the same drugs using different names.

Please note that recommended formulations and doses may differ between drug databases drug names and brands, drug formularies, or locations. A local drug formulary should always be consulted for full prescribing information.

Treatment recommendations in BMJ Best Practice are specific to patient groups. Care is advised when selecting the integrated drug formulary as some treatment recommendations are for adults only, and external links to a paediatric formulary do not necessarily advocate use in children (and vice-versa). Always check that you have selected the correct drug formulary for your patient.

Where your version of BMJ Best Practice does not integrate with a local drug formulary, you should consult a local pharmaceutical database for comprehensive drug information including contraindications, drug interactions, and alternative dosing before prescribing.

Interpretation of numbers

Regardless of the language in which the content is displayed, numerals are displayed according to the original English-language numerical separator standard. For example 4 digit numbers shall not include a comma nor a decimal point; numbers of 5 or more digits shall include commas; and numbers stated to be less than 1 shall be depicted using decimal points. See Figure 1 below for an explanatory table.

BMJ accepts no responsibility for misinterpretation of numbers which comply with this stated numerical separator standard.

This approach is in line with the guidance of the International Bureau of Weights and Measures Service.

Figure 1 – BMJ Best Practice Numeral Style

 © BMJ Publishing Group Ltd 2024. All rights reserved.

5-digit numerals: 10,000

4-digit numerals: 1000

numerals < 1: 0.25

Our full website and application terms and conditions can be found here: Website Terms and Conditions.

Contact us

+ 44 (0) 207 111 1105
support@bmj.com

BMJ
BMA House
Tavistock Square
London
WC1H 9JR
UK

DISCLAIMER

This PDF of the BMJ Best Practice topic is based on the web version that was last updated: Jan 05, 2024. BMJ Best Practice topics are regularly updated and the most recent version of the topics can be found on bestpractice.bmj.com . Use of this content is subject to our disclaimer (. Use of this content is subject to our) . © BMJ Publishing Group Ltd 2024. All rights reserved.

Contributors:

// Authors:

Ramia Zakhour, MD

Assistant Professor
Department of Pediatrics, University of Texas, McGovern Medical School, Houston, TX
DISCLOSURES: RZ declares that they have no competing interests.

// Acknowledgements:

Dr Ramia Zakour would like to gratefully acknowledge Dr Kevin Steiner and Dr William Petri previous contributors to this topic.
DISCLOSURES: KS and WP declared they have no competing interests.

// Peer Reviewers:

Felix Lui, MD, FACS

Associate Professor of Surgery
Yale School of Medicine, New Haven, VT
DISCLOSURES: FL declares that he has no competing interests.

Shiranee Sriskandan, MA, MBBChir, FRCP, PhD

Professor of Infectious Diseases and Hon. Consultant
Section of Infectious Diseases, Imperial College London, London, UK
DISCLOSURES: SS declares that she has no competing interests.