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Abstract: Necrotizing fasciitis (NF) is a severe bacterial infection that attacks subcutaneous fat tissues, superficial fascia, deep fascia, and muscle. NF is a rare condition with a mortality that requires nurse practitioners to be adept at identifying signs and symptoms to prompt a quick diagnosis and treatment.

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Necrotizing fasciitis (NF) is a severe bacterial infection that attacks subcutaneous fat tissues, superficial fascia, deep fascia, and muscle.[1] According to the CDC, "necrotizing" is defined as "causing the death of tissues."[2] Causative bacteria multiply releasing toxins and enzymes, which creates thrombosis in blood vessels that quickly progress to massive destruction of soft tissues and fascia.[3,4] NF spreads rapidly; in a matter of hours to days, the infection can progress from a seemingly benign skin lesion (often mistaken for a spider or insect bite) to a highly lethal disease.[5]

NF is a rare condition with a mortality between 24% and 34% and as high as 40% to 70% in patients with diabetes.[6-9] In 1996, the CDC estimated that of the 500 to 1,500 cases of NF reported, 20% were fatal.[1] The incidence of NF due to Group A *Streptococcus* (GAS) is rising, but researchers have not been able to pinpoint a root cause for escalating rates.[5] In 2010, Olsen and Musser estimated as many as 1,500 to 3,000 cases occurred annually.[5] Nationally, in 2010, the CDC reported 12,500 cases and an estimated 1,250 deaths attributed to GAS-related NF.[10] In total, NF is fatal in approximately 38% to 50% of adults and 25% of children.[11,12]

Cost of disease

Onset of acute illnesses such as NF can have negative effects on family finances and have an even larger impact on uninsured patients.[13] NF is primarily an acute illness, although it can lead to chronic, devitalizing consequences and a need for continued healthcare. Postinfection deformity can have considerable economic effects on the patient.[14-16] The loss of a limb could prevent return to a specific profession, or work in general, and place a substantial financial burden on the patient.[14,15]

Risk factors

Most patients who are diagnosed with NF have comorbidities that may lower their body's ability to fight infection.[2] Risk factors for NF include, but are not limited to, diabetes mellitus, peripheral vascular disease, varicella infection, alcohol use, impaired kidney function, malnutrition, and the use of nonsteroidal anti-inflammatory drugs due to their possible impairment of lymphocytic function.[17] Precipitating events include traumatic injury, surgery, soft-tissue infections, minor invasive procedures (joint aspirations, acupuncture), burns, childbirth, or a penetrating injury (insect and animal bites).[17] Although there are known risk factors and precipitating events, this fatal disease can also be idiopathic.[6,18]

Categories/pathology

NF can be caused by different types of bacteria and divided into two clinical types based on bacterial origin. Any site of the body can become infected with NF, but it is most commonly seen in the lower extremities, abdomen, perianal area, groin, and postoperative surgical sites.[19]

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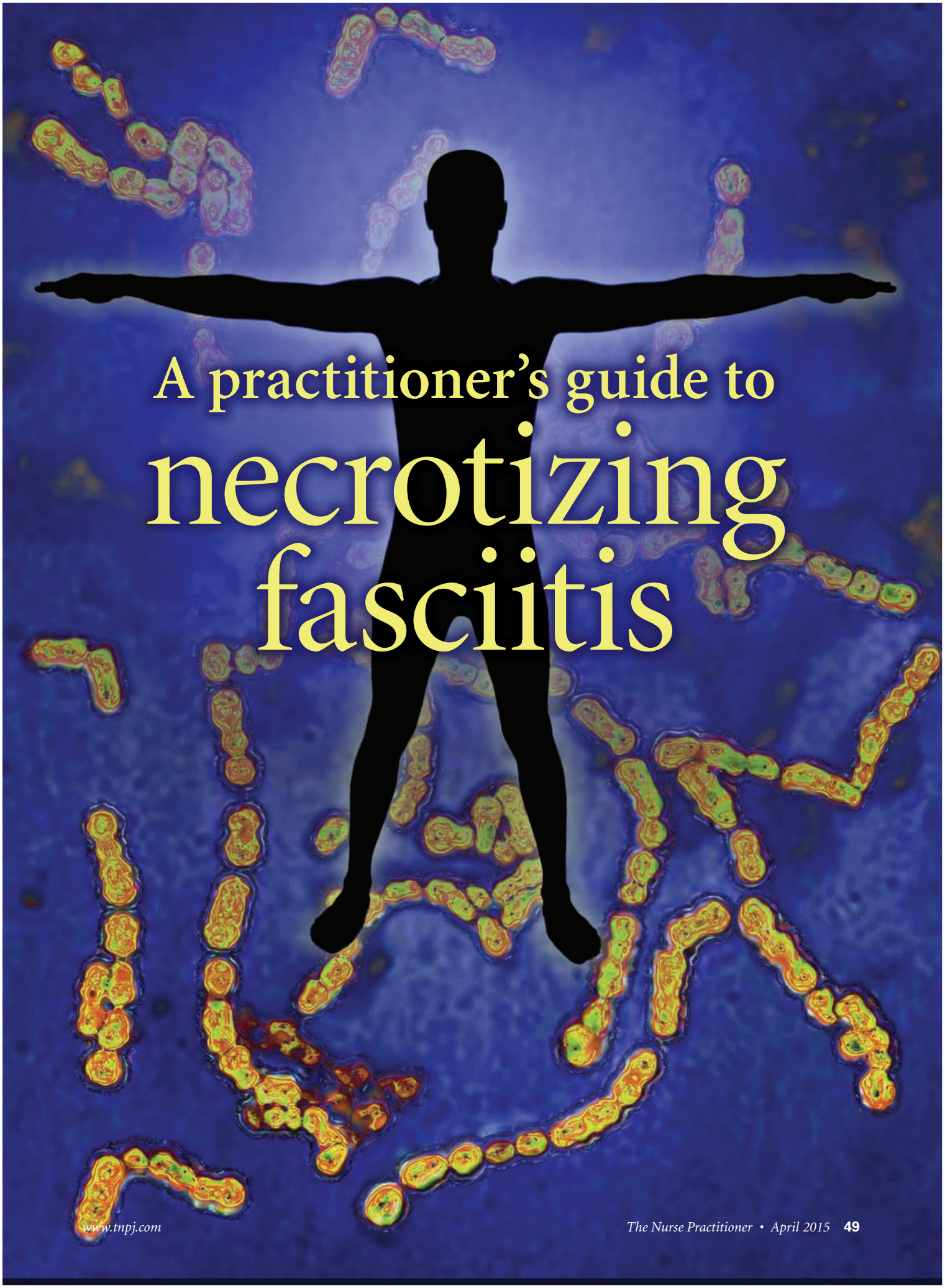
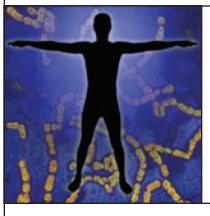
Keywords: bacterial infection, Fournier gangrene, group A *Streptococcus*, necrotizing fasciitis, nongroup A *Streptococcus*



A practitioner's guide to necrotizing fasciitis

Bacteria penetrate into the subcutaneous tissue, track into the superficial fascia, and migrate into the soft tissue and muscle planes.[19,20] As a result of their metabolism, anaerobic and aerobic organisms produce toxins.[14,21] These toxins accumulate in soft tissues, due to reduced water solubility, producing subcutaneous gas formation and crepitus.[14,21] Once the tissues begin to die, circulation ceases in that area; therefore, tissues are deprived of needed oxygen. Facultative aerobic organisms grow as a result of decreased polymorphonuclear (PMN) leukocyte function under hypoxic wound conditions.[20] Subsequent abundance of organisms further lowers the oxidation/reduction potential, leading to an even greater proliferation of anaerobic organisms and progression to disease.[14,21] Moreover, toxins and enzymes released by the bacteria involved further facilitate spread of the organism along superficial and deep fascia, resulting in a deep-seeded infection with vascular occlusion, superficial nerve damage, ischemia, and tissue necrosis.[14,21]



Necrotizing fasciitis is difficult to diagnose in its initial and advanced stages as it can mimic other diseases.

Type 1, polymicrobial, NF is caused by a mixture of nongroup A streptococcal (non-GAS) anaerobes, aerobes, and facultative anaerobes, such as staphylococci, enterococci, and enterobacteriaceae.[3,14,22,23] Type 1 NF occurs most commonly after a surgical procedure or in patients with coexisting comorbidities, such as diabetes mellitus, peripheral vascular disease, or other causes of immunosuppression.[14] Toxins and adhesion proteins produced by the bacteria play an important role in the disease process.[19]

Fournier gangrene is an aggressive variant of type 1 NF that affects the genital, perianal, and perineal regions with potential for extension into the abdominal wall between fascial planes.[14,24,25] The cause of infection is identifiable in 95% of cases, mainly arising from anorectal, genitourinary, and cutaneous sources.[24] Infection can spread rapidly to gluteal muscles, the anterior abdominal wall, and, in males, the scrotum and penis.[14,25] Another variant of type 1 NF is saltwater NF, which occurs when a minor skin wound becomes contaminated with saltwater-containing *Vibrio* species.[14,26] *Vibrio*, which is a bacteria living in warm waters, can also result in contamination through ingestion of infected seafood.[26] *Vibrio* infection has a high incidence of septicemia development and is often fatal, carrying a mortality of 30% to 40%.[22]

Type 2, Gram-positive, monomicrobial, NF is usually caused by bacteria, such as a GAS and *Staphylococcus aureus* in the absence of comorbities.[3,6,14,23] GAS is considered the most common cause of NF and is also the cause of strep throat.[2,27] As GAS organisms proliferate in the area of entry, there is rapid influx of acute inflammatory cells.[5] Loose organization of fibrous connective tissue and neurovascular structures within the superficial and deep fascial planes pose little anatomic barrier to local dissemination.[5] The combined action of many potent protease and other degradative virulence factors expressed by invading GAS organisms and tissue-damaging enzymes released by host PMN leukocytes results in severe tissue damage.[5] As the infection progresses, fascial sheaths are breached, and nutrient-rich skeletal muscle fibers are exposed.[5] The severe tenderness and pain a patient experiences initially might later diminish due to subsequent nerve damage, leading to localized anesthesia of the affected area.[28] Progression of NF to include widespread muscle cell death is termed necrotizing myositis and is associated with a poor prognosis.[5]

Although GAS has been noted to be the most common cause of NF, a resistant form of staphylococcus, methicillin-resistant *Staphylococcus aureus* (MRSA), is on the rise, causing growing concern due to its antibiotic resistance.[29] According to results of a Taiwan research study conducted during 2004 to 2008, patients with MRSA infection had more severe clinical outcomes than those affected by any other microbe.[30] Moreover, of the 247 cases of NF reviewed, 42 microbial species were identified; staphylococcus was the major prevalent pathogen with MRSA accounting for 19.8% of all cases reviewed.[30]

There are many different types of bacteria and organisms that can cause NF, but most function similarly by attacking tissue and decreasing immune response through production of toxins that block the body's defense mechanisms.[29] M protein, an important virulence factor of GAS, produces pyrogenic exotoxins that act as super-antigens, causing a rapid proliferation of T cells.[14,31] Bacterial factors M-1 and M-3 surface proteins also increase the adherence of streptococci to tissues and protect bacteria from phagocytosis by neutrophils.[20] The T cells stimulate the host's immune system resulting in production of cytokines, such as TNF-a, IL-1, and IL-6. This in turn causes over activation of the host's immune system, leading to chills, fever, hypotension, shock, tissue destruction, and possible organ failure.[14,19,31]

Differential diagnosis

NF is very difficult to diagnose in its initial and advanced stages, as it mimics other diseases such as cellulitis,

Case study

A 52-year-old Asian male presented to the clinic with an initial complaint of pain and swelling in the left 4th digit of his hand for approximately 2 weeks. The patient stated he worked as a shrimper for a local company. Two weeks ago, he received a minor cut when cleaning out his shrimp nets. Over time, he began to experience pain that progressively worsened and is now intolerable. Additional history revealed diabetes currently under control with oral diabetes agents. The patient also mentioned his glucose had been higher than normal over the past week, which he attributed to stress.

Vital signs on admission to the hospital revealed a temperature of 100.8° F (38.2° C), pulse of 112, and BP of 98/64. Physical exam revealed an enlarged, erythematous left 4th digit extremely sensitive to even minor palpation. A small amount of purulent drainage was noted on the dorsal surface of the finger where the patient had cut himself.

Lab results revealed an elevated WBC count of 26,000 cells/mm^3 with 8% bands (left shift) and an elevated plasma lactic acid of 3.4 mmol/L. His plasma blood glucose was elevated at 256 mg/dL as was his C-reactive protein of 152 mg/L. All other labs were normal. Based on a LRINEC score of 7, the NP suspected a high probability of NF. Drainage was obtained from the site and sent to the lab for culture and sensitivity. Plain radiography of the involved area revealed a moderate amount of visible gas concentrated at the base of the phalanx and extending distally in smaller strands.

The patient was immediately started on a regimen of I.V. ciprofloxacin secondary to a strong clinical suspicion for *Vibrio vulnificus* infection. The orthopedic hand surgeon was consulted and the patient was taken directly to surgery for debridement as soon as radiographs were reviewed. Once initial debridement was complete, the patient was admitted to the postoperative unit and continued on an I.V. regimen of ciprofloxacin and pain medication. Oral diabetes agents were discontinued and the patient was placed on subcutaneous insulin to better control his glucose levels while in the hospital. Multiple attempts at debridement were performed over the course of the next few days. Culture and sensitivity results confirmed clinical suspicion for *Vibrio vulnificus* and sensitivity to ciprofloxacin. A wound care specialist was also consulted and hyperbaric oxygen therapy was included in the plan of care.

Despite all efforts to save the finger, risk of a spreading infection became too great and the patient, family, wound care specialist, and surgeon agreed amputation was the best course of action. Once the amputation was performed, care was eventually transitioned from the orthopedic hand surgeon to the patient's primary care provider and the wound care specialist. Once discharged from the hospital, outpatient care consisted of continued hyperbaric oxygen therapy sessions 5 days per week for 2 weeks and twice weekly office visits to his primary care provider. The patient resumed his oral diabetes agents for diabetes control at home and completed 14 days of an oral ciprofloxacin regimen. The patient experienced no postoperative complication and has resumed his normal activities.

erysipelas, pyoderma gangrenosum, gas gangrene, compartment syndrome, deep vein thrombosis (DVT), and osteomyelitis with soft-tissue involvement.[14,32-34] Diagnosing NF should be aggressively pursued because a delay may allow the disease to progress to later, more critical stages before appropriate antibiotics and surgical intervention are initiated.[35-37]

Clinical presentation

Symptoms of NF often start within hours after an injury or spontaneously.[2] Nausea, fever, diarrhea, dizziness, general malaise, and pain in the area of localized inflammation may or may not be present in affected individuals.[3,32] In a matter of hours to days, NF can cause altered consciousness, necrosis of tissues, dyspnea, hypotension, and fatal shock syndrome.[19,23] Although a diagnosis of NF may not be presumptively made initially based upon a person's history, physical exam findings, lab tests, and imaging studies, the attending nurse practitioner (NP) should have a high index of suspicion; this is the first and most important tool for early diagnosis of NF.[33,38]

During the first 24 to 48 hours, the patient may present with fever, chills, localized pain, swelling, and erythema that mimic cellulitis, erysipelas, osteomyelitis, or DVT; the necrotizing infection is deep within the skin and not visible.[32,28] Those infected with NF may also initially complain of pain or soreness, similar to that of a "pulled muscle."[2]

Continual progression of untreated or misdiagnosed NF results in the formation of ulcers, blisters, and black spots within 2 to 4 days.[1] As time continues to lapse without treatment, bullae form indicating skin ischemia, which may become hemorrhagic.[28,32] Skin becomes discolored, gray, and necrosed; subcutaneous tissue eventually breaks down and transforms into a hard wooden texture.[32] The formation of bullae and apparent necrosed skin should rule out the diagnosis of a severe case of cellulitis, and support the diagnosis of NF.[28,32] Pain sensation may progress from intense tenderness to anesthesia as the nerves are destroyed.[28,32] The NP may also assess crepitus resulting from subcutaneous gas formation.[28,32] (*See Case study*.)

Diagnosis

Notably, a variety of cutaneous findings can slow down and/or complicate the process of achieving an accurate diagnosis of NF. Imaging studies such as magnetic resonance imaging scans, computed tomography scans, and ultrasounds

have all been used successfully to identify cases of NF.[38,39] These tests may assist in identifying areas of fluid collection, inflammation, and gas within a person's soft tissues in addition to helping delineate the extent of infection.[38,39] Nonetheless, obtaining imaging studies should not delay definitive treatment in those cases highly suggestive of NF.[38,40] The diagnosis of NF is clinical: if there is strong clinical suspicion of NF, exploratory surgery is required regardless of test results, and the NP should consult the surgeon immediately.[32] Some surgeons may attempt the "finger test" prior to resorting to surgery, which can be done surgically in the OR or as a bedside procedure.[32]

After application of local anesthetic, a 2-cm incision is made down to the deep fascia, and a gloved index finger is used to probe at the level of the deep fascia. A positive finger test is signified by lack of resistance of tissues to blunt dissection, lack of bleeding secondary to thrombosed vessels, gray necrotic tissue, "dishwater" purulent drainage, and noncontracting muscle; all suggest a diagnosis of NF.[32] Rapid frozen section, tissue biopsies can be sent, but most practitioners advocate immediate debridement.[8] Without prompt action, the patient will develop hypotension, decreased level of consciousness, and septicemia may develop.[28,32]

Patients presenting with an unexplained pain out of proportion and/or an anesthesia effect (insensitivity to pain) to the affected area may be exhibiting one of the first clinical findings of NF. In addition to history, physical assessment, and diagnostic imaging, the NP must explore serum analysis by obtaining a complete blood cell count, serum electrolytes, renal indexes, serum creatine kinase (CK), serum C-reactive protein as well as blood and tissue cultures. Clinical findings warranting immediate intervention, such as admission or transfer to a higher level of care initiated by the NP include, but are not limited to hypotension, elevated creatinine, elevated CK, elevated white blood cell (WBC) count with marked left shift, and/or low serum bicarbonate, and/or elevated lactic acid level. A simple and objective scoring system is available to assist the NP with differentiating NF from other less-severe, soft-tissue infections. The Laboratory Risk Indicator for Necrotizing Fasciitis (LRINEC) provides an elementary scoring system to assist with identification for high suspicion of NF (see *Laboratory Risk Indicator for Necrotizing Fasciitis*).[7]

Laboratory Risk Indicator for Necrotizing Fasciitis

Variable	Value	Score
C-reactive protein (mg/L)	<150	0
	≥150	4
WBC count (per mm^3)	<15	0
	15-25	1
	>25	2
Hemoglobin (g/dL)	>13.5	0
	11-13.5	1
	<11	2
Sodium (mmol/L)	≥135	0
	<135	2
Creatinine (micromol/L)	≤141	0
	>141	2
Glucose (mmol/L)	≤10	0
	>10	1

LRINEC score ≥ 6 should raise suspicion for NF, and a score ≥ 8 is strongly predictive of the disease. The maximum score is 13. To convert the glucose value to mg/dL, multiply by 18.015. To convert the creatinine value to mg/dL, multiply by 0.01131.
Source: Wong CH, Khin LW, Heng KS, Tan KC, Low CO. The LRINEC (Laboratory Risk Indicator for Necrotizing Fasciitis) score: A tool for distinguishing necrotizing fasciitis from other soft tissue infections. *Crit Care Med.* 2004;32(7):1535-1541. Used with permission.

Medical management

Initial management of NF symptoms include I.V. fluids, supplemental oxygen, cardiac monitoring, and assisted ventilation if applicable.[38] Vasopressors, blood transfusions, I.V. immunoglobin, and a hyperbaric oxygen chamber are sometimes used in cases involving mixed bacterial infection.[1] Successful, definitive treatment of NF involves removing all necrotic tissue, immediate initiation of I.V. antibiotic coverage for a wide array of organisms, and support of failing organs.[8,38] Commonly recommended antibiotics include penicillin, linezolid, vancomycin, clindamycin, carbapenems, metronidazole, cephalosporins, and aminoglycosides.[23,38] A majority of clinicians choose to treat with more than one antibiotic due to bacterial resistance seen in many of the bacteria responsible for NF. Additionally, many NF infections are caused by more than one bacterial genus.[23,38] To adequately treat resistant bacteria such as MRSA, antibiotic coverage can be adjusted after culture results identify the causative organism(s) and antibiotic sensitivity results become available.[23,38]

The most important determinant of mortality is timing and adequacy of initial debridement.[8] Wide and deep incisions might be necessary to excise all infected tissue until healthy tissue is visualized.[38,41] Skin edges should be healthy and free from cellulitis and bleeding. The amount of debridement is often much larger than is appreciated on physical exam, as the infection tracks along the fascia.[8] Repeated surgical debridement is necessary within the ensuing hours to days after initial surgical intervention because progression of the disease might be sudden, severe, and unrelenting.[38,41] Continual, vigilant assessment of the

patient with NF performed by the NP could assist in arresting progression of the disease and potentially prevent amputation.

Amputation is needed in approximately 26% of extremity NF cases, either to gain control of ascending infection or to remove a functionless limb when large volumes of muscle have been debrided.[8] Septicemia could potentially lead to other infection sites, which may also require surgical intervention, resulting in multiple amputations for some patients.[38] In some instances (despite repeated surgical debridement), a life-saving amputation may be required if the necrosis is too widespread and there is imminent risk of overwhelming septicemia.[38] Legs, hands, fingers, toes, and arms have all been sacrificed to save the life of NF patients.[1]

Wounds are usually left open and treated with simple dressings and the use of vacuum-assisted closure devices. Skin grafting is commonly required once the wound is clean and granulated. Reconstruction with full-thickness, free, or rotational flaps is sometimes necessary.[8] If less than 25% of the body surface area is involved, skin grafts and flaps can provide adequate coverage. Otherwise, alternative skin grafting measures should be considered, such as artificial skin and acellular dermal matrix.

Management of NF usually requires a multidisciplinary approach involving surgeons, infectious-disease specialists, pathologists, critical care specialists, wound care specialists, as well as others to provide comprehensive care.[38] Patients who survive NF often require extensive follow up with these specialists. Psychological intervention is needed for those who might experience anxiety, depression, or additional psychological repercussions.[38,42] It is important for NPs to understand that this is not merely about pathology or a diagnosis beyond this point. The individual and family develop their own meanings and perceptions of the condition and need the opportunity to accept and cope with the illness.[42]

Moving forward

NF is an emergent condition requiring immediate evaluation, diagnosis, and surgical evaluation and/or intervention to prevent the continued loss of affected tissue and surrounding organs. The prognosis for patients with NF depends on a number of factors, including the patient's age, causative organisms, any underlying comorbid medical conditions, location, extent of the infection, and time from diagnosis to initiation of treatment.[38] Early diagnosis of NF is critical for improved outcomes: It should be suspected in individuals who present with unexplained pain out of proportion and/or accompanied soft-tissue anesthesia. A delay in diagnosis can lead to higher morbidity and mortality.[5] Acquisition of knowledge to rapidly recognize and diagnosis possible NF based on presenting clinical signs and symptoms is a necessity for NPs and may ultimately decrease the severe consequences of NF.

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INSTRUCTIONS

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