Review Article

Necrotizing Infections of the Hand and Wrist: Diagnosis and Treatment Options

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Abstract

Necrotizing infections of the hand and wrist are important clinical entities because of their rapidly progressive and potentially lethal nature. These infections encompass a spectrum of diseases with overlapping signs and symptoms, which can be subtle and nonspecific. If the brief prodromal period of these infections goes unrecognized, a local area of devitalized tissue can evolve into fulminant infection, multiorgan failure, and potentially death. Early recognition and treatment including administration of broad-spectrum antibiotics and surgical débridement are paramount to improving patient outcomes.

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None of the following authors or any immediate family member has received anything of value from or has stock or stock options held in a commercial company or institution related directly or indirectly to the subject of this article: Dr. Choueka and Dr. De Tolla.

J Am Acad Orthop Surg 2020;28: e55-e63

DOI: 10.5435/JAAOS-D-17-00716

Necrotizing infections of the hand and wrist represent a spectrum of rare limb and life-threatening soft-tissue infections involving the skin, subcutaneous tissue, fascia, and muscle. The term necrotizing fasciitis (NF) is often used interchangeably with necrotizing soft-tissue infection (NSTI) in the literature and classically describes an infection that spreads rapidly along fascial planes causing necrosis of the surrounding tissues with relative sparing of the muscle.[1] Anaya et al encouraged the use of the term necrotizing soft-tissue infections over other terms (ie, hospital gangrene, necrotizing erysipelas, streptococcal gangrene, and suppurative fasciitis) because it encompasses all forms of this disease process regardless of the level of involvement or microbiological entity.[2] All NSTIs use the same general diagnostic and treatment algorithm and rely on prompt recognition to improve patient outcomes.[3] A multidisciplinary team approach including surgeons of various specialties, critical care physicians, infectious disease physicians, and supportive services is required in the treatment of these patients.

Approximately 1,000 cases of NSTIs occur annually in the United States; but a concern is that the incidence is on the rise.[4] In a large case series, the most common site of infection was the extremities, accounting for 57.8% of NSTIs.[3] NSTIs of the extremities have been shown to have a higher overall mortality rate compared with infections of the abdomen and perineum.[3] Espandar et al prospectively studied NSTI in the extremities of 24 patients and found NSTI to be more common in the lower extremity (67.5%) than in the upper extremity (37.5%). Although less common, NSTI of the upper extremity had a similar mortality rate to NSTI of the lower extremity (20% versus 22%).[5] Yeung et al[6] looked at factors affecting mortality in patients with upper limb NSTI and found that infections of the forearm and wrist resulted in greater mortality than infections at the digital level. Infections that began at the dorsum of the hand and wrist were noted to be rapidly progressive secondary to the

continuity of the fascial planes of the wrist and forearm.[6]

NSTI is one of the few surgical emergencies in upper extremity surgery that can result in the loss of limb or life. Patients typically present with nonspecific signs and symptoms of swelling, erythema, and pain that are often initially difficult to distinguish from cellulitis or abscess formation.[4] When left untreated, the infection spreads proximally causing necrosis of the fascia and subcutaneous tissues, leading to sepsis and ultimately multiorgan failure. Knowledge of the complex anatomy and potential spaces of the hand and wrist along with a high index of suspicion are essential to the timely diagnosis of these infections. This phenomenon is particularly important in immunocompromised patients such as those on immunosuppressive medications and those with a history of diabetes, HIV, or intravenous (IV) drug use, who are at increased risk of necrotizing infection.[7] This article provides an evidence-based review of the diagnosis and treatment of necrotizing infections of the hand and wrist.

Diagnosis

Clinical Signs and Symptoms

The clinical diagnosis of NSTI of the upper extremity can be challenging secondary to the complexity and density of anatomic structures in this region. Although NSTI commonly occurs after local trauma, the trauma can be benign or remote. The early stages of NSTI are characterized by nonspecific findings such as localized edema and erythema. These subtle findings can be difficult to differentiate from indolent soft-tissue infections of the hand and wrist such as cellulitis or abscess.[6] In apparent indolent soft-tissue infections, careful monitoring is warranted to identify cases demonstrating the potential for rapid deterioration into a more fulminant condition.

Wong et al described the cutaneous manifestations of NSTI in three stages (Table 1).[8] Stage 1 is characterized by local erythema, swelling, and poorly localized pain. The skin may appear normal while underlying fascial ischemia and necrosis lead to rapid bacterial dissemination and excruciating pain.[9] A key to early diagnosis of NSTI at this stage is recognizing the tenderness to palpation these patients experience is out of proportion to examination findings and extends beyond the margins of the involved skin. Stage 2 (Figure 1, A and B) is characterized by serous blisters and bullae formation. Stage 3 findings include hemorrhagic bullae, crepitus, skin anesthesia, and necrosis, which are pathognomonic of necrotizing infections. Late cutaneous findings occur in conjunction with signs and symptoms of septic shock and multiorgan failure.

NSTI presents in various forms. The fulminant, hyperacute form is characterized by a rapid clinical course with deterioration into septic shock and multiorgan failure in a matter of hours. In these cases, the extent of infection is often not appreciated until surgical débridement because the pathognomonic cutaneous signs do not have a chance to develop. The skin may look deceptively normal despite toxic systemic manifestations.[8] A more subacute form can also occur during which the course is spread out over days to weeks.[4]

The literature stresses that systemic manifestations provide an important clue to the early diagnosis of NSTI. However, in NSTI of the upper extremity, systemic manifestations are relatively uncommon,[6] which may be related to initial treatment with broad-spectrum antibiotics at the primary care level resulting in a reduction in the bacterial load.[6] Also, diabetic and IV drug use patients who are at higher risk of these infections often have blunted immune responses and may not mount the response one would expect in the presence of a necrotizing infection.[8]

Table 1

Clinical Features of NSTI as the Disease Progresses Through Clinical Stages

Stage 1 (Early)	Stage 2 (Intermediate)	Stage 3 (Late)
Tenderness to palpation (extending beyond the apparent area of skin involvement)	Blister/bullae (serous fluid)	Hemorrhagic bullae
Erythema	Skin fluctuance	Skin anesthesia
Swelling	Skin induration	Crepitus
Warmth		Skin necrosis with dusky discoloration progressing to frank gangrene

NSTI = necrotizing soft-tissue infection

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Diagnostic Tests

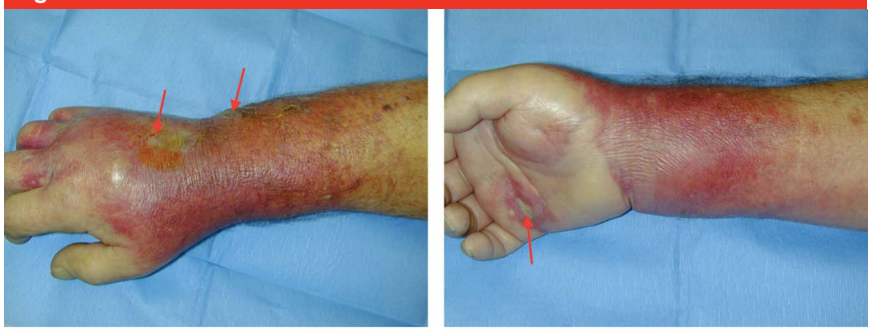
The diagnosis of NSTI is generally made clinically and later corroborated with intraoperative findings and frozen section. Laboratory and radiologic studies can aid in confirming the clinical diagnosis and providing important diagnostic and prognostic information, however should never delay early treatment of this life-threatening disease.

The LRINEC score (Laboratory Risk Indicator for Necrotizing Fasciitis) was developed with the goal of assisting practitioners in differentiating NSTI from other soft-tissue infections based on laboratory findings[10] (Table 2). It has also been used as a tool to risk-stratify these patients. Although its role as a diagnostic adjunct for upper extremity soft-tissue infections has not been well established, it is easily obtained from routine blood testing.[11]

The scoring system uses C-reactive protein level, total white blood cell count, hemoglobin level, sodium level, creatinine level, and glucose level. Patients with a LRINEC score of greater than or equal to 6 should be carefully evaluated for the presence of NF.[10] The authors who developed this scoring system found a positive predictive value of 92% and a negative predictive value of 96% for a score of greater than or equal to six. Risk stratification was divided into low (<50% for a score less than or equal to 5), moderate (50% to 75% for a score of 6 to 7), and high (>75% for a score greater than or equal to 8).

Chauhan et al[12] reviewed several studies that applied the LRINEC score and identified that this scoring system is better at ruling out NSTI than ruling it in. A recent retrospective review case series showed that the score was helpful for diagnosis but did not correlate markedly with disease severity or outcome.[9] Ultimately, surgical intervention should not be delayed in the setting of a low LRINEC score if clinical suspicion of NSTI exists.

Figure 1



A B

A, Photograph showing stage 2 NSTI of the dorsal aspect of the hand and wrist. Swelling and erythema has progressed to serous blister/bullae formation (arrows). **B**, Photograph of the stage 2 NSTI of the volar aspect of the hand and wrist. Fluctuance and induration is noted in the palm (arrow). NSTI = necrotizing soft-tissue infection

Imaging

Imaging studies can assist in the diagnosis of NSTI of the hand and wrist in equivocal cases. However, surgical intervention should never be delayed when waiting for advanced imaging if one has a high clinical suspicion of NSTI. Ultrasonography is preferred by Leiblein et al[9] because of its accessibility. On ultrasonography, hypoechoic fluid between the muscle and subcutaneous tissue and hyperechoic gas adjacent to the fascia can indicate fascial necrosis.

Plain radiographs in early disease are often normal, though may show findings similar to cellulitis such as increased opacity and thickness of the soft tissues.[13] Characteristic findings such as air in the subcutaneous tissues or tracking along the fascial planes due to gas-producing organisms may not manifest until advanced disease and should not be relied on for diagnostic purposes.

Findings on CT that are consistent with NSTI include dermal thickening, increased soft-tissue attenuation, fat stranding, and superficial and deep crescentic fluid and air in the subfascial planes. Soft-tissue air with deep fascial fluid collections is highly characteristic of NSTI, but not always seen.[13] As such, the absence of these CT findings should not rule out a diagnosis of NSTI. CT is the most sensitive test for detection of gas and is advantageous over MRI because it is fast and accessible. Therefore, CT has some value in severe NSTI evaluation where cutaneous manifestations lag behind subcutaneous necrosis and can quickly provide an evaluation of extent of disease.

MRI, which is the best available imaging modality for soft-tissue characterization, can help distinguish NSTI from other soft-tissue infections in select cases. Kim et al in 2017 examined contrast-enhanced MRIs of patients with extremity soft-tissue infections and found that patients with NSTI were more likely to have thick (>3 mm) abnormal fascial signal intensity on fat-suppressed T2-weighted images, low-signal intensity in the deep fascia on fat-suppressed T2-weighted images, nonenhancing portions in the areas of abnormal signal intensity in the deep fascia, extensive involvement of the deep fascia, and involvement of three or more

Table 2

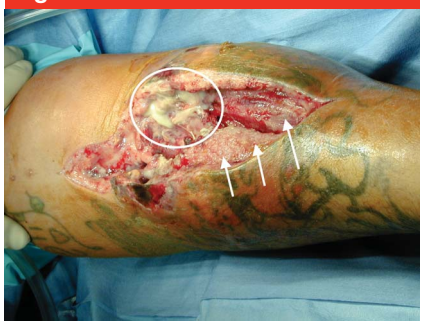
Laboratory Risk Indicator for the Necrotizing Fasciitis Score

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

WBC = white blood cell
A sum > 6 has a high correlation with necrotizing soft-tissue infection.
(Adapted with permission from 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;32:1535-1541. Adaptations are themselves works protected by copyright. So in order to publish this adaptation, authorization must be obtained both from the owner of the copyright in the original work and from the owner of copyright in the translation or adaptation.)

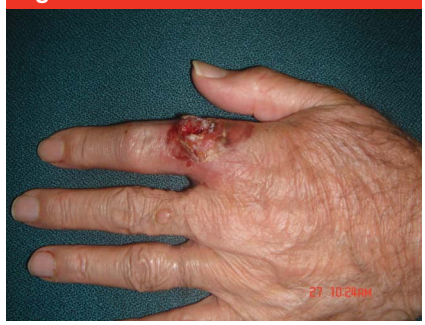
compartments in one extremity when compared with patients who were diagnosed with non-necrotizing infections. Findings such as fascial edema, enhancement of portions of the deep fascia contiguous with the superficial fascia, peripheral muscle enhancement, and subcutaneous abscesses are nonspecific, though can be helpful in altering management and displaying disease extent. In general, MRI can be helpful in select cases with a more subacute presentation, though is often not recommended because MRI can lead to a delay in treatment.[14]

Figure 2



Photograph showing intraoperative findings that demonstrate characteristic gray fascia (arrows) and dishwater fluid (circle).

Figure 3



Photograph showing pyoderma gangrenosum.

Bedside Tests

In addition to imaging, a bedside cut-down or "finger test" may be used in patients with signs and symptoms suspicious for early NSTI before definitive surgery.[15-17] This procedure is performed under local anesthesia. The finger test has historically been described as a 2-cm incision made in the area of interest down to the deep fascia followed by gentle probing with a finger. In the hand and wrist, the authors recommend making a 1-cm incision and passing a blunt hemostat. Findings that correlate with NSTI include the presence of "dishwater" fluid (which is a product of polymorphic cell lysis[16]), lack of resistance to blunt dissection, and absence of bleeding tissue. Rapid frozen-section biopsy following the bedside cut-down test as well as culture and gram stain should be performed.[15]

To be clear, the "finger test" should not be used when a high index of suspicion exists for NSTI because this scenario warrants immediate surgical intervention, which should not be delayed by adjunctive studies or procedures.

Histology and Surgical Findings

Intraoperative examination and histopathology are the benchmark for confirming the diagnosis of NSTI.[4] Histology of the affected tissue reveals infiltration of the fascia and dermis by polymorphic nuclear cells, thrombosis and necrosis of the arteries and veins, and the presence of microorganisms within the necrotic fascia and dermis. These histologic findings are present even in early stages of NSTI.[8]

The histologic characteristics correlate with gross pathologic findings highlighted by liquefied fat and lack of frank suppuration. The fascia has a characteristic gray appearance (Figure 2), which is devoid of bleeding, and a distinct separation of the skin and subcutaneous tissue from the fascia is found.[16,18] Tissue specimens for gram stain and culture should be performed intraoperatively, as the susceptibility of the microorganism will guide antibiotic therapy.

Differential Diagnosis

Early clinical signs and symptoms of NSTI are often confused with cellulitis. Standard laboratory tests, bedside cut-downs, and imaging can assist in the early differential diagnosis of NSTI and cellulitis as

Table 3

Classification of Pathogens According to Type of Infection

Types of NSTI	Etiology	Organism	Clinical Progress	Mortality
Type I (70%-80% cases)	Polymicrobial/ synergistic, often bowel flora derived	Mixed anaerobes and aerobes	More indolent, better prognosis, and easier to recognize clinically	Variable, depends on underlying comorbidities
Type II (20%-30% cases)	Often monomicrobial, skin or throat derived	Usually group A beta-hemolytic *Streptococcus* (GAS), *S aureus*	Aggressive, protean presentations easily missed	>32%, depends if associated with myositis or toxic shock
Type III (commoner in Asia)	Gram-negative, often marine-related organisms	*Vibrio* spp	Seafood ingestion or water contamination wounds	30%-40%
Type IV (fungal)	Usually trauma associated in immunocompetent patients	*Candida* spp immunocompromised patients, Zygomycetes immunocompetent patients	Aggressive with rapid extension, especially if immunocompromised	>47% (high if immunocompromised)

NSTI = necrotizing soft-tissue infection
(Adapted with permission from Morgan MS: Diagnosis and management of necrotising fasciitis: A multiparametric approach. *J Hosp Infect* 2010;75:249-257. Adaptations are themselves works protected by copyright. So in order to publish this adaptation, authorization must be obtained both from the owner of the copyright in the original work and from the owner of copyright in the translation or adaptation.)

discussed. Practitioners should also be aware of other mimickers of NSTIs to provide appropriate treatment and avoid unnecessary and invasive surgery. Pyoderma gangrenosum (Figure 3) is a dermatologic condition that is often associated with ulcerative colitis and can easily be mistaken for infectious conditions such as NSTI. Clinically, this condition has four main variants with the ulcerative form being the most common. This condition begins with the presence of a papule or nodule, which progresses rapidly to painful ulcerated lesions with central necrosis.[19] Treatment includes local wound care, steroids, and immunosuppressant medications. Surgical excision is contraindicated because it may exacerbate the disease and result in extension of necrosis.[20]

Microbiology

NSTIs can be classified based on anatomic location, depth of involvement, or microbial pathogen. These classification systems provide context for typical presentations and affected cohorts, however do little to dictate clinical management or predict morbidity and mortality.[4] Four microbial subtypes of NSTI have been described (Table 3). Type I and II infections were first described by Giuliano et al[21] in 1997 and comprise most cases.

Type I infections, which represent 70% to 80% of cases, are polymicrobial involving both aerobic and anaerobic organisms and are often synergistic. They occur in older patients with underlying medical comorbidities and are more commonly found in the trunk and perineum. These infections tend to be more indolent in nature with the exception of clostridial infections.[1,4,16,22] *Clostridium perfringens* is now a rare cause of NSTI secondary to improvements in sanitation and hygiene.[1,4]

Type II infections are monomicrobial and comprise approximately 20% to 30% of cases. They are caused by group A beta-hemolytic streptococci (GAS) either alone or in association with *Staphylococcus aureus*. Unlike type I infections, type II infections occur in any age group and often occur in persons without medical comorbidities. These infections classically occur in the extremities, and patients often have a history of trauma, surgery, or IV drug abuse.[1,4] Associated group A streptococcal toxic shock syndrome contributes to the aggressive clinical course of these infections.[23]

Type III infections comprised gram-negative marine bacteria, most commonly *V vulnificus*. These infections are associated with marine injury and moderate to severe liver disease.[1,6] They occur more commonly in coastal communities, particularly in Asia.[6] Similar to type II infections with associated toxic shock syndrome, *V vulnificus* NSTI has a fulminant course with early signs of septic shock and a mortality rate of 30% to 40%.[24]

Type IV fungal infections are rare and primarily affect immunocompromised patients. These infections often have a high mortality rate and aggressive clinical course with rapid extension of involved areas.[16]

Pathophysiology

NSTIs are believed to be caused by a breakdown in tissue integrity; however, many infections arise spontaneously in subcutaneous tissues without an apparent wound or lesion. These infections occur through either direct or hematogenous spread. Microbial introduction is followed by subcutaneous tracking of bacteria along fascial planes. Bacteria produce toxins that cause vessel thrombosis, tissue ischemia, and liquefactive necrosis. The thrombosis of perforating vessels to the skin promotes further dissemination of infection and eventually skin necrosis.[1]

Although the underlying pathophysiology is analogous in all types of NSTIs, the rate of progression and presence of systemic toxicity varies depending on the causative organism or organisms. For instance, clostridial and GAS NSTI usually progress within a few hours after initial inoculation.[4,25]

The lethal nature of clostridial infections can be attributed to its alpha and theta exotoxins. The alpha-toxin causes platelet aggregation and thrombus formation, leading to tissue ischemia and necrosis. This condition creates an environment primed for bacterial proliferation.[4] Systemically, the alpha and theta toxins impede phagocyte function, cause intravascular hemolysis, increase endothelial permeability, and decrease vascular tone, leading to cardiovascular collapse. In addition, the alpha-toxin produced by the *Clostridium* species causes extensive muscle necrosis and is responsible for clostridial myonecrosis, which is often referred to as gas gangrene. The distinguishing feature of clostridial myonecrosis is its almost uniform involvement of muscle, which is usually spared in other forms of NSTI.[1]

The virulence of GAS NSTI is enhanced by several microbiologic mechanisms. Streptococci elaborate M proteins that enhance the microbe's ability to adhere to tissue, evade phagocytosis, and induce super antigen activity.[1,4,16] Super antigens cause nonspecific activation of T cells and a massive cytokine cascade.[26] Systemic release of IL-1, IL-6, and tumor necrosis factor-α is also caused by GAS production of exotoxins A, B, C, and streptolysin O, which activate CD4 cells and macrophages. This inflammatory cascade is responsible for the "toxic shock" often seen with GAS NSTI.[4] In addition, exotoxins A and B cause endothelial damage, resulting in tissue edema, diminished blood flow, hypoxemia, and tissue necrosis. Tissue necrosis impairs the ability of neutrophils to fight bacteria through oxidative destruction and also impairs antibiotic delivery.[1]

Treatment

Antibiotic Treatment

When NSTI is suspected, initial treatment should begin with broad-spectrum antibiotics. The Infectious Disease Society of America provides guidelines for the treatment of skin and soft-tissue infections. For broad-spectrum coverage, they currently recommend vancomycin, linezolid, or daptomycin combined with one of the following: piperacillin-tazobactam; carbapenem, ceftriaxone, and metronidazole; or fluoroquinolone and metronidazole.[27] Empiric treatment should be guided by the microbiologic classification of the suspected type of NSTI. Once the microbial pathogen has been speciated, antibiotic treatment should be modified accordingly.[27]

Both GAS NSTI and clostridial NSTI should be treated with clindamycin and penicillin. Clindamycin has notable activity against both species, which decreases alpha-toxin production by clostridial species and reduces M protein production by streptococcal species. Penicillin protects against GAS resistance to clindamycin.[27] *S aureus* should be treated with vancomycin, and *V vulnificus* infections should be treated with a combination of doxycycline plus either ceftriaxone or cefotaxime. Antibiotic therapy should be continued until the surgical control of the infection is achieved, and the patient is hemodynamically stable and afebrile for 48 to 72 hours.[4,27]

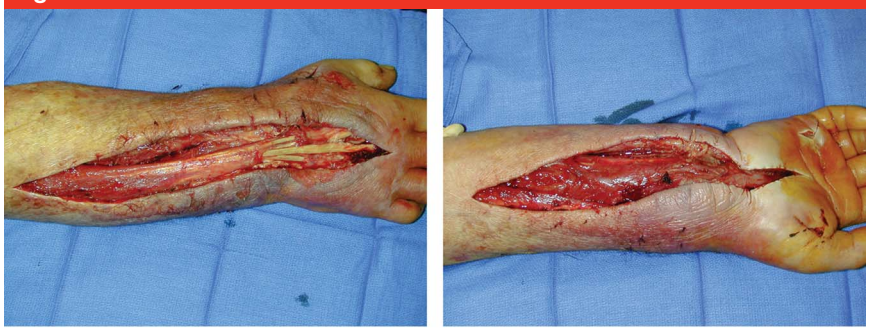
Surgical Management

Emergent, aggressive surgical débridement remains the hallmark of treatment of NSTIs. The goal of débridement is to reduce the bacterial load and arrest fascial necrosis, which is the only intervention in NSTIs that has been shown to reduce mortality.[28] Delayed and inadequate index débridement are the greatest risk factors for increased mortality in NSTIs. Delaying surgery by 24 hours has been shown to quadruple mortality rates.[29]

Débridement should be performed as soon as clinical suspicion of NSTI is found with the goal of removing all devitalized tissues, including a margin of healthy tissue. Any involved skin, fascia, and muscle must be removed (Figure 4, A and B). The recommended incisions for drainage of thenar space infections and deep palmar space infections should be used.[30] All incisions should be designed in an extensile approach using Bruner incisions at joint creases. If débridement extends to the level of the digits, a midaxial incision can be used to provide wide exposure to the volar digital structures. The midaxial incision is extensile and keeps the incision away from the tendon sheath. Wounds should be left open and packed, or drains can be placed.[30]

Surgeons should be prepared to return to the operating room for repeat débridement based on the timing and quality of the initial débridement and the clinical course of the infection. Patients often require revision

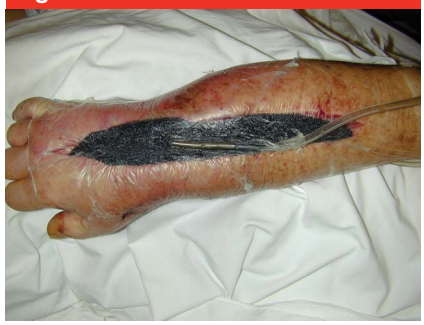
Figure 4



A B

A, Photograph showing postsurgical débridement of the dorsal aspect of the hand and wrist. **B**, Photograph showing postsurgical débridement of the volar aspect of the hand and wrist.

Figure 5



Photograph showing negative-pressure wound therapy placement.

surgery within the first 24 hours if signs of hemodynamic instability and progressive tissue necrosis are present. Microbiologic and pathologic specimens should be taken at each operation to corroborate the diagnosis and tailor antibiotic treatment.

NSTI of the hand and wrist has unique surgical considerations because of the realistic possibility of limb amputation. The extent of initial débridement is controversial because the skin in early NSTI of the extremities often appears normal. As stated earlier, the cutaneous manifestations of NSTI often lag behind the subcutaneous disease process. One should be aware of the extensive vascular microthrombosis and vasculitis that lead to full-thickness necrosis.[31] As such, the treating surgeon should be prepared to take adjacent viable soft tissue and be prepared to amputate if necessary. Tang et al described guidelines for amputation in NSTI of the extremities. The most important criteria for amputation are rapidly progressing infection with a large area of tissue necrosis and extensive necrosis involving the underlying muscle.[32] Amputation should also be considered in patients with concurrent medical disease with high anesthetic risk, shock requiring more than one inotrope, and concurrent vascular insufficiency.[32] Limb amputation is generally a shorter procedure with less blood loss than radical débridement and may be better tolerated by patients who are hemodynamically unstable and will probably not tolerate multiple procedures. It should be noted however that amputation does not decrease mortality rates.[33]

Adjunctive Treatment

Management of extensive fasciotomy wounds after débridement requires special attention. Use of negative-pressure wound therapy (NPWT) is well supported in the orthopaedic literature with several randomized control studies demonstrating improved wound healing compared with standard wound care in cases of open fractures, fasciotomies, and soft-tissue defects.[28] NPWT is used frequently in the treatment of NSTI after repeated débridement, once the infection is controlled[28] (Figure 5). Proposed benefits include promotion of granulation tissue and decreased bacterial load and wound size.[34] Although a lack of evidence reporting the effectiveness of NPWT in NSTI is found, we believe that it is a valuable tool for managing wounds in NSTI. In our experience, NPWT reduces patient discomfort, improves wound care efficiency, and keeps the wounds isolated. Although many surgeons apply NPWT to large wounds at the time of index débridement, some authors advise that NPWT should not be initiated until the offending organism is identified and the wound is devoid of necrotic tissue.[9,35] If anaerobic bacteria are responsible, NPWT may exacerbate the infection.[36] Therefore, we believe NPWT should be initiated only after anaerobic infection has been excluded and the wound is free of necrotic tissue.[9] This usually occurs after initial débridement, when the patient has been stabilized.

Hyperbaric oxygen (HBO) therapy has been suggested as a systemic adjunct in the treatment of NSTI. HBO provides enhanced tissue oxygenation to the penumbra, leading to a number of positive effects. Importantly, elevated leukocyte oxygen levels enhance the killing of pathogenic bacteria, and increased tissue perfusion and oxygen improve antibiotic uptake and effectiveness. HBO therapy also improves lipid peroxidation and free radical scavenging.[35,37] Although NSTI is one of the primary indications for HBO therapy, the proposed benefits must be weighed against the cost and accessibility of treatment because HBO therapy is offered in a limited number of centers.

IV immunoglobulin (IVIg) therapy has been advocated as an adjunctive

treatment in patients with NSTIs because of its ability to neutralize the exotoxins that mediate the cytokine cascade.[16] However, owing to the lack of double-blind controlled studies and limitations of published/available studies suggesting benefits to IVIg therapy, the Infectious Disease Society of America does not recommend IVIg therapy for necrotizing GAS infections.[22]

Successful treatment of NSTI requires input from multiple specialties and support from ancillary services. General surgeons, orthopaedic and plastic surgeons, and intensive care and infectious disease specialists are often involved in the care of these patients. These patients require extensive supportive care including adequate fluid resuscitation, blood pressure support, and close monitoring. Nutritional support is also important because of the loss of fluid, protein, and electrolytes from surgical wounds. Finally, care must also be coordinated between those administering adjuvant therapeutic treatments such as wound care and HBO therapy.

Summary

NSTIs represent a spectrum of clinically challenging disease processes that are uncommon but limb and life threatening. The literature addressing these infections is sparse as they pertain to the hand and wrist. Despite surgical, technological, and pharmacological advances, the morbidity and mortality rates of these infections remain high. Indications for amputation are not well defined, and amputation has not been shown to improve mortality. A high index of suspicion is necessary for early diagnosis, and early and aggressive débridement have been shown to be beneficial.

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