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WILDERNESS MEDICAL SOCIETY CLINICAL PRACTICE GUIDELINES

Prevention and Treatment of Nonfreezing Cold Injuries and Warm Water Immersion Tissue Injuries: Supplement to Wilderness Medical Society Clinical Practice Guidelines for the Prevention and Treatment of Frostbite CME

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We convened an expert panel to develop evidence-based guidelines for the evaluation, treatment, and prevention of nonfreezing cold injuries (NFCIs; trench foot and immersion foot) and warm water immersion injuries (warm water immersion foot and tropical immersion foot) in prehospital and hospital settings. The panel graded the recommendations based on the quality of supporting evidence and the balance between benefits and risks/burdens according to the criteria published by the American College of Chest Physicians. Treatment is more difficult with NFCIs than with warm water immersion injuries. In contrast to warm water immersion injuries that usually resolve without sequelae, NFCIs may cause prolonged debilitating symptoms, including neuropathic pain and cold sensitivity.

Keywords: trench foot, immersion foot, warm water immersion foot, tropical immersion foot, paddy foot

Introduction

Cold and warm water immersion injuries have afflicted military personnel and civilians for centuries. Nonfreezing cold injury (NFCI) can result from prolonged exposure to cold, often wet, conditions that do not cause freezing of tissue. It is likely that trench foot, immersion foot, and cold immersion injury are types of NFCI. Warm water immersion injuries, including warm water immersion foot (WWIF) and the more severe tropical immersion foot (TIF), differ from NFCIs. Both NFCIs and warm water immersion injuries can be painful, debilitating conditions. Nonfreezing cold injuries can be difficult to manage and may cause prolonged debilitating symptoms, including neuropathic pain and cold sensitivity that may be refractory to treatment. Warm water immersion injuries, once recognized, can be easily treated, resolve completely, and seldom have chronic sequelae.

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Methods

We convened a multidisciplinary expert panel to develop evidence-based clinical guidelines for evaluation,

treatment, and prevention of NFCIs and warm water immersion injuries. We selected panelists based on clinical and research experience. The panelists generated a list of questions to define the most significant areas of interest (see online Supplemental Table 1). We performed a literature search to identify relevant articles using a keyword search of the MEDLINE database. Keywords were nonfreezing cold injury, trench foot, immersion foot, immersion injury, warm water immersion foot, tropical immersion foot, jungle foot, and paddy foot. We also searched manually for additional articles using the reference lists of the articles we identified in the original search. We relied on peer-reviewed randomized controlled trials, observational studies, case series, and case reports to formulate recommendations.

We assessed the level of evidence supporting each diagnostic, therapeutic, and preventive modality. We cited review articles only to provide background information. We did not use conclusions from review articles as bases for recommendations.

We used a consensus approach to develop recommendations. The panel graded each recommendation based on the quality of supporting evidence and the balance between the benefits and risks/burdens according to the criteria of the American College of Chest Physicians (see online Supplemental Table 2).[1]

Definitions

NFCI (TRENCH FOOT OR IMMERSION FOOT)

Nonfreezing cold injuries occur after prolonged exposure of extremities to cold, often wet, conditions, with water temperatures <15°C (59°F). Nonfreezing cold injuries without exposure to wet conditions may be a different condition and likely require exposure to air temperatures significantly <15°C (59°F).[2,3] Feet are the most commonly affected body parts, but any peripheral body parts, especially legs and hands, can also be affected. Nonfreezing cold injury affecting the hand of a diver exposed to water at 6°C has been reported.[4] Dependent body parts, such as the buttocks when sitting and knees when kneeling, may also be injured, most likely from pressure in combination with cold and often wet conditions. Other terms for NFCI, related to the mechanism of exposure, include "sea boot foot" and "bridge foot."[3]

Conditions such as "shelter foot" or "shelter limb"[3] affected people who sat for long periods in cold air-raid shelters in World War II.[5] These conditions were the result of nerve compression and dependent limbs and did not involve moisture. They are different than the injuries we refer to as NFCIs and are beyond the scope of the current guidelines. Also beyond the scope of the current guidelines are other conditions that have sometimes been called NFCIs, including chilblains (pernio) and cold-induced peripheral neuropathies that do not involve exposure to wet conditions.

We consider trench foot and immersion foot to be similar or identical injuries. Immersion foot in sailors is equivalent to trench foot in soldiers. Both injuries exhibit the same progression of stages. In the absence of standard diagnostic criteria, some clinicians believe that NFCIs, with mild or no sequelae, can occur after a few hours of exposure without moisture. There is a paucity of data on these possible injuries. We do not discuss them in the guidelines.

WARM WATER IMMERSION INJURIES

Warm water immersion foot and TIF are transient syndromes caused by prolonged exposure to warm water. Warm water immersion foot presents with white, wrinkled soles of the feet. Tropical immersion foot is more severe than WWIF. Tropical immersion foot presents with symmetrical erythema, swelling, and tenderness of the skin of the ankles and dorsa of the feet. Warm water immersion foot is caused by exposure to water at temperatures of 15 to 32°C (59–90°F) for <3 d.[6] Another term for WWIF is "paddy-field foot."[7] Warm water immersion foot has also been incorrectly called "tropical immersion foot."[8] It resolves in 1 to 3 d if the feet are dried and elevated.[9] Warm water immersion foot can also affect the hands.[10] Tropical immersion foot is caused by exposure to warm water at temperatures of 22 to 32°C (72–90°F) for >3 d.[8] It is also treated by drying and elevation of the feet. Recovery takes from 4 to 12 d. Neither WWIF nor TIF causes sequelae. The term "jungle foot" and the related terms "tropical jungle foot," "jungle rot," and "paddy foot" are not well defined. Jungle foot usually refers to TIF.[8]

NFCIs

EPIDEMIOLOGY OF NFCIs

Nonfreezing cold injuries can occur when extremities are subjected to cold, often wet, conditions that overwhelm their capacity to maintain warmth, leading to peripheral cooling, with or without central cooling. Nonfreezing cold injuries usually occur only after continuous exposure to cold for at least 1 to 3 d,[11,12] although they have been reported after immersion in cold sea water at 0 to 8°C (32–46°F) for 14 to 22 h.[2] Some authors have suggested, based on anecdotes, that an NFCI can occur in as short a time as 1 h.[13] There is 1 report of shipwrecked sailors who sustained injuries that might have been NFCIs at

water temperatures about 16 to 21°C (61–70°F) after 8 d with continuously wet feet.[14]

Environmental factors that increase the rate of cooling, such as wind or moisture, likely increase the risk of NFCI. Nonfreezing cold injuries are especially common with wet footwear in cold conditions and are often associated with fatigue, malnutrition, dehydration, immobility, and dependent limbs. Military personnel are believed to have the greatest risk during combat and training. Other occupational groups, such as harbor and cannery workers, may also be affected. Wilderness travelers, mountaineers, homeless people, alcoholics, and the elderly are at risk when they are unable to remove wet footwear during prolonged exposure to cold, wet conditions.

INDIVIDUAL RISK FACTORS FOR NFCI

Individuals vary widely in susceptibility to cold injury. Associated injuries, such as fractures of the extremities or multiple trauma, increase the risk of NFCI. Conditions that affect the circulation, such as peripheral vascular disease and Raynaud's phenomenon, may increase the risk and severity of NFCI. Mental illness and alcohol use may impair behavioral responses, increasing the risk of NFCI. Smoking, older age, and African ethnicity have also been suggested as possible risk factors.[15] A retrospective study of UK soldiers suggested that NFCI had the highest incidence during winter training in younger, less-experienced soldiers. This study did not find that smoking was a risk factor. Personnel with Afro-Caribbean ethnicity seemed to have greater risk than Caucasians.[16]

PHYSIOLOGY OF SKIN COLD EXPOSURE

The skin contributes to thermoregulation by acting as a surface for heat exchange. Vasodilation leads to increased skin blood flow, facilitating heat loss, while vasoconstriction decreases skin blood flow, limiting heat loss. Skin blood flow is controlled by sympathetic innervation and local vascular mechanisms. Central hypothalamic control mechanisms respond to central and peripheral temperature afferent information to cause reflex neurogenic vasoconstriction or vasodilation. If core and average skin temperatures allow, local temperature changes in the skin can also cause locally mediated vasodilation or vasoconstriction independent of the central nervous system or reflex neurogenic mechanisms. Skin blood flow can decrease by approximately 90% in a cold environment compared with resting blood flow in a thermoneutral environment while still meeting the lower metabolic needs of cold skin. Skin blood flow can also decrease in dependent extremities during immobility and from pressure produced by constrictive clothing or footwear in combination with edema.

Cooling of the skin to about 15°C (59°F) increases vasoconstriction of hands or feet, making them vulnerable to cold injury. Further local cooling causes cold-induced vasodilation (CIVD), with cyclic increases in blood flow that likely protect against cold injury. Cold-induced vasodilation, also known as the "Lewis hunting response," occurs in cycles as short as 5 to 10 min that lengthen with duration and increased cooling.[17] Decreasing the core temperature decreases the magnitude and frequency of the cycles.[18] Cold-induced vasodilation can be abolished in hypothermia. People who have repeated, long-duration exposures to cold have more rapid cycles, with warmer peak temperatures than those without such exposures.

PATHOPHYSIOLOGY OF NFCI

Nonfreezing cold injury is a neurovascular injury, with impaired circulatory control and damage to the microcirculation.[13] Animal studies have demonstrated thrombosis and endothelial injury in the microcirculation. Nerves can be injured directly by cold or can be affected by injury to their microvascular circulation.[19,20] Direct neural damage from cold has been reported in victims with severe hypothermia and may also occur in victims with isolated NFCI.[21,22]

Prolonged, profound vasoconstriction plays a role in the pathogenesis of NFCI[13,23] but is likely not the only cause.[24] Nonfreezing cold injuries occur when an extremity is subjected to cold, usually with external moisture, for prolonged periods of time.[3] The extent of tissue injury seems to be greater with lower temperatures and longer exposures. Repeated cold exposures with incomplete recovery between exposures cause greater injury than a single, long exposure, possibly because of reperfusion injury.[20] A rat study showed that nerve injury and production of reactive oxygen species were greater when nerves were subject to intermittent cooling with periods of warming compared to continuous cooling conditions.[25] This suggests an important role of free radicals. The exact parameters of temperature and duration that can cause NFCI are unknown.

We believe that NFCI alone is unlikely to cause loss of tissue. Tissue loss is most likely caused by pressure necrosis from edema caused by NFCI, associated with constricting footwear or clothing. Extreme cases of pressure can potentially lead to a compartment syndrome. Victims who must walk with swollen feet may also sustain tissue loss caused by mechanical factors.

DIAGNOSIS AND CLINICAL COURSE OF NFCI

Nonfreezing cold injury is a clinical diagnosis. In severe NFCI, affected extremities pass through 4 stages.[2,13] There can be great variability in the lengths of the stages.

First stage: during cold exposure

The first stage, during cold exposure, is characterized by a loss of sensation. Victims often complain that the affected areas feel numb and like a block of wood. A retrospective study found that the most common initial symptom of NFCI was loss of sensation lasting >30 min.[16] Loss of proprioception can cause clumsiness and gait disturbances. Affected extremities are usually painless. They can be bright red initially, later becoming pale or white because of severe vasoconstriction. In the later part of the first stage, peripheral pulses are diminished.

Second stage: following cold exposure

The second stage occurs when the victim is moved to a warm environment and continues during and after rewarming. Usually, this stage lasts for a few hours but may continue for several days. Hands and feet become a mottled pale blue because of slightly increased blood flow. The color change may not be readily visible in victims with highly pigmented skin. Peripheral pulses are diminished initially but later become bounding, although capillary refill is still delayed. Affected extremities may swell. They remain cold and insensate during the second stage.

Third stage: hyperemia

The third stage usually begins abruptly and lasts for days or weeks, as long as 6 to 10 wk in severe cases. This stage is characterized by bright red, edematous extremities with bounding pulses and delayed capillary refill, likely caused by microvascular injury. Affected extremities are extremely painful and hyperalgesic, although some distal areas may remain insensate. There is usually no tissue damage, but areas that were affected by pressure and are nonviable may develop blisters or discoloration.

Fourth stage: following hyperemia

The fourth stage may last from a few weeks to years and may be permanent in severe cases. Unless there is tissue loss, affected extremities appear normal. One of the most common persisting manifestations of NFCI is increased sensitivity to subsequent cold exposure. Cold sensitivity may begin at any time, as long as 6 wk after the injury, even in mild cases when neuropathic symptoms resolved in the first week. Injured extremities cool more easily when exposed to cold, are more uncomfortable, and are slow to rewarm. Subsequent cold exposure may cause intense vasoconstriction, often persisting for hours, even after a short cold exposure. In a retrospective case series, victims had subjectively colder extremities, abnormal pinprick sensation, and decreased sensation to light touch but no proprioceptive loss or gait disturbance.[16] Many victims with severe injuries have chronic pain, often exacerbated by cold exposure. There may still be small insensate areas. Hyperhidrosis is a common sequela in severe cases. Victims may also develop chronic neuropathic conditions such as complex regional pain syndrome (CRPS) or similar syndromes. Rarely, tissue affected by pressure necrosis at the time of injury may become frankly necrotic in the fourth stage, requiring amputation. Sensory neuropathy can also lead to complications such as trauma and infections, including osteomyelitis or sepsis.

Nonfreezing cold injury is not a progressive condition. The worst symptoms occur in the first few days. Afterward, symptoms caused by NFCI usually improve or remain stable. Worsening symptoms should not be attributed to NFCI.

Recommendations. In conditions sufficient to cause significant peripheral cooling, especially in a wet, cold environment with a water temperature <15°C (59°F), a victim with an extremity that has been cold and numb for hours to days may have NFCI (1C).

A victim who is asymptomatic, with a normal physical examination and no persistent neurologic symptoms after rewarming, does not have NFCI (1C).

When neurologic symptoms or signs persist beyond 1 wk after cold exposure affecting 1 or more extremities and other causes of peripheral neuropathy have been excluded, the diagnosis is likely NFCI (1C).

Worsening of symptoms attributed to NFCI after the first 2 to 3 d following the injury should prompt a search for other causes (1C).

DIFFERENTIAL DIAGNOSIS OF NFCI

Frostbite, unlike NFCI, occurs only at air temperatures well below freezing and cannot occur in cold water, even sea water. Frostbite and NFCI can theoretically occur together if tissue with NFCI subsequently freezes. Frostbite that is still frozen appears pale blue, yellow, or white. Unlike tissue with NFCI, frostbitten tissue that has not been thawed is firm or hard and looks waxy.

Pressure injury can occur in association with NFCI because of swelling inside constrictive clothing and footwear. Pressure injury is painful unless the tissue is

insensate, as in cold injury. Pressure injury can cause ischemia, with local tissue loss (gangrene). In severe cases, compartment syndrome, with distal loss of tissue, may also occur. Tissue necrosis after exposure to temperatures well below freezing is most likely to be caused by frostbite.

Infections can cause redness and swelling of the skin, with subsequent necrosis, usually unilaterally. Infections seldom involve only the distal portion of an extremity. Systemic symptoms and signs are often present with infection and are rare with uncomplicated NFCI.

Raynaud's phenomenon, severe vasoconstriction in response to cold, usually affects hands or feet bilaterally. Blanching is rapid and well demarcated with adjacent areas of unaffected skin. There is full recovery, without sequelae, after rewarming.

Frostbite, pressure necrosis, infection, and Raynaud's phenomenon can all occur in association with NFCI.

Recommendations. Use the history of environmental exposure, clinical manifestations, and physical examination to distinguish NFCI from frostbite, pressure necrosis, infection, and Raynaud phenomenon (1C).

TREATMENT OF NFCI

There are no well-conducted case-control or cohort studies to support recommendations for treatment of NFCI. Our recommendations are based primarily on research, experience, and retrospective studies.

Prehospital care

Further cooling should be prevented.[26] Hypothermia should be treated before treating other injuries, including frostbite and NFCI.[27] Rapid rewarming of NFCI has not been shown to improve outcomes. Immersion in warm water increases pain and edema[28] and also increases the metabolic demands of injured skin.[2] Extremities with NFCI are damaged and susceptible to further trauma. Nonfreezing cold injuries are likely to be insensate until they are at least partially rewarmed.

Recommendations. Victims with swollen feet should not walk unless walking is necessary for evacuation. Victims who must walk should wear thick socks for padding in nonconstricting footwear, if possible (1C). Treat associated conditions, including hypothermia and frostbite, before treating NFCI (1B). Extremities with NFCI should be rewarmed passively at room temperature (1A) and should be elevated (1B). Rewarming with warm water, forced air, or a heating pad should not be attempted (1A). Administer analgesia as needed for pain (1A).

Emergency department and in-hospital care

Tetanus prophylaxis, according to standard guidelines, is indicated for associated injuries such as frostbite and other wounds.[27] A case report described the use of iloprost within 24 h to limit tissue loss in severe NFCI.[29] The authors of the report suggested that the use of iloprost was safe and allowed delayed amputation, with less tissue loss than expected based on apparent tissue damage on initial assessment. Antibiotics are not indicated in NFCI unless there is evidence of infection. Although there are no specific data, prophylaxis of venous thromboembolism may be indicated in immobile victims.

Recommendations. Treat hypothermia before treating NFCI (1A).

Allow extremities with NFCI to rewarm passively at room temperature (1A), with bed rest, elevation, and air drying at room temperature (1B).

Administer analgesia as needed for pain (1A). Give tetanus prophylaxis if indicated (1C).

No recommendation can be made concerning the early use of iloprost because there are insufficient data.

Administer antibiotics only if there are signs of infection (1A).

Pain relief

Elevation of hands and feet can decrease pain during the hyperemic stage.[13] Hands and feet can be open to air or covered with light, loose dressings. Cooling the feet with ice bags wrapped in towels has been studied but is contraindicated because it may cause frostbite.[30]

Nonsteroidal anti-inflammatory drugs and opioids are usually ineffective as the sole treatment for relief of pain.[13] Nifedipine, a vasodilator, has not been shown to be effective for pain relief.[13] Amitriptyline (50–100 mg daily at bedtime) may be effective, especially if started as soon as pain develops.[13] If amitriptyline is not adequate, medications that treat neuropathic pain, such as gabapentin, can be administered. Lumbar sympathectomy is an obsolete procedure that was reported to relieve cold sensitivity, in some cases, for as long as a few months, after which symptoms returned, as severe as, or worse than, those before the procedure.[13]

Recommendations. Elevate affected extremities. Protect extremities from constriction. Keep affected extremities open to air or lightly dressed using loose dressings (1A). If nonsteroidal anti-inflammatory drugs and opioids are not effective, administer amitriptyline for pain (1C). If

pain is not controlled adequately by amitriptyline, consider a trial of gabapentin (1C).

Infection

A low-grade fever (38–38.5°C [100.4–101.3°F]) often develops in the first 12 to 36 h.[31] Infection is rare. Infection does not usually develop after NFCI unless traumatic blisters become secondarily infected.

Recommendations. Do not treat a victim with an isolated low-grade fever with antibiotics unless there are other clinical manifestations of infection (1C). Prophylactic antibiotics are not indicated (1C). Obtain emergent surgical consultation for suspected tissue necrosis (1C). Antibiotic treatment for cellulitis should cover staphylococci, streptococci, and pseudomonas species (1C).

Laboratory and imaging studies

There are no specific laboratory or imaging studies that can help establish the diagnosis of NFCI. Diagnostic studies may be necessary for coexisting conditions. There is no known role for computed tomography angiography, magnetic resonance angiography, or radioisotope scanning to assess circulation in an extremity with NFCI. Infrared thermography is no longer used in the UK to guide postdischarge management. Use of infrared thermography alone was not shown to be effective in controlled trials.

Recommendations. Obtain x-rays of affected extremities if trauma is known or suspected (1A). Obtain computed tomography or magnetic resonance imaging to assess coexisting conditions, when indicated (1A).

LONG-TERM MANAGEMENT, PROGNOSIS, AND SEQUELAE

Sequelae can include chronic neuropathic pain and CRPS. In an open-label study, nicotinyl tartrate (oral nicotine) alleviated symptoms in 16 of 36 victims, with improvement in pain, paresthesias, and exercise tolerance.[32] Other vasodilators, including theophylline and papaverine, were not effective.[32]

In a case report, iloprost, a prostaglandin analog with anti-inflammatory and vasodilator properties, was used to treat a 41-y-old military veteran with foot pain and limited mobility 20 y after an NFCI.[33] The first infusion of iloprost was followed by 4 wk of pain relief and increased mobility, with symptoms returning to the preiloprost baseline over the next several weeks. The second infusion, given 3 mo later, was followed by increased pain, again returning to preiloprost baseline a few months later.

An open-label study of capsaicin patch treatment of 16 military participants with long-term sequelae of NFCI showed a reduction in neuropathic pain that was statistically significant but not clinically significant.[34] The study also found a statistically significant increase of intraepidermal nerve fibers. The study was limited by the lack of a control group.

Recommendations. No recommendations can be made for the use of iloprost or capsaicin patches for chronic neuropathic pain associated with NFCI because of insufficient data.

The UK military has a standard regimen for management of soldiers with NFCI after hospital discharge. Some soldiers recover with time. Individuals who are asymptomatic, with a normal examination, are gradually re-exposed to increasingly cold environments. If they have normal responses to cold, they are returned to full duty. Outdoor work is allowed if symptoms are minimal and if the individual can stay warm enough to prevent numbness. Individuals meeting these criteria have no increased risk of further episodes of NFCI, although they may experience cold sensitivity symptoms. Individuals with peripheral neuropathy are referred to a clinic for further assessment, including intraepidermal nerve fiber density. A low density in an individual with a clinical history and physical examination suggesting NFCI increases the likelihood that the individual has small-fiber neuropathy secondary to NFCI. Measurement of intraepidermal fiber density can also help predict occupational recovery time. There is still no definitive test for NFCI. The use of diagnostic testing will likely continue to evolve. Individuals with persistent pain are referred to a specialist pain clinic.

Recommendations. Follow a standard regimen for postdischarge management (1B). Asymptomatic individuals with a normal examination and normal responses to cold can resume normal activities (1B). Individuals with neuropathy should undergo assessment, including intraepidermal nerve fiber density, to aid in diagnosis and prediction of recovery time (1C). Victims with chronic neuropathic pain and CRPS should be referred to a pain specialist (1B).

PREVENTION OF NFCI

Regular rotation of personnel out of cold, wet environments is the mainstay of prevention of NFCI.[35] There is little evidence about specific regimens to prevent NFCI.

A prospective study suggested that prevention of NFCI could be facilitated by pairing soldiers with instructions to regularly inspect the feet and footwear of their paired companions. A soldier was more likely to remove boots and dry socks and feet if reminded by their companion soldier.[36]

Other helpful measures include adequate nutrition and insulating, nonconstricting clothing that keeps personnel, especially their hands and feet, warm and dry. Specific recommendations for foot care are based on prevention of warm water immersion injuries. The recommendation to air dry feet for at least 8 h daily is based on prevention of WWIF.[6,28] Recommendations to keep feet dry for 24 h after 48 h of immersion apply to TIF.[9]

Stress can precipitate vasoconstriction. Preparation and training for operating in cold, wet conditions may decrease stress and mitigate vasoconstriction, reducing the likelihood of NFCI.[37]

In World War I, trench foot was almost completely eliminated in allied troops when waterproof bags with clean, dry socks were sent to the trenches every night with increased rations.[12] Other measures that were effective were the prohibition against using puttees (constricting wraps around the lower leg above the boots), encouraging soldiers to move around as much as possible, and the use of gum boots and foot powder (asbestos-free talcum powder) instead of oils.[12]

Vapor barrier boots provide insulation but can become wet on the inside from insensible loss of water vapor or from sweating. Neoprene socks are warm but constricting. They may pose a risk for WWIF.

Recommendations. Limit exposure to cold, wet conditions (1A). Ensure adequate nutrition and insulating, nonconstricting clothing that keeps personnel, especially their hands and feet, warm and dry (1B). Do not re-expose victims with previous NFCI to cold environments (1A). Rotate personnel regularly out of cold, wet environments (1A). Encourage personnel to move around frequently and to avoid having the legs and feet dependent (1C). Train for operations in cold, wet conditions (1C).

Keep feet as dry as possible (1A). In wet conditions, change into dry socks 2 or 3 times daily (1C). If using vapor barrier boots, use asbestos-free talcum powder in addition to regular changes of socks (1C). Do not use neoprene socks (1C). Keep hands dry and warm (1C). Do not use grease or oils on feet or hands (1A).

Warm Water Immersion Injuries

EPIDEMIOLOGY OF WARM WATER IMMERSION INJURIES

Warm water immersion injuries primarily occur in military settings in warm climates, such as the Vietnam War in the mid-1960s, when soldiers with wet feet are unable to remove their footwear and dry their feet for periods longer than a day.

INDIVIDUAL RISK FACTORS FOR WARM WATER IMMERSION INJURIES

The primary risk factor for warm water immersion injuries is continuous immersion of the feet in water >15°C (59°F) for longer than a day with no opportunity to dry the feet or to replace wet socks and boots with dry ones.[8] Heavy callouses are a risk factor for WWIF.[8] Victims with prior episodes of TIF are more likely to have subsequent episodes.[8]

PATHOPHYSIOLOGY OF WARM WATER IMMERSION INJURIES

There has been little research regarding the pathogenesis of warm water immersion injuries. Warm water immersion foot is likely due to hyperhydration (waterlogging) of the plantar stratum corneum in warm water exposures of <72 h.[8] In TIF, water diffuses through the waterlogged epidermis into the dermis with warm water exposures of >72 h.[8]

DIAGNOSIS AND CLINICAL COURSE OF WARM WATER IMMERSION INJURIES

Warm water immersion injuries can usually be diagnosed clinically by history and physical examination.

Warm water immersion foot results from immersion in warm water at 15 to 32°C (59–90°F) for as long as 3 d.[8] The feet are painful, with white, wrinkled plantar surfaces and sometimes with mild swelling. Weight bearing causes pain and paresthesias of the feet, with tingling or a feeling of "walking on rope."[8]

Tropical immersion foot results from immersion in warm water at 22 to 32°C (72–90°F) for >3 d.[8] Initially, there is burning pain, greater on the dorsal aspects of the feet than on the soles, that increases with walking. The feet are usually too swollen to replace the boots after the victim removes them.[8] By the time the victim is admitted to the hospital, the feet are usually bright red, with demarcation above the level of the top of the boots and pitting edema of the dorsa and the ankles. The skin is initially cool but,

within 12 h, becomes warm, with full pulses and brisk capillary refill. In severe cases, fever and inguinal lymphadenopathy can occur, without lymphangitis.[8]

Tropical immersion foot is often associated with abrasions from wet socks and ulcers in areas of the feet or ankles where footwear causes pressure on swollen tissue. Ulcers are uncommon over bony prominences, where there is little tissue to become swollen. Plantar surfaces are hyperhydrated and wrinkled. During the Vietnam War, victims with severe TIF developed fever and inguinal lymphadenopathy, in contrast to victims described in the South Pacific during World War II, who did not develop these signs.[8]

In victims recovering from TIF, fever, adenopathy, pain, and tenderness resolve and plantar surfaces return to normal within 2 to 3 d following removal from the wet environment. Red dorsal surfaces and ankles transition to diffuse blotchy ecchymoses, with tiny vesicles and a fine maculopapular rash. Edema resolves in 4 to 7 d. The skin desquamates and subsequently returns to normal. There are no sequelae.[8]

Recommendations. A victim whose feet were immersed in warm water for no longer than 3 d and who has painful, white, wrinkled soles and paresthesias most likely has WWIF (1C). A victim whose feet were immersed in warm water for >3 d and who has redness of the dorsal surfaces and ankles, with a burning sensation more severe on the dorsal surfaces than on the plantar surfaces, most likely has TIF (1C).

DIFFERENTIAL DIAGNOSIS OF WARM WATER IMMERSION INJURIES

Differential diagnosis of warm water immersion injuries includes NFCI and frostbite. These can be distinguished from WWIF or TIF by a history of exposure to cold temperatures or cold water rather than warm water. Pressure necrosis can mimic or coexist with warm water injury. Tropical immersion foot can cause ulcerations in areas of swollen tissue.[8] Like TIF, soft-tissue infections can cause erythema and edema, sometimes with fever and lymphadenopathy. Bacterial infections, including streptococcal cellulitis, and fungal infections can occur as sequelae and should also be considered in the differential diagnosis. Infections are usually unilateral and well demarcated, unlike TIF, which is usually symmetrical and diffuse. Wet-sock erosions are superficial skin erosions that occur only in areas that experience friction or pressure, especially areas under bootlaces, unlike the lesions in TIF that cover the dorsum and ankle.[8]

Recommendations. Use clinical characteristics to distinguish WWIF and TIF from each other and from NFCI or frostbite, pressure-induced injury, soft-tissue infection, and wet-sock erosions (1C).

TREATMENT OF WARM WATER IMMERSION INJURIES

If the feet are kept dry, WWIF resolves in 2 to 3 d, without sequelae.[8] To treat TIF, the feet should be dried. The victim should be placed at bed rest with the feet elevated. Recovery, with resolution of edema, takes 4 to 5 d, except in severe cases, which may require 10 to 12 d of treatment. If skin integrity is compromised, there may be a risk of tetanus. As with NFCI, amitryptiline[13] or medications with neuropathic pain, such as gabapentin,[38,39] may be effective for pain during the recovery period.

Recommendations. Treat WWIF by drying the feet and keeping them dry for 2 to 3 d (1B). Treat TIF by drying the feet, keeping them dry, and placing the victim at bed rest with the feet elevated for 4 to 5 d (1B). Give tetanus prophylaxis according to standard guidelines if skin integrity is compromised (1C). Administer medications, including amitryptiline (2B) or gabapentin (2B), for pain relief. Once symptoms resolve, the victim can return to previous activities (1A).

COMPLICATIONS OF WARM WATER IMMERSION INJURIES

Fungal infections are the most common complication of warm water immersion injuries.[6] Bacterial infections can also occur. Prophylactic antibiotics are not indicated. Fungal infections should be treated with topical antifungal agents. Cellulitis or other bacterial soft-tissue infections should be treated with systemic antibiotics. There are no known long-term sequelae of WWIF or TIF.

Recommendations. Do not give prophylactic antibiotics (1B). Treat infections that occur as complications of warm water immersion injuries with topical antifungals or antibiotics, as appropriate (1A).

PREVENTION OF WARM WATER IMMERSION INJURIES

Warm water immersion foot can be prevented by allowing the feet to dry every night after prolonged exposure to warm water.[6] If drying the feet every night is not possible, prevention of WWIF can be achieved by twice-daily application of silicone grease to the soles, between the toes, and to the dorsa and ankles up to the malleoli.[9]

Tropical immersion foot can be prevented by keeping the feet dry for 1 d after each 2 to 3 d period of continuous exposure to water.[8]

Recommendations. When feet are continuously wet, prevent warm water immersion injuries by drying them every night or by applying silicone grease (1C). If feet must be continuously wet for 2 to 3 d, keep them dry for 1 d between exposures (1C).

Conclusions

To improve care for victims, we have developed evidence-based guidelines for diagnosis, treatment, and prevention of NFCI and warm water immersion injuries. Prevention is key. Treatment strategies are limited and not well studied. Many of our recommendations are based on weak evidence. There is ample scope for further research.

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Supplemental Material(s)

Supplementary material associated with this article can be found in the online version at https://doi.org/10.1016/j.wem.2023.02.006.

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