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Therapeutic recommendations for frostbite: A narrative overview

Jurij Gorjanc[1] A,B,D-G, Rosmarie Oberhammer[2] A,B,D-G

[1]Klagenfurt am Wörthersee Hospital, Surgery, Klagenfurt, Austria
[2]Bruneck Hospital, Department of Anesthesia and Intensive Care, Bruneck, Italy

Review article

Abstract

Background: Frostbite is a localized cold injury resulting from tissue freezing, primarily affecting the extremities. Although rare in the general population, it is more frequent among individuals exposed to extreme cold, such as soldiers and mountaineers. Severe cases can result in permanent disability and tissue loss.

Objective: To provide a narrative overview of current evidence-based recommendations for the prevention and treatment of frostbite, based primarily on recent guideline updates from the Wilderness Medical Society (2024) and the American Burn Association (2024).

Methods: This narrative review summarizes and compares recommendations from two major clinical guideline publications, supplemented by supporting literature. No systematic literature search or meta-analysis was conducted.

Results: Immediate management focuses on protecting the patient from further cold exposure, treating hypothermia, and rapidly rewarming affected areas in a warm water bath (37–39°C), followed by sterile wound care, possible blister management, splinting (immobilisation) and rapid hospital transport. Ibuprofen is recommended early for its anti-inflammatory and antiplatelet effects. In hospital settings, treatment includes rapid rewarming (if not yet applied), sterile wound care, analgesia, hydration, and tetanus prophylaxis. Thrombolytic therapy with rt-PA within 24 hours of rewarming may reduce amputation rates in deep frostbite. Iloprost is recommended as first-line therapy for grade 3–4 injuries, particularly when thrombolysis is contraindicated. Antibiotics are reserved for confirmed infection.

Conclusions: Frostbite results from prolonged cold exposure, causing ice crystal formation, microvascular injury, inflammation, and ischemia. Early recognition and rapid, controlled rewarming, pain management, and immobilization are essential. Hospital care includes severity assessment, pharmacologic therapy (ibuprofen, thrombolytics, iloprost, anticoagulants), selective blister management, and supportive measures. Timely intervention reduces tissue loss, prevents progression to necrosis, and improves functional outcomes.

Keywords

- frostbite
- cold injury
- guideline review

Contribution

A – Preparation of the research project
B – Assembly of data
C – Conducting of statistical analysis
D – Interpretation of results
E – Manuscript preparation
F – Literature review
G – Revising the manuscript

Corresponding author

Jurij Gorjanc
e-mail: jurij.gorjanc@mf.uni-lj.si
Klinikum Klagenfurt am Wörthersee
Chirurgie
Feschnigstrasse 11
9020 Klagenfurt am Wörthersee
Austria

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Introduction

Frostbite occurs when tissue freezes.[1] The incidence of frostbite in the general population is low.[2,3] In colder regions, however, it is significantly higher among certain occupational groups (e.g., soldiers) and individuals engaged in particular outdoor activities (e.g., ice climbing, high-altitude mountaineering).[4,5] The most commonly affected areas are the extremities: toes (60%) and fingers (30%), while the ears and nose are less frequently involved.[6] Severe frostbite of the hands can lead to significant disability.[5-7]

Etiology – risk factors for frostbite: The risk of frostbite increases due to environmental and individual factors.

Environmental factors: Low temperature, wind chill, long cold exposure, high humidity, immersion in cold water, hypoxia at high altitude, radiant heat loss, and direct contact with cold objects.[8]

Individual factors: Short-term: fatigue, stress, immobility, wet or tight clothing, dehydration, hunger. Long-term: previous cold injury, cold sensitivity, Raynaud's phenomenon, smoking, poor fitness, chronic heart disease, certain medications, extreme age (elderly, newborns), gender (females), low body fat, malnutrition, alcohol or drug abuse.[9]

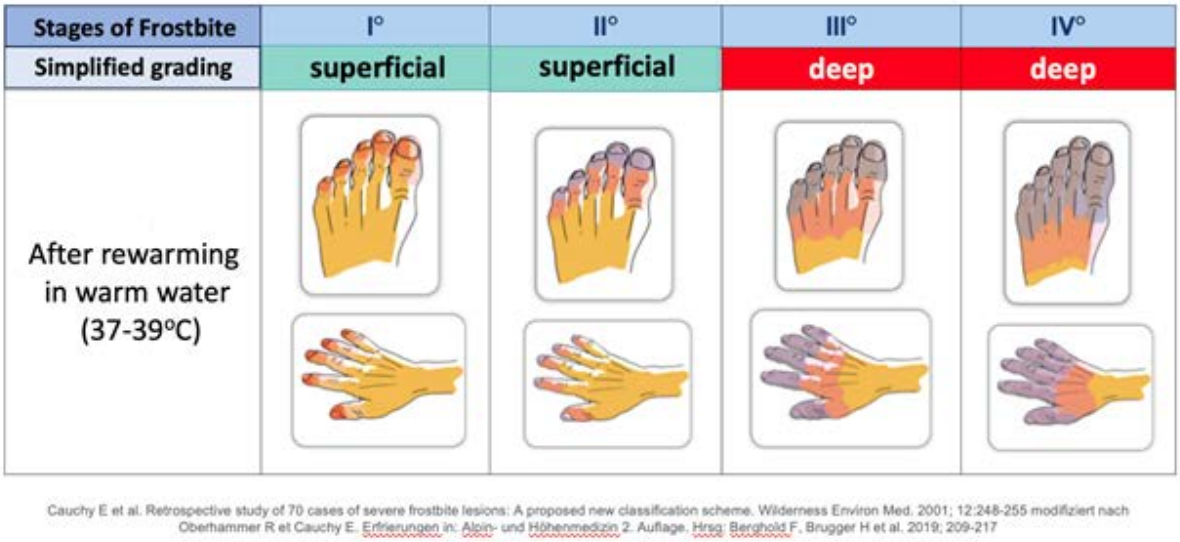
Classification of frostbite

Frostbite is classified into four degrees of severity (Figure 1). For simplicity, grades 1 and 2 are often referred to as superficial frostbite, and grades 3 and 4 as deep frostbite.[10] In assessing severity and determining treatment, both the anatomical structures involved and the extent of the injury must be considered.[11] Figure 1 provides a quick overview.

Pathophysiology: ischemia, inflammation, coagulation

Humans tolerate heat better than cold. In cold environments, the skin of fingers and toes undergoes cyclic vasodilation and vasoconstriction ("hunting response"), which varies in intensity depending on genetic predisposition.[12] If exposure to cold continues, this protective mechanism ceases. The resulting predominant vasoconstriction further reduces peripheral blood flow. Tissue freezes when its temperature drops below its freezing point (approximately –0.5 to –2°C).[13] In cold exposure, skin blood vessels constrict to reduce heat loss. If heat loss is faster than heat delivery by blood, skin temperature drops critically. Frostbite occurs when tissue fluid freezes and forms ice crystals.

- Freezing phase: Skin sensation disappears at 8–10°C. Strong vasoconstriction causes rapid cooling. Ice crystals form in tissues and blood vessels, blocking circulation and causing lack of oxygen. Cells lose water, become dehydrated, and die. Rapid freezing causes larger crystals that mechanically damage cells. The main injury mechanism is damage to microcirculation.[14] During slower freezing, endothelial damage in small peripheral



Cauchy E et al. Retrospective study of 70 cases of severe frostbite lesions: A proposed new classification scheme. Wilderness Environ Med. 2001; 12:248-255 modifiziert nach Oberhammer R et Cauchy E. Erfrierungen in: Alpin- und Höhenmedizin 2. Auflage. Hrsg: Berghold F, Brugger H et al. 2019; 209-217

Figure 1. Classification of frostbite according to affected tissue layers and extent, the need for specific therapy, hospitalization, and prognosis after 2 days. Modified from R. Oberhammer[10]

vessels and intravascular microthrombus formation also play a critical role.[15]

- Thawing phase: Ice melts and water rushes into cells and surrounding tissue, causing swelling. Damaged blood vessel walls leak fluid. Free radicals worsen vessel injury, increasing edema and ischemia.[16]
- Ischemia and reperfusion phase: Inflammatory mediators (PGD_2, PGF_2, TxA_2, TxB_2) and free radicals cause unstable vessel reactions, clot formation, and blockage of small blood vessels. Without treatment, tissue ischemia worsens and superficial frostbite can become deep.[17]
- Late ischemic phase: Persistent clotting and lack of blood flow lead to tissue death and gangrene.[18]

Methodology

This overview summarizes some actual papers on frosbite management, but mainly highlighting data from two recent guideline publications on frostbite treatment:

1. *Wilderness Medical Society Clinical Practice Guidelines for the Prevention and Treatment of Frostbite: 2024 Update*,[1] and
2. *American Burn Association Clinical Practice Guidelines on the Treatment of Severe Frostbite.*[19]

While the first publication was prepared by an expert panel based on existing literature, the second addresses several clinical questions through a systematic literature review following the PICO framework. The American Burn Association guidelines do not define a standard of care for the diagnosis and treatment of acute severe frostbite but serve as a reference for clinicians managing such patients. Clinical bedside assessment always takes precedence in managing individual cases.[19]

There are several protocols for basic guidance in treating patients with frostbite. Among them, the Planica Protocol, developed by the Jožef Stefan Research Institute in Ljubljana, Slovenia, is well known.[20]

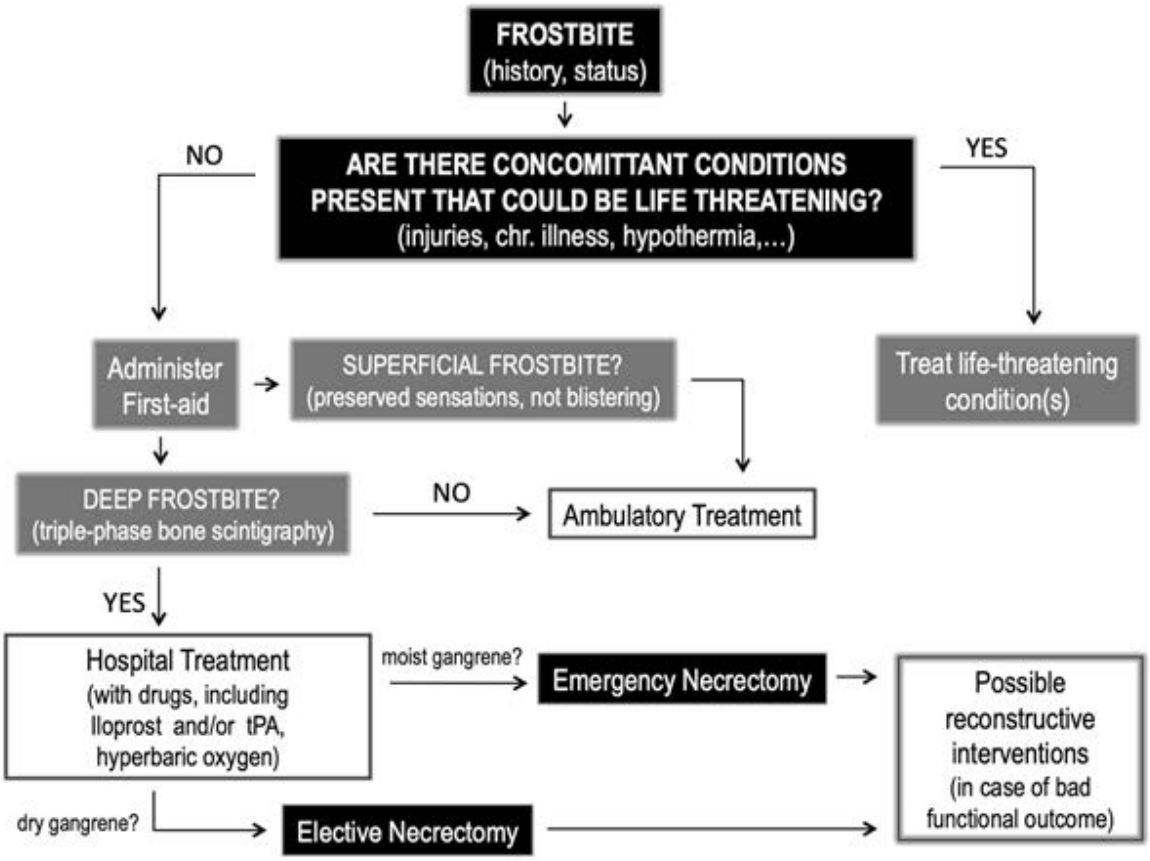


Figure 2. Planica protocol for frostbite management [20]

First Aid on Site
1. Protect from wind and cold.
2. Treat hypothermia and/or severe injuries or illnesses first.
3. Rewarm the affected limb in a warm water bath (37–39°C).
4. Administer ibuprofen 6 mg/kg twice daily if available and not contraindicated.
5. Provide sterile wound care; clear, tense blisters may be drained by needle aspiration, but blood-filled blisters should remain intact.
6. Immobilize and splint the affected limb.
7. Arrange rapid transport to an appropriate hospital.

Hospital Treatment
1. Treat hypothermia and other serious injuries or illnesses first.
2. Rapid rewarming in a warm water bath (37–39°C) until the tissue softens, if not already done prehospital and if spontaneous rewarming has not occurred. Begin analgesia as needed.
3. Perform clinical assessment to determine frostbite severity.
4. Administer ibuprofen (12 mg/kg/day in two divided doses).
5. Ensure tetanus prophylaxis.
6. Debridement: selectively drain clear blisters if tense; leave hemorrhagic blisters intact.
7. Change dressings twice daily using topical aloe vera gel under sterile conditions. Apply loose, dry dressings; elevate and splint limbs to prevent edema.
8. Maintain adequate hydration (oral or intravenous).
9. Start antibiotics, if wound infection is suspected.
10. Consider thrombolytic therapy for deep, extensive frostbite within 24 hours after rewarming, combined with heparin or low molecular weight heparin (LMWH) for 3–5 days.
Intravenous thrombolysis: 0.15 mg/kg over 15 min, followed by 0.15 mg/kg/hour for 6 hours. Use angiography before and during treatment if available. Intraarterial thrombolysis: after vasodilation (e.g., with nitroglycerin or iloprost), administer a bolus of 2–4 mg, followed by 1 mg/hour per catheter over 6 hours for the upper limb.

Iloprost therapy: consider within 72 hours after thawing for deep frostbite. Dosage:

- Days 1–3: start at 0.5 ng/kg/min, increasing by 0.5 ng/kg/min every 30 min up to 2 ng/kg/min for 6 hours/day.
- Days 4–5: begin at the maximum tolerated dose, up to 6 ng/kg/min for 6 hours/day.
- Treatment duration: 8–10 days.
- Monitor for side effects such as nausea, headache, flushing, hypotension, bradycardia, or myocardial ischemia.

Discussion

When following protocols for treating frostbite in the field and in hospitals, it is important not to overlook other emergency conditions (accompanying injuries, hypothermia) and to recognize deep frostbite as soon as possible and begin appropriate treatment early.[20]

First aid

The priority in prehospital frostbite management is always the safety of rescuers and patients. Life-threatening conditions, such as severe hypothermia, take precedence. The patient should be protected from further cooling, ideally by moving to a shelter.[21]

Most authors recommend rapid rewarming in a warm water bath (37–39°C) after removing constrictive clothing.[19,22] The American Burn Association guidelines suggest 37–42°C. Data on the optimal water temperature are limited, but it should likely not exceed 45°C.[23] Frozen clothing should be thawed before removal to prevent tissue damage. The affected limb should be loosely dressed with a sterile bandage and kept immobile.

Because frostbite is a vascular emergency, prompt transport to an appropriate facility is crucial. The time between rewarming and initiation of pharmacologic therapy should ideally be less than 24 hours. If hospital transport can be achieved within 2 hours and refreezing cannot be excluded, rewarming should be deferred until hospital arrival.[1,20] Cycles of thawing and refreezing must be avoided due to the risk of increased tissue loss.

Long-term footwear removal in frostbitten alpinists, who need to evacuate themselves from high altitude by walking should be avoided due to swelling of tissue. Care should be taken, not to rewarm with dry heat (gas burners) or not to open frostbite blisters as this might lead to tissue infection.

Hospital Management

Diagnosis

In most cases, clinical examination 24–48 hours after hospital admission allows accurate assessment. To avoid delays, treatment should generally begin immediately. If thrombolysis is considered, digital subtraction angiography (DSA) or technetium-99 scintigraphy can help assess peripheral tissue perfusion.[23] Bone scintigraphy or SPECT/CT may also assist in early prognosis,[24] though limited availability and evidence mean these techniques do not significantly affect amputation rates or timing.[18] Standard X-rays should be taken if fractures are suspected or thrombolysis is planned.[25]

Therapy

rt-PA (Thrombolysis): Early systemic or intraarterial thrombolysis appears to reduce amputation rates in severe frostbite, though evidence remains limited.[26] Recommended i.v. dosing: 0.15 mg/kg over 15 min, followed by 0.15 mg/kg/hour for 6 hours.[27] For intraarterial therapy: after vasodilation (e.g., nitroglycerin or iloprost), administer a 2–4 mg bolus, followed by 1 mg/hour per catheter for 6 hours. Note that rt-PA use for frostbite is off-label.[28] Concurrent use with iloprost may be beneficial, but evidence is currently weak.[29]

Iloprost: Intravenous iloprost should be considered as first-line therapy for grade 3–4 frostbite within 48 hours of rewarming, particularly when rt-PA is contraindicated or delayed.[30,31] Iloprost was approved for frostbite treatment in the U.S. in February 2024 but remains off-label in Europe.

Heparin/ Low-Molecular-Weight Heparin (LMWH): Evidence for pre- or intrahospital use of heparin in frostbite is limited. It is typically used alongside thrombolysis. Data on combined LMWH and iloprost therapy are insufficient. Clinicians should note that concurrent use of multiple anticoagulant or antiplatelet agents (iloprost, rt-PA, ibuprofen, heparin) increases bleeding risk.[32]

Ibuprofen: Ibuprofen should be started as early as possible due to its anti-inflammatory, antiplatelet, and analgesic effects—12 mg/kg/day divided into two doses, up to a maximum of 2400 mg/day divided into four doses.[33]

Antibiotics: Frostbite wounds are not inherently infection-prone; therefore, prophylactic antibiotics are not recommended. Some experts use them when significant edema develops, believing it may increase susceptibility to gram-positive infections, though evidence is lacking. Systemic antibiotics (oral or parenteral) should be used only for associated trauma, infection, cellulitis, or sepsis.[34]

Pentoxifylline: Pentoxifylline, a methylxanthine-derived phosphodiesterase inhibitor used in peripheral arterial disease, improves blood rheology but lacks controlled studies in frostbite. Hayes et al. recommend 400 mg sustained-release tablets three times daily with meals for 2–6 weeks.[35]

Tetanus Prophylaxis: Unlike antibiotics, tetanus status must always be checked, and prophylaxis administered if necessary.[36]

Hyperbaric Oxygen Therapy (HBOT): HBOT is established for wound healing[37] and may improve outcomes in severe frostbite, even with delayed presentation.[20,38] However, due to limited evidence, cost, and accessibility issues, routine use is not currently recommended.

Conclusions

Frostbite is a vascular and tissue injury caused by prolonged cold exposure, leading to ice crystal formation, microcirculatory damage, inflammation, and ischemia. Nowadays, it is considered a medical emergency and early recognition and prompt management are critical to limit tissue loss. Prehospital care should prioritize patient safety, protection from further cold, rapid but controlled rewarming, pain control, immobilization and rapid transport. Hospital treatment focuses on severity assessment, continued rewarming, pharmacologic therapy (ibuprofen, thrombolytics, iloprost, anticoagulants), selective blister care and supportive measures. Advanced imaging can guide prognosis, and adjunctive therapies such as hyperbaric oxygen may aid recovery in select cases. Timely, protocol-driven intervention reduces complications, prevents progression to deep tissue necrosis, and improves functional outcomes. In case of severe frostibe to functional body parts (fingers, ears, nose), early psychological support is an integral component of acute frostbite management.

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