Cold Injuries

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KEYWORDS

• Frostbite • Limb salvage • Thrombolytics • Bone scan • Urban

KEY POINTS

- Cold injuries can be divided into the following spectrum: frostnip, superficial frostbite, and deep frostbite.
- Under ideal circumstances, severely frostbitten extremities are rapidly warmed and treated with thrombolytic therapy within 6 to 24 hours.
- Many victims of cold injury, particularly those in urban environments, do not meet inclusion criteria for thrombolytic administration.
- Technetium-99m bone scans or single-photon emission computed tomography/computed tomography can predict tissue demarcation within 48 hours, allowing for determination of level of amputation or limb salvage.
- Limb salvage or maximizing limb length with grafts and flaps play a leading role in management.

INTRODUCTION

The populations affected by cold injuries are diverse. Historically, the first cases of frostbite were seen in soldiers on military campaigns. Cold-related injuries almost completely destroyed Napoleon's army during the Russian invasion of 1812 to 1813, and more than a million people succumbed to the condition during World Wars I and II and the Korean Wars.[1] Within the last 20 to 30 years, an increased interest in outdoor sports and recreation led to frostbite injuries within the civilian population, in particular, skiers or mountaineers and others who venture outdoors in cold weather for work. Civilians are also affected by frostbite in the inner cities, hence the term, *urban frostbite*. In an urban environment, most patients who suffer frostbite injuries present in a delayed fashion and sustain repeated cold injuries as a result of psychological issues, intoxication, or homelessness. These social aspects present unique barriers to treatment. Frostbite can also be categorized as experimental, when injuries are inflicted on anesthetized experimental animals' ears and paws in an attempt to elucidate mechanism of injury and test therapeutic modalities.

Irrespective of the population affected by frostbite, the early management of cold injuries relied primarily on the principles of rapid rewarding, watchful waiting and delayed amputation,[2,3] following the adage "freeze in January, amputate in July."[4] The treatment of frostbite then remained stagnant until the 1980s and 1990s, when a paradigm shift in management occurred based on the recognition that the pathogenesis of progressive dermal ischemia was related to the arachidonic acid cascade and the introduction of thrombolytic therapies for attempted limb salvage.[5] Previous investigations into adjuvant treatments such as antiplatelet agents, digital sympathectomies, and hyperbaric oxygen therapy are discussed in this article but have not proven as effective as prostacyclin analogs and thrombolytics in the clinical arena. To appreciate the mechanism by which these various modalities function in frostbite management, it is imperative first to understand the pathophysiology of cold injuries.

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PATHOPHYSIOLOGY

Most frostbite occurs when tissues freeze slowly and form ice crystals. The temperatures necessary to produce this injury are typically less than 28.4°F (−2°C). Injuries are circumferential, progress distal to proximal, and are potentially reversible. Local tissue damage is influenced by the susceptibility of specific body tissues to cold, the cooling rate, the lowest tissue temperature achieved, the duration of the cold exposure and ischemia time, and the rewarming conditions. Skin will freeze faster at lower temperatures but the degree of tissue disruption depends mainly on the duration of the freezing process. Cellular damage occurs as the tissue freezes and when it thaws (during and after rewarming).

Tissue Freezing

Cold exposure of an extremity causes vasoconstriction, which leads to reduced peripheral blood flow and cooling of the skin. This cooling then results in further vasoconstriction. In an attempt to rewarm the extremity and protect against freezing, the body responds with cycles of vasodilation and vasoconstriction transiently every 5 to 10 minutes. This reaction is called the *hunting response*.[6] Unfortunately, this vasodilation then brings cold blood back to the main circulation, which causes the core temperature to drop. At very cold temperatures, the hunting response is blunted to allow maintenance of core temperature at the expense of extremity rewarming. As the extremity is cooled, skin blanches, producing a burning, heat sensation. Once the core temperature decreases, arteriovenous shunting occurs, which shifts blood flow away from the skin. Sensory nerve dysfunction occurs at 10°C, and the digits become stiff and numb. As the skin cools further, blood viscosity increases, producing microvasculature constriction with transendothelial plasma leakage.

During rapid freezing, intracellular ice crystals can form. Except in accidental injuries, such as liquid nitrogen emersions, rapid freezing generally only occurs in experimental conditions. Typically, with slower freezing as seen clinically, ice crystals form initially in the extracellular space.[7,8] Increased extracellular osmotic pressure draws free water across the cell membrane, producing intracellular water loss, hyperosmolality, and decreased cell volume. Extracellular and intracellular electrolyte and acid-base disturbances ensue with a resultant destruction of enzymes. Growing ice crystals can also damage cells and their membranes directly. Endothelial injury leads to microvascular damage, which harms tissues indirectly. This disruption of the microvasculature, along with ice crystal formation in plasma and sludging of red blood cells, results in eventual cessation of the microcirculation.[9]

Tissue Thawing/Rewarming

Most of the tissue damage sustained from frostbite injury is caused by the damaging effects during and soon after rewarming. Once the extremity is rewarmed, the damaged capillary endothelium leaks fluid and protein into the interstitial space, which is manifested by swelling and leads to blister formation. Occasionally, blisters do not form, which signifies either a very superficial injury or an extremely deep injury. Typically, blisters are filled with straw-colored or blood-tinged fluid.[10] The most significant injuries are most likely caused by reperfusion injury with the generation of oxygen free radical formation and initiation of the arachidonic acid cascade with prostaglandin-induced vasoconstriction, leukocyte adherence, and aggregation of red and white blood cells and platelets.[7,11] This mechanism is hypothesized because prostaglandin F_{2a} and thromboxane A_2, which have been implicated in vasoconstriction, platelet aggregation, and thrombosis, are found in the blister fluid.[12] Although the exact timing and sequence of events are not clear, it is evident that these changes will lead to significant tissue loss if no intervention is initiated in the crucial first 24 to 48 hours after rewarming.

CLASSIFICATION

Cold injuries have been classified in a variety of different ways. The simplest is to separate prefreezing and freezing injuries. The prefreezing injuries include trench foot and chilblains or pernio. Trench or immersion foot mainly affects military personnel with prolonged exposure to damp conditions at freezing temperatures. The affected feet may become numb, erythematous, or cyanotic as a result of poor circulation. Chilblain (pernio) occurs in response to repeated cold, nonfreezing temperatures in dry conditions. Symptoms include: 189 burning, pruritus, swelling, erythema, and blistering that can progress to ulceration. Usually, the lesions resolve within 2 weeks but can lead to chronic problems such as vasculitis, typically seen in young, middle-aged women.[6]

Historically, frostbite injuries were classified like burns as a range of first to fourth degree injuries. These categories can be difficult to assess in the field, as often the still-frozen part is hard, pale, and anesthetic. Thus, the previous system was tough to apply and led to confusion among providers. A simpler scheme by Mills and Whaley[13] divides tissue injury into the following