Frostbite

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Contents

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Frostbite is defined as the injury sustained by tissues subjected to temperatures below their physiological freezing point (−0.55 C). The severity of a frostbite injury is related to the temperature, wind chill and the duration of exposure to that temperature, as well as the volume of tissue subjected to cooling. As a result a description of a frostbite injury is somewhat of an umbrella term used to describe wounds ranging from those with minimal tissue damage over small areas to substantial necrosis of entire limbs, necessitating amputation. Advances in frostbite management have improved the potential outcome in frozen injuries, and thus it is important that front-line medical staff can recognise and treat frostbite effectively [1].

9.1 Aetiology

Frostbite typically affects the extremities with 90 % of injuries affecting the fingers and toes although the nose, ears and external genitalia are

L. Téot et al. (eds.), *Skin Necrosis*,
DOI 10.1007/978-3-7091-1241-0_9, © Springer-Verlag Wien 2015

Table 9.1 Aetiological factors predisposing to frostbite injury

Extrinsic factors	• Extreme cold exposure • Innapropriate clothing • Homelessness • Smoking
Systemic pathology	• Vascular/raynauds disease • Diabetes • Psychiatric illness • Previous frostbite injury
Pharmacologic	• β-blockers • Sedatives • Anxiolytics • Illicit drugs
Injury	• Head injury • Spinal cord injury • Multi-trauma • Injuries compromising circulation

also commonly affected [2]. Although historically it was most frequently observed in military personnel, there has been a shift over the last few decades with increasing numbers of civilians being affected. Modern epidemiological evidence suggests that high-risk groups still include those in military organisations but also the homeless, extreme sports enthusiasts, those in poor health, individuals intoxicated through drink or drug use and genetic susceptibility, e.g. people from warm climates (see Table 9.1) [1, 2].

9.2 Classification of Frostbite

There are a number of ways one can classify frostbite injuries, and the most commonly utilised classification is simply one of mild or severe injury, but the first predictive outcome classification was suggested by Cauchy et al. 2001 [3]. Cauchy's classification is based on anatomical location, radiotracer uptake on technetium scanning and skin blistering. This was based on a retrospective study of 70 frostbite patients presenting to the Department of Mountain Medicine in Chamonix, France (Table 9.2).

9.3 Pathology

A frostbite injury results both from the direct and indirect effects of freezing and pathologically is characterised by a continuum of overlapping pathological phases that ultimately cumulate in cellular ischaemia and necrosis of the affected tissues. These phases can be broadly divided into the following:

- Pre-freeze phase
- Freeze-thaw phase
- Vascular stasis phase
- Late ischaemic phase

9.3.1 Intracellular Effects

The direct effects of freezing are principally due to the formation of ice crystals within the tissues themselves. These crystals increase the oncotic pressure within the extracellular space, dehydrating cells via the osmotic movement of water out of the cellular membranes and disturb the intracellular homeostasis. With the rewarming of tissues, the crystals melt producing interstitial tissue oedema. Indirectly the freezing injury stimulates

Table 9.2 Predictive classification of frostbite injuries affecting the limb extremities

Severity grade	Lesion location (day 0)	Radioactive tracer uptake on bone scan (day 2)	Character of blistering (day 2)	Likely outcome
1.	Not visible	N/A	None	No tissue loss or long-term sequelae
2.	Distal phalanx	Hypo-fixation	Clear	Soft tissue loss with nail changes
3.	Mid-phalanx	Absence of uptake at phalanx	Haemorrhagic	Amputation of digit. Functional sequelae
4.	Carpus/tarsus	Absence of uptake at carpus/tarsus	Haemorrhagic	Extensive amputation with likely sepsis or thrombosis. Functional sequelae

Adapted from Grieve et al. [2]

the release of a variety of pro-inflammatory cytokines.

9.3.2 Extracellular Effects: The Freeze-Thaw-Refreeze Injury

Initial exposure to cold temperatures results in an immediate, localised vasoconstriction. This may sometimes be followed by a transient vasodilatory reflex known as the "hunting response", or cold-induced vasodilatation. This physiological reflex results in a redistribution of flow from the core and is thought to be a primitive reflex to protect the extremities from freezing. Ultimately this results in a drop in core body temperature and furthermore is ineffective in protecting the peripheries against extreme cold stress. The reduced blood flow secondary to vasoconstriction in turn further exacerbates localised cooling producing a vicious cycle of ever-increasing vasoconstriction and tissue cooling. An oedematous state results through a combination of increased plasma viscosity, microvascular damage and fluid migration. The microvascular (endothelial) damage produces a pro-thrombotic environment through activation of the clotting cascade in which microthrombi form, occluding the capillaries, resulting in ischaemia. When such a time as tissue rewarming occurs, further microvascular clot occlusion occurs due to the promotion of a pro-thrombotic state through the lysis of frozen cells. Local mast cells degranulate in response to the lytic cell membranes, releasing histamine which further increases vascular permeability and oedema. The end point of all of these processes is potentially devastating local tissue ischaemia, and as first noted by Baron Larrey, Surgeon General to Napoleon, the most significant ischaemia, and therefore tissue necrosis, is seen in injuries that freeze, thaw and are then frozen again.

9.3.3 Long-Term Sequelae

It should also be appreciated that aside from these immediate-type effects seen following a freezing injury, there is also long-term pathological damage sustained by tissues which may result in chronic dysfunction or impact upon the patient many years post-injury. Although rare, one of the most serious observed syndromes is probably related to chronic vasomotor dysfunction, manifesting clinically as chronic pain affecting the previously frostbitten area that is often unresponsive to conventional analgesia and requires anaesthetic or pain specialist input. Those that develop these complex regionalised pain syndromes frequently also suffer with associated problems such as paraesthesia and cold intolerance. It should also be noted that all patients who have experienced frostbite are at increased risk of further

future episodes (presumably again secondary to vasomotor dysfunction) and all patients must be warned of this and given appropriate preventive advice. More serious long-term sequelae of frostbite that have been reported include the malignant transformation of frostbitten tissues and bone and joint pathologies including osteoporosis and arthritic changes [1].

9.4 Clinical Evaluation of Frostbitten Patients

9.4.1 History

Critical details of the patient history include the likely temperature, duration and timing of exposure, as this will help predict the severity of injury and may affect subsequent management. It is also important to obtain information relating to the patient's premorbid state such as peripheral vascular disease and pertinent risk factors such as smoking or the use of β-blockers.

9.4.2 Examination

In rare cases frostbite injuries may present as a purely uniform frozen injury, but more frequently there is a mixed clinical picture with overlapping areas between deeper frozen tissues and more superficial nonfrozen tissues. Even in the case of a purely frozen injury, there is much variation in severity from the lesser affected forms (frostnip) to large areas of frozen tissues or indeed whole limbs.

It can be seen that frostbite presents in variable fashions and the injury evolves with time, and thus one is often unable to determine the full or likely extent of injury for some time after the injurious cooling from clinical examination alone, illustrating the importance of a thorough history. Nonfreezing injuries may be managed locally without the need for specialist intervention, and they typically follow a short exposure to (relatively) warmer temperatures and involve the feet most commonly with patients complaining initially of localised numbness and or paraesthesia. As the tissues rewarm severe pain is experienced with the rapid onset of a reactive hyperaemia and tissue oedema. The pain is usually transient but may become chronic with patients suffering long after tissues have recovered. Actual tissue loss is uncommon in these cases with most injuries only exhibiting mild discolouration, and very occasionally small areas of watery blisters may develop.

Any potentially serious frostbite injury however must be discussed with a suitably experienced unit for consideration of patient transfer, especially if the patient has presented less than 24 h post-injury as they may be a candidate for thrombolytic therapy.

9.4.3 Radiological Investigations in Frostbite

Due to some of the difficulties outlined in accurately assessing the initial severity of tissue devitalisation in frostbite injuries (hence avoiding early surgical debridement), a variety of radiological investigations have been suggested as clinical assessment adages. It is important to appreciate however that no radiological investigation is currently predictive in isolation and these studies are designed only to augment clinical opinion in cases that may be unclear.

Many investigations have been suggested over the years to image frostbite injuries, but the most clinically useful appear to be technetium 99 (Tc-99) triple phase scanning and magnetic resonance angiography. There is convincing evidence from a large retrospective review of 92 patients with severe frostbite injuries that Tc-99 scanning in the first few days can predict the subsequent level of amputation in up to 84 % of cases [4]. However further large-scale studies are required in this area, and currently there is little role for complex imaging in routine and less severe frostbite cases. Possible exceptions to this include severe injury with early presentation and no associated traumatic injuries, those rare cases where early surgery is being undertaken, or if thrombolytic therapy is being considered.

9.5 Acute Frostbite Management

9.5.1 Prioritisation of Life-Threatening Injuries and Specialist Referral

It is vital to appreciate that patients presenting with frostbite frequently present with coexisting severe and life-threatening emergent conditions such as hypothermia or significant traumatic injury. In line with the management of any emergency situation, such coexisting morbidities must be treated and stabilised before commencing treatment or transferring a patient to an expert centre for localised frostbitten tissue(s). Hypothermia should be corrected and core temperature should be raised to 34° [2] In any case, frostbite injuries often occur in remote regions where transfer or access to expert centres is not immediately possible and immediate management must be commenced locally. In today's technologically advanced age, those treating frostbitten patients can seek expert advice through the use of satellite phones and the Internet; indeed, many remote facilities rely on such technology.

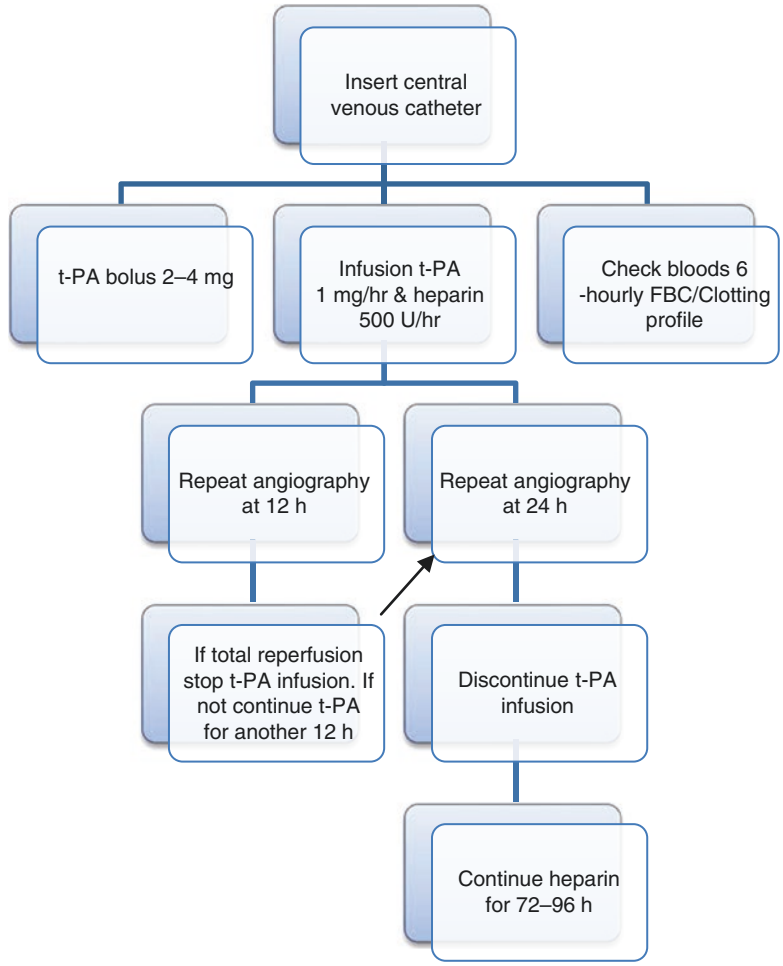
9.5.2 Prevention of Refreezing and Differential Approach to Rewarming

It is also imperative that rewarming of the locally frostbitten tissues must only be commenced once there is absolutely no risk of refreezing as this will result in further tissue damage; similarly it is advised that frostbitten tissues should not be rubbed or massaged for similar reasons. Once a patient with a frozen injury has been secured in a safe environment and any emergent conditions stabilised, directed therapy can be commenced. In such injuries (be they minor areas of freezing or more extensive), the principal goal of treatment is to rapidly rewarm the affected area in a water bath with an antiseptic agent such as chlorhexidine in an attempt to preserve the dermal circulation. State of Alaska guidelines suggest a warming bath of 37–39 °C, whilst others suggest 40–41 °C [1, 5]; in practice, especially if in the field, it is nearly impossible to maintain exact water temperatures and the critical factor is that the water should be warm, not hot, as the tissue will be neuropathic and one must avoid scalding. The rewarming should continue for a minimum of 30 min or as long as necessary for all affected tissues to be defrosted, thoroughly rewarmed and pliable exhibiting a deep-red or purple colour, carefully monitoring the water temperature throughout. This 30 min regime of rewarming should be repeated at twice-daily intervals until such a time that there is either evidence of tissue regeneration or clear demarcation of necrotic tissues, and the tissues should be kept warm and dry during in-between periods. There are a variety of commercial whirlpool or foot spa devices suitable for this purpose. It should be noted that a differential approach to rewarming is used in true frozen injuries in comparison to milder, nonfrozen cases. Milder cases should be warmed more slowly at normal room temperature, as rapid rewarming may exacerbate injury.

9.5.3 Pharmacological Support During Rewarming

Patients with frostbite require pharmacological support with respect to analgesia, vasodilatation and antibiosis, but certain caveats apply to the drugs that should be used and those that should be avoided. The rewarming of frozen tissues is frequently accompanied with severe pain and all patients must therefore be provided with judicious amounts of analgesia. Analgesia should always, where possible, include ibuprofen due to its selective anti-prostaglandin activity which may improve tissue perfusion in addition to providing analgesia. Some authors have recommended avoiding aspirin-based analgesia as it irreversibly blocks prostaglandin function and some vasodilatory prostacyclins, which may be beneficial in the healing wound [6]. Regarding vasodilators, surgical sympathectomy used to be used routinely but has now been superseded by vasodilatory drugs such as iloprost. Iloprost should be infused over a 5-day period in an appropriate high-care facility that is capable of performing regular (at least every 30 min) patient observations. Most units have locally approved infusion protocols for

Table 9.3 Treatment algorithm for t-PA infusion



Adapted from Bruen et al. [7]

iloprost but a suggested regime is to commence a 10 ml/h infusion of 100 mcg iloprost in 500 mls 0.9 % saline. The dose can then be titrated up incrementally to a maximum of 50 mls/h or until there are observed side effects and should be run over a period of 6 h in any one day. Necrotic or devitalised tissues are at risk of infection which may secondarily worsen tissue damage, and prophylactic broad-spectrum antibiotics, such as co-amoxiclav, should be administered together with tetanus vaccination where appropriate.

Thrombolytic therapy (see Table 9.3) with tissue plasminogen activator (t-PA) aims to lyse the multiple small intravascular thromboses that occur during the vascular stasis phase of frostbite and restore perfusion to the affected area, improving tissue survival. The use of t-PA reduced amputation rates from 41 to 10 % in a study when administered within the first 24 h of injury [7]. Treatment with thrombolytic agents is not without risk (severe haemorrhage) and thrombolysis should only be reserved for patients presenting with severe frostbite within 24 h of injury and without any contraindications to treatment such as concurrent traumatic injuries. This form of treatment should only be considered in

facilities that are equipped with high-dependency care facilities and are familiar with caring for patients undergoing thrombolytic therapy. Aside from the risk of associated major haemorrhage, the restoration of perfusion following thrombolysis to a limb may cause a compartment syndrome (secondary to oedema from damaged capillaries), and the requirement for prophylactic fasciotomies must always be considered. The vasodilatory agent iloprost has been shown to be a suitable alternative to t-PA in a randomised controlled trial [8]. Cauchy et al. randomised 47 patients with frostbitten digits to receive either buflomedil, iloprost or iloprost + t-PA treatments following rewarming and antiplatelet therapy. Results showed that the risk of amputation was significantly lower in the iloprost and the iloprost + t-PA groups compared with the group that received buflomedil alone. No evidence was gained to suggest superiority of either treatment, and the study recommends that prostacyclin (iloprost) be used in patients with severe (grade 3) frostbite and the addition of t-PA be reserved for severe (grade 4) frostbite [8].

9.6 Post-thaw Frostbite Care

9.6.1 Management of Blisters

Areas subject to a freezing insult will frequently exhibit blistering, and a clinical decision must be taken as to the management of such areas. Generally speaking small blistered areas that are not tense with clear fluid should be left intact as de-roofing these blisters may increase susceptibility to opportunistic infection. Blisters are sterile until they burst, and immunoglobulins have been shown to be present in blister fluid [9]. However in most circumstances, more extensive areas of tense blistering or haemorrhagic blisters should be carefully de-roofed in aseptic conditions by a specialist. In rare cases where the patient is at particular risk of opportunistic infection, such as in dirty wounds, or if the patient is known to be colonised with as resistant organism, then it may be appropriate to leave all blistered areas intact to reduce the risk of a potentially devastating tissue infection, and all such cases must be discussed with a specialised unit. Topical aloe vera is a commonly suggested therapy in minor frostbite cases due to its anti-prostaglandin actions, and whilst there is little evidence to recommend its use, it may be considered in minor cases [1, 2].

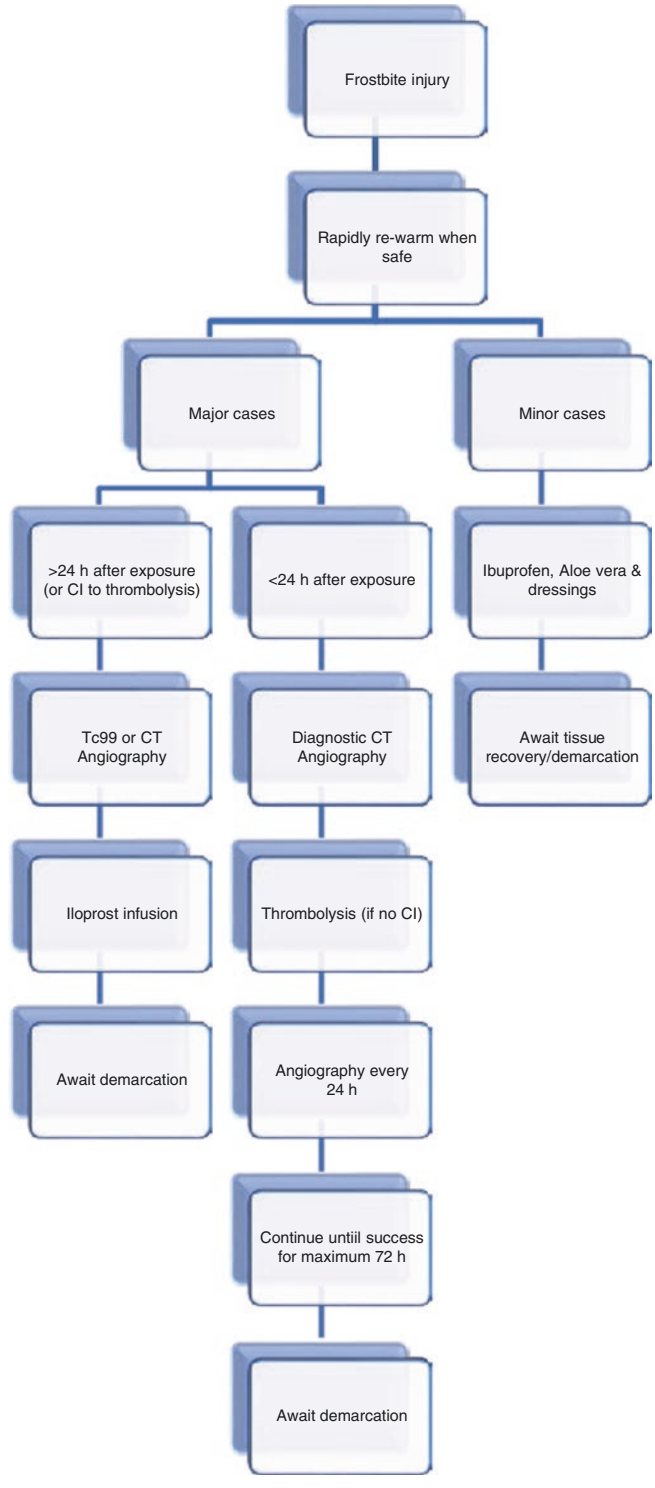
9.6.2 Physiotherapy Protocols

Regardless of the clinical appearance, the affected area needs to be elevated in order to reduce venous stasis and tissue oedema. Similarly, all affected tissues will be fragile and easily disrupted through even gentle mechanical stresses; thus, all lower limb injuries must be placed on a strictly non-weight-bearing status to protect against ischaemia. These measures are designed to prevent extension of injury through progressive tissue oedema, thrombosis and ischaemia, and frequently areas that initially appeared non-salvageable will recover.

9.6.3 Surgery

Early surgical debridement of frostbite injuries is nearly always contraindicated, as the reversibility and progression of the frostbite injury cannot be quantified in the early stages. Debridement is best delayed until definitive demarcation of devitalised tissues at approximately 6–8 weeks post-injury. With appropriate management surgery is frequently not required, despite the initial appearance of the injury. Similarly it may be appropriate to leave demarked areas to autoamputate if expert surgical input is not available or if the patient has substantial co-morbidities making anaesthesia unsafe. Exceptions to this conservative approach to surgery include injuries with uncontrolled severe infection, concurrent severe limb trauma and compartment syndrome, all of which may require urgent limb surgery. Fortunately, these are infrequent and are more commonly seen in freeze-thaw-refreeze injuries (Table 9.4).

Table 9.4 Treatment algorithm for frostbite injuries



Adapted from Hallam et al. [1]

9.7 Summary Points

- Prevention is paramount.
- Treat any serious or life-threatening conditions as a priority.
- Do not rewarm frozen tissues until there is no risk of refreezing occurring.
- Rewarm nonfreezing cold injuries slowly in air.
- Rewarm freezing cold injuries at 37–39 °C or 40–41 °C in a whirlpool device or foot spa for minimum 30 min with a mild antiseptic, and continue treatment twice daily until improvement is seen.
- Any patient with actual tissue loss should be given empirical broad-spectrum antibiotics.
- Discuss all significant cases with a specialised unit.
- Consider treatment adjuncts such as thrombolysis following discussion with a specialised unit.
- Avoid early surgical debridement.

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