

Frostbite in an Adolescent Football Player: A Case Report

Michael Rivlin, MD*†; Marnie King, OTR/L‡; Richard Kruse, DO‡;
Asif M. Ilyas, MD*†

*Department of Orthopaedic Surgery, Thomas Jefferson University Hospital, Philadelphia, PA; †Rothman Institute, Philadelphia, PA; ‡Department of Pediatric Orthopaedics, A.I. duPont Hospital, Wilmington, DE

Objective: To present the case of vascular compromise of a finger from a confluent circumferential blister due to an inappropriately applied commercial cold pack in a high school athlete and to describe the mechanism of iatrogenic injury, acute surgical management, rehabilitation, and pathophysiology of frostbite and constriction injuries.

Background: A 17-year-old male football player presented with a frostbite and constriction injury to the index finger secondary to prolonged use of a cooling pack after a mild traumatic injury to the digit. He developed a prolonged sensory deficit from thermal injury, as well as acute vascular compromise requiring urgent operative intervention.

Differential Diagnosis: Frostbite and constriction injury to the index finger.

Treatment: Emergency surgical decompression and occupational therapy.

Uniqueness: Frostbite injuries can occur iatrogenically because of inappropriate use of cooling devices or gel packs. Fingers are commonly injured extremities that are particularly susceptible to frostbite and compression injuries. To our knowledge, no case of vascular compromise from the blister constriction of digits has been reported.

Conclusions: Patients and their caregivers must be educated about how to properly use cooling devices. Clinicians need to fully evaluate patients with iatrogenic frostbite injuries, giving particular attention to neurovascular status, and must recognize the need for surgical release of constriction syndrome to prevent substantial morbidity.

Key Words: constriction syndrome, freezing, cryotherapy, vascular compromise, hand injuries, ice packs

Frostbite injuries are common in colder climates and during winter recreational activities. The most frequently involved areas are the distal and terminal structures, such as the fingers, toes, nose, and ears. With the wide acceptance of the rest, ice, compression, and elevation protocol, iatrogenic frostbite has become a risk that cannot be ignored. Thermal injuries have been described in the literature secondary to the use of ice packs and other cooling objects and devices.^{1–3} We present the case of a teenaged athlete who developed vascular compromise of a finger from a confluent circumferential blister due to an inappropriately applied commercial cold pack on the football field. To our knowledge, no case of vascular compromise from the blister constriction of digits has been reported in the literature. The patient's legal guardian provided written informed consent for this case report.

OBJECTIVE

A healthy, 17-year-old, right-hand-dominant, male high school football player fell after being tackled, injuring his right index finger because of contact with the ground. On the field, a coach was reported to have applied a commercially available cooling gel pack, wrapping it around the athlete's finger directly on the skin with an elastic wrap. The patient did not recall being instructed to remove it and believed that leaving it on could only help. After 2 hours, he removed the cooling pack and noticed that his index finger and the radial half of his uninjured middle

finger were red and swollen. He did not experience any pain at the time. He was evaluated that same day in a local emergency department where radiographs of the finger showed no bony injury or other condition, and he subsequently was diagnosed with a low-grade sprain of the right index finger. Per family report, the skin appeared intact at the time. Generic discharge instructions for managing sprains that detailed icing techniques were provided. The patient did not apply any more cold to the affected area after evaluation in the emergency department. On the next day, he noticed that his right index finger and the border half of his middle finger that was initially in contact with the cooling gel pack were covered with a large confluent blister. Later the same day, he presented to our outpatient orthopaedic office for evaluation without an appointment.

On encounter, he denied any pain but described numbness in his index fingertip on his right hand. His initial examination revealed a grossly dusky distal phalanx of the right index finger with a well-demarcated, large, confluent, circumferential tensile blister and hemorrhagic areas over the volar aspect (Figure 1). The blister appeared to create a tourniquet effect that prevented blood flow to the fingertip. Most of the tissue involvement demonstrated second-degree frostbite characterized by erythema and a large confluent vesicle with clear fluid content (Table). Some areas, mainly over the palmar aspect of the finger, had third-degree tissue involvement denoted by the hemorrhagic area of blistering and dusky discoloration of

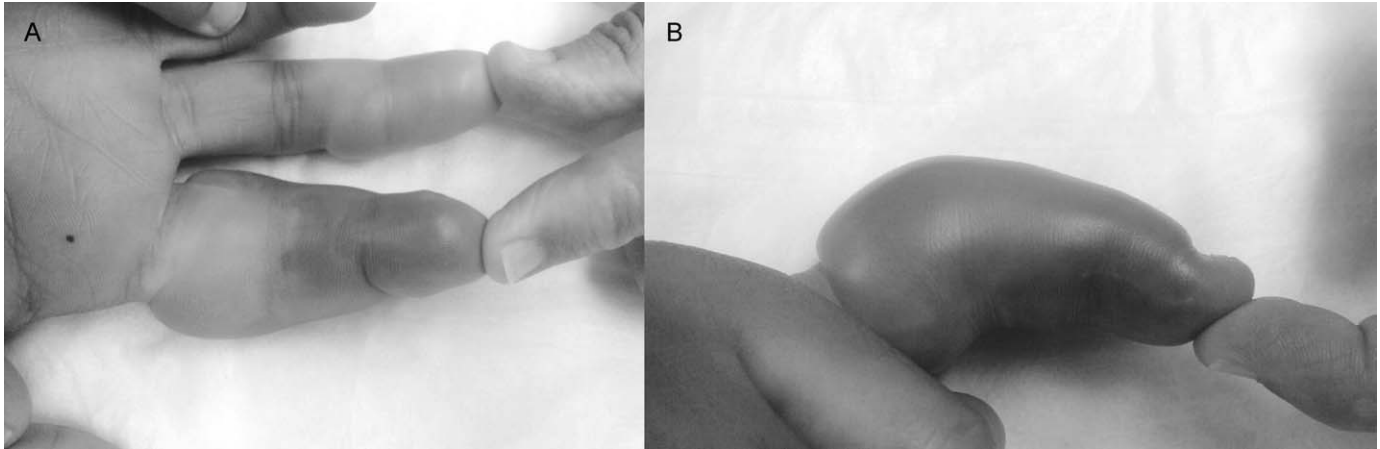


Figure 1. A and B, Initial presentation of the finger demonstrating circumferential blister and distal cyanosis.

the underlying tissue. The fingertip was cold, and the distal phalanx had no capillary refill at that time. His digit was grossly insensate distal to the blister, and he felt only deep pressure. The patient held the finger in a partially flexed position and had no gross pain on gentle, passive motion of the finger. Active range of motion was trace. He had no isolated band of tissue that created a tourniquet effect; the entire blister appeared to function as such. The long finger had a tense blister, which was not circumferential, on the radial fingertip alone. Sensation and circulation were intact. No fracture was seen on radiographs. He had no other injuries.

DIFFERENTIAL DIAGNOSIS

Many conditions or insults can cause blisters or vesicles to form. Burns, frostbite, and chemical agents can cause similar-appearing wounds that are differentiated easily if surrounding circumstances are known. With fractures, shearing-type insults, and other traumatic injuries, blood blisters can form secondary to tissue damage. Allergic reactions or infections also can cause vesicular appearance and sometimes can create bullae.

Compartment syndrome may present as loss of sensation to the affected finger or extremity due to inadequate blood flow. In these cases, entire muscular compartments are affected by a high-pressure gradient usually from internal sources, such as bleeding, causing ischemia and necrosis of the tissues within the compartment. Although these are characteristically painful, nerve damage or anesthesia may blunt the discomfort.

TREATMENT

Given the constriction and impending ischemic compromise of the finger, the patient was indicated for emergent surgical decompression. During the procedure, we longitudinally opened the tensile blisters and released the fluid, thereby decompressing the constriction at the base of the finger (Figure 2A through C). Given the dusky skin of the finger, approximately 0.5 inches (1.27 cm) of nitroglycerin paste was placed on the radial and ulnar aspects of the base of the finger in the constricted area to help vasodilate the digital vessels. We noted degloving of the epithelium of the entire index finger distal to the site of constriction. A small margin of devitalized epithelium was debrided to prevent it from becoming a secondary source of constriction on the finger. Given that only the epidermal layer was lifted off by the blister fluid, skin graft was not indicated. The finger compartments were soft and compressible, and no signs of compartment syndrome were noted. Finally, use of intraoperative Doppler ultrasound now confirmed active perfusion across the constriction site to the tip of the finger along both the radial and ulnar borders of the finger. If we had not identified adequate flow, we could have accomplished open vascular exploration and arteriolysis or arterial bypass. Upon closure, we irrigated the wound and applied silver sulfadiazine ointment. A wrist-based volar splint was applied in neutral position. The patient began antibiotics for prophylaxis, and we confirmed he was immunized against tetanus.

He started occupational therapy 4 days after the surgical intervention. At that time, traction splinting was used to position his index finger in extension using a hook glued to his fingernail (Figure 2D). Active motion was encouraged

Table. Classification of Freezing Injury⁵

Classification	Depth	Characteristics
Superficial		
First degree	Superficial skin involvement	Edema, erythema
Second degree	Full-thickness skin involvement	Blistering, desquamation
Deep		
Third degree	Subcutaneous tissue involvement	Blue-gray discoloration, hemorrhagic blisters
Fourth degree	Deep tissue involvement	Deep necrosis to muscle, tendon, bone, etc



Figure 2. A and B, Release of the constriction of the finger. C, The affected finger 2 weeks postinjury. D, Daily exercises with physical therapy were accomplished with a custom-made splint.

at the metacarpophalangeal and proximal interphalangeal joints while he was in traction for 6 more days. His dressing changes included application of silver sulfadiazine to the open wound. His therapy regimen initially concentrated on elevation and exercises that included active-assisted range of motion, later transitioning to active range of motion. He also had activities that incorporated the use of therapy putty (AliMed, Inc, Dedham, MA), finger extension, and grasp-and-pinch exercises modified and graded for pain and finger circulation. After 24 days, the devitalized skin and the nail had sloughed off, the underlying skin was pink with good vascularity, and new nail had begun to emerge. On postoperative day 25, conditioning and exercise training was added; however, he felt throbbing in the finger when he ran. His grip strength was 70% of that of the other hand; lateral pinch, 52%; and tripod pinch, 47%. On postoperative day 28, his skin was almost completely closed; however, tenderness to touch persisted, so the gauze dressing was replaced with a gel polymer digital cap (Silipos; Isokinetics, Inc, De Queen, AR). On postoperative day 30, sensory testing indicated complete deficit to all modalities from the distal interphalangeal finger flexion crease distally only on the palmar surface. On postoperative day 32, he had full extension and flexion of all finger joints. He could catch and throw a football with the digital cap on the index finger. Dribbling a basketball was uncomfortable. Running did not cause throbbing, and he no longer had to keep the finger elevated. On postoperative day 35, his sensation improved to 2 mm distal to the distal interpha-

langeal crease. He was able to begin jogging and strength and agility training with the football team by postoperative day 40. By final follow-up (postoperative day 46), normal nail growth had resumed, and sensation of his fingertip had improved; however, residual sensory deficit at the fingertip persisted. By 10 weeks after surgery, he reported that he was ready to return to participating in football. He indicated that his tactile sensation was subjectively adequate to activities of daily living and sports. He practiced with caution with the digital gel sleeve under his glove. He had no restrictions but was instructed to inspect his finger multiple times throughout participation in sporting activities. In addition, he avoided extremes of temperatures and carrying thin, hard handles (eg, buckets, plastic grocery bags) that would stress his interphalangeal joints for about 6 months to protect reinnervation.

DISCUSSION

Fingers are uniquely prone to vascular compromise with frostbite injuries. Freezing of the digits frequently is encountered because we often come in contact with cold surfaces or materials through our interaction with the environment. We rely so much on hand function that protecting fingers from the elements during recreational activities, vocational exposures, or everyday life sometimes may be difficult. However, given their anatomy, fingers may be prone to frostbite injuries. Their small size and tight fascial compartments provide easy paths for warmth to escape. Furthermore, limited blood supply and little

collateral circulation put the oxygen supply at risk, especially with the application of constriction, such as tight bandages or wraps or, as we saw in our patient, a commercial cold pack wrapped around the fingers. In addition to these factors, an adaptation to maintain core body temperature in a cold environment or, as in our patient, the local cold affecting the digits may further reduce finger perfusion as the body shunts blood from the extremities.

Frostbite injuries are well described in the literature. Until recently, researchers have not been able to clearly define which temperature below freezing produces frostbite. Investigators⁴ have shown that injury may occur even after brief finger contact with highly conductive materials (conductivity close to that of metals) in 3 seconds at the temperatures of a conventional freezer (-15°C or 5°F). As commercial cooling packs and other cryotherapy devices become more efficient and powerful, the chances of these treatments causing more damage than the injury itself are increased. Melting ice packs (water-ice combinations) predictably produce a 0°C environment and likely decrease iatrogenic frostbite. However, cooling gel packs and frozen items (ie, items stored in freezers or ice [not in melting phase]) may create environments that are below freezing and may be more dangerous to use.

External insult to the extremities often can affect deep anatomic structures. Given that constriction syndrome and compartment syndrome may cause similar abnormalities and have similar mechanisms, constriction syndrome implies an external agent that impedes circulation from reaching the distal parts of the extremity. Constriction syndrome is a circumferential obstruction, whereas compartment syndrome may involve 1 division (compartment) or multiple divisions of the extremity. Compartment syndrome inherently implies an internal pressure gradient through which blood cannot flow. The finger is a unique structure because it depends on 2 well-defined but highly vulnerable digital vessels. Compromise of these vessels by compression, constriction, or transection will result in immediate digital ischemia and potential demise of the digit.

The classification system used for cold injuries according to severity is similar to the categorization used for burns. McAdams et al⁵ explored the once commonly used 4-stage classification in degrees and refined it for depth of tissue injury (Table). Cases of first- and second-degree frostbite are categorized as superficial insults. They are recognized by local effects limited to the skin, with advanced forms causing blisters that later desquamate and form an eschar. Third- and fourth-degree freezing injuries involve the subcutaneous tissues and other deep tissues, respectively. These stages present with hemorrhagic blisters and necrosis and, eventually, mummified black tissues on the spectrum. On the microscopic level, cellular damage and physiologic changes to the changing environment cause tissue destruction. In addition to macroscopic mechanical destruction, ice crystal formation in the extracellular environment leads to water shifting outside the cell, causing dehydration and cell death that progresses to necrosis of the affected tissue bed.⁶ Inflammation, vascular stasis, and thromboses lead to local ischemia on the cellular level. Understanding the microscopic pathologic process of freezing has helped guide the development of treatment principles for these injuries.

Whereas the general management of freezing injuries starts with rewarming, the offending cold source in cases of iatrogenic frostbites most often has been removed before presentation and the environment has warmed up the affected area. The protocol for treating frostbite injuries has been described by Su et al⁷ based on the initial guide of management of McCauley et al.⁸ The authors recommended careful monitoring if the tissue involvement is superficial. However, according to their treatment scheme, deep-tissue involvement warrants further investigation and possible surgical intervention. They used a triple-phase bone scan at 48 hours and 5 days to delineate blood flow and viable areas to aid in the debridement efforts by 10 days after initiation of treatment. Some debate has surrounded the utility of bone scans, and their clinical use for this purpose has been questioned.⁹

The management of blisters has been a topic of long-standing debate. Researchers¹⁰ have thought that the contents of these vesicles are similar in composition to those seen in burns (high in prostaglandins and thromboxanes), so they may predispose surrounding tissues to ischemia and produce vasoconstrictive effects. The accepted practice is to leave blisters alone unless they are ruptured, are too tight, or become infected.¹¹ However, others⁹ advocate opening the clear vesicles and draining the hemorrhagic ones to eliminate the potentially damaging effect of the fluid contents. As demonstrated in the presented case, contiguous vesicles may become so taut that they may temporarily or permanently cut off circulation to distal aspects of the digits on the hand.

Thermal injuries due to cold exposure have long been compared with burn injuries. The mechanism of tissue injury is similar; the presentation and clinical picture also coincide. However, the pattern of injury is greatly different for reasons that may be due to the temporal relationship and the analgesic and anesthetic effects of the cold item. Cooling therapy provides hypoalgesic or analgesic effects locally and possibly centrally by lowering the pain threshold.^{12,13} Cryotherapy or contact with other cold objects may create a dangerous scenario in which the analgesia locally leads to the inability to feel the pain associated with the onset of permanent nerve damage. Other adaptive responses, such as the hunting response of the extremities, have long been implicated to prevent cold-induced ischemia. This process, also called *cold-induced vasodilatation*, has unknown effects on the development of frostbite injuries of the acral areas.^{14,15}

The presented case demonstrates a unique clinical consequence of cold injury. Two seemingly distinct processes led to the substantial morbidity of an otherwise healthy teenaged athlete. The frostbite led to paresthesia of the involved digits and disabled the conscious protective response that usually is mediated by pain to remove the offending agent (commercial cold pack). This is also likely the reason the patient did not notice or describe pain, delaying his presentation to us. The prolonged freezing caused permanent nerve changes and sensory deficit. In addition, the tissue damage from the freezing induced blister formation. The blister that propagated circumferentially around the digit created a tourniquet-like constriction that cut off circulation to the distal portions of the finger. If the direct, cold-induced nerve damage and the indirect vascular compromise had not been addressed

urgently, irreversible damage and compromise of the affected digit might have resulted. The lack of circulation, as well as frostbite, first damages the nerves and then the other tissues. If the injury had been left untreated, further tissue death might have followed and involved other structures of the finger, requiring amputation.

Despite widespread use and acceptance of the rest, ice, compression, and elevation protocol, inadvertent injuries can happen from improper application of commercial cold packs. Children may be even more susceptible to these adverse events. Furthermore, long-term complications in children have been reported because of the incompletely developed anatomy and physiology of the affected areas. When these injuries occur near open growth plates, growth retardation and physeal arrest may occur.¹⁶ Frostbite also has been implicated in early arthritis of children with a history of such wounds.¹⁷

In the case of circumferential confluent blistering, distal ischemia may ensue and cause a constriction syndrome, with vascular and then ischemic changes leading to irreversible tissue damage. The external circumferential constraint functions like a tourniquet, which may lead to insufficient blood flow to the distal structures and requires immediate surgical release to prevent necrosis and neurologic dysfunction, as was presented in our case.

UNIQUENESS

In this report, a teenaged athlete developed a prolonged sensory deficit from thermal injury and acute vascular compromise, requiring urgent operative intervention because of the constriction syndrome caused by the tense circumferential blister on his finger. The triggering insult was a commonly available cooling gel pack that many in the athletic population use.

CONCLUSIONS

Cooling and elevating areas of traumatic injury are effective and widely used ways to decrease swelling, erythema, and pain. Clinicians must educate patients and their caregivers about how to properly use cooling devices, especially commercial gel packs. As seen in the case presented, improper icing methods can cause substantial, long-standing morbidity. Patients with iatrogenic frostbite injuries need to be evaluated fully, giving particular attention to neurovascular status, because compromised circulation can lead to permanent neurologic deficit. Constriction syndrome, in which a peripheral constriction causes decreased blood flow to the distal aspects of

appendicular structures (eg, fingers, toes), has to be recognized and the constriction has to be surgically released, because delay in treatment may carry substantial morbidity. Proper wrapping techniques that ensure loose-fitting, intermittent application of cooling devices without direct skin contact and careful monitoring of the affected area can prevent serious freezing injuries.

REFERENCES

1. Keskin M, Tosun Z, Duymaz A, Savaci N. Frostbite injury due to improper usage of an ice pack. *Ann Plast Surg*. 2005;55(4):437–438.
2. O'Toole G, Rayatt S. Frostbite at the gym: a case report of an ice pack burn. *Br J Sports Med*. 1999;33(4):278–279.
3. Tobalem M, Modarressi A, Elias B, Harder Y, Pittet B. Frostbite complicating therapeutic surface cooling after heat stroke. *Intensive Care Med*. 2010;36(9):1614–1615.
4. Geng Q, Holmer I, Hartog DE, et al. Temperature limit values for touching cold surfaces with the fingertip. *Ann Occup Hyg*. 2006;50(8):851–862.
5. McAdams TR, Swenson DR, Miller RA. Frostbite: an orthopedic perspective. *Am J Orthop*. 1999;28(1):21–26.
6. Marx J, Hockberger R, Walls R. *Rosen's Emergency Medicine: Concepts and Clinical Practice*. 7th ed. Philadelphia, PA: Mosby/Elsevier; 2010.
7. Su CW, Lohman R, Gottlieb LJ. Frostbite of the upper extremity. *Hand Clin*. 2000;16(2):235–247.
8. McCauley RL, Hing DN, Robson MC, Heggors JP. Frostbite injuries: a rational approach based on the pathophysiology. *J Trauma*. 1983;23(2):143–147.
9. Golant A, Nord RM, Paksima N, Posner MA. Cold exposure injuries to the extremities. *J Am Acad Orthop Surg*. 2008;16(12):704–715.
10. Robson MC, Heggors JP. Evaluation of hand frostbite blister fluid as a clue to pathogenesis. *J Hand Surg Am*. 1981;6(1):43–47.
11. Campbell WC, Canale ST, Beaty JH. *Campbell's Operative Orthopaedics*. 11th ed. Philadelphia, PA: Mosby/Elsevier; 2008:4114–4117.
12. Herrera E, Sandoval MC, Camargo DM, Salvini TF. Motor and sensory nerve conduction are affected differently by ice pack, ice massage, and cold water immersion. *Phys Ther*. 2010;90(4):581–591.
13. Swenson C, Sward L, Karlsson J. Cryotherapy in sports medicine. *Scand J Med Sci Sports*. 1996;6(4):193–200.
14. Daanen HA. Finger cold-induced vasodilation: a review. *Eur J Appl Physiol*. 2003;89(5):411–426.
15. O'Brien C. Reproducibility of the cold-induced vasodilation response in the human finger. *J Appl Physiol*. 2005;98(4):1334–1340.
16. Bigelow DR, Ritchie GW. The effects of frostbite in childhood. *J Bone Joint Surg Br*. 1963;45(1):122–131.
17. Carrera GF, Kozin F, McCarty DJ. Arthritis after frostbite injury in children. *Arthritis Rheum*. 1979;22(10):1082–1087.

Address correspondence to Michael Rivlin, MD, Department of Orthopaedic Surgery, Thomas Jefferson University, 1015 Walnut Street, Room 801 Curtis, Philadelphia, PA 19107. Address e-mail to rivlin.md@gmail.com.