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An Unusual Case of Hand Frostbite Caused by Liquid Oxygen

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Frostbite injuries, often associated with exposure to extreme cold during outdoor activities, have been reported in the literature. However, few cases of frostbite caused by liquid oxygen have been documented. We report a 53-year-old man who was exposed to a liquid oxygen leak at work that caused severe frostbite injury to both hands. Early wound debridement and split-thickness skin graft for wound coverage were performed on day 8 after the frostbite injury. A second skin graft surgery was performed because of a failure of a patch of skin graft over the first web space of the left hand 20 days after the initial procedure. This unusual case of frostbite injury of the hands caused by liquid oxygen may help elucidate the characteristics of progressive deep-tissue injury that may lead to inadequate wound debridement and subsequent contracture scar formation over the web spaces of the hands despite early surgical treatment, which may impair hand function and lengthen hospitalization. Accurate clinical evaluation followed by early surgical wound management and the release of the web spaces of the hand with frostbite involvement may be beneficial in the treatment of deep hand frostbite injury.

Key words: Frostbite, Liquid oxygen

INTRODUCTION

Patients with frostbite, the most serious type of cold injury, present frequently with severe local deep-tissue freezing injury as well as multisystem injuries. The clinical manifestations of cold-induced injury are related to thermoregulation, cellular dysfunction, ischemia, and edema[1]. The mechanisms responsible for localized cold injury can be divided into phenomena that affect cells and extracellular fluids, and those that disrupt the function of the organized tissue and the integrity of the circulation[2]. In general, no serious local tissue damage is seen until freezing occurs. Local tissue may experience severe freezing injury by exposure of sites to extreme cold even though no systemic injuries are involved. For example, freezing injury of the corneas has been reported in individuals who keep their eyes open in high wind-chill situations without wearing protective goggles[3]. In theory, early diagnosis and adequate wound treatment of a frostbite injury with localized deep-skin tissue damage on some critical areas, such as the hands, should benefit from early wound coverage and should produce a better outcome in terms of function and esthetic restoration. However, the appearance of superficial tissue is often an unreliable indicator of deep-tissue viability in cases of frostbite. Therefore, the treatment of frostbite injury is still controversial and only a few principles are accepted universally.

Increased participation in outdoor activities and the epidemic of homelessness have increased the incidence of cold injuries in the civilian population over the last 20 years[4]. Rare cases also occur in warm areas such as Taiwan. Even though cold injuries might happen from exposure to a type of extreme cold in any number of circumstances, rarely do such incidents happen in a medical faculty. For example, oxygen, the most important air supply used for saving lives on a daily basis in a hospital, is stored usually as a liquid in a cylinder under extreme low temperature (-183°C or 90°K) and high pressure conditions, and should be free of danger. However, once vaporized, liquid oxygen increases in volume and takes the heat from the surrounding environment, and can cause frostbite injury even with

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brief exposure to the body. As an essential component of a hospital, the central oxygen supply system should be designed with features allowing backup or redundancy in the event of system failure. Under these circumstances, few reports of accidental frostbite caused by liquid oxygen have been presented. A case of frostbite injury was reported after the accidental fall of a portable liquid oxygen device in a medical faculty[5]. In a different scenario, we report an unusual case of localized hand frostbite caused by a liquid oxygen leak while exchanging the liquid oxygen containers in response to a failure of the air-supply system in a regional hospital. We discuss the rationale of wound care and surgical treatment.

CASE REPORT

A 53-year-old man, a member of the security staff in a regional hospital, sustained a severe frostbite injury to both hands caused by liquid oxygen during work. The injury happened as he rushed to exchange the liquid oxygen containers when the oxygen supply in the respiratory intensive care unit was found to be under low pressure in the early morning and could not be refilled automatically by the other reserve container because of a malfunction of the airflow switch. His injury was caused by direct contact with leaking liquid oxygen from the container because of incorrect instructions in the manual about keeping the air valve open. Exposure to liquid oxygen caused severe deep-tissue frostbite injury to both of his hands, which caused significant pain and swelling over both hands soon afterwards. The wounds were managed with warm water irrigation for about 40 min and application of a topical silver sulfadiazine cream followed by covering with sterile gauze dressings in the emergency room of the regional hospital. Tetanus toxoid and analgesics were also administered. With symptoms of intractable pain and swelling over his hands, he was transferred to our burn center for further management eight hours after the frostbite injury.

On admission to our hospital, the initial evaluation disclosed no systemic hypothermic injury except severe local cutaneous damage over the volar surface and the first web spaces of both hands, estimated as 2%-3% of total body surface area involved, combined with a deep laceration on the left hand. Blister formation and blue-gray skin discoloration over the injured areas were also noted (Fig. 1). The patient complained initially of local stiffness and numbness over the distal fingers and bulging swelling over the palmar area of both hands, and later experienced progressive throbbing, burning, and shooting painful sensations. Considering the possibility of compartment

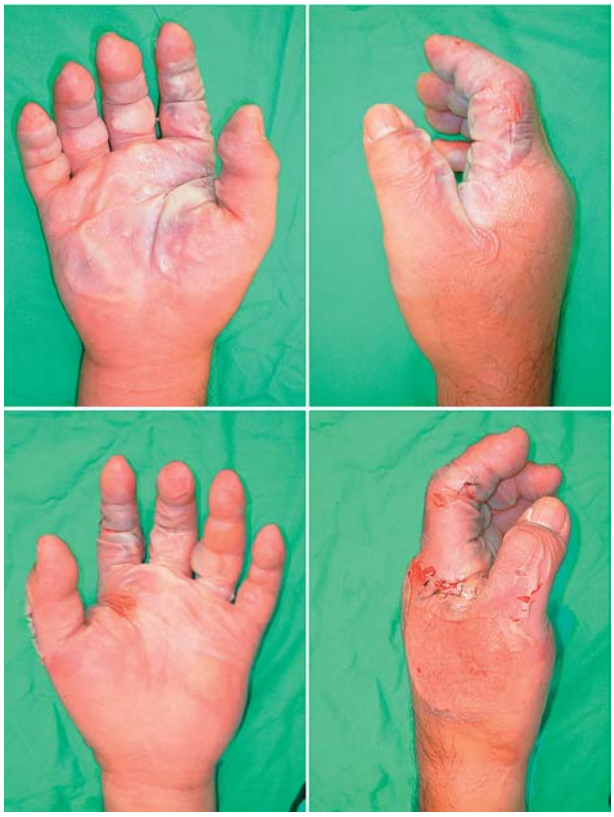

Fig. 1. Localized frostbite injuries were noted over the palms and the first web spaces of both hands presenting as generalized blue-gray discoloration of skin and blister formation with 2% to 3% of total body surface area involved.

syndrome, emergency surgical intervention was launched that included superficial wound debridement, primary closure of lacerations, and fasciotomy on both dorsal hands. Deep second- to third-degree skin tissue injury was obvious in the initial inspection, and porcine skin was used for temporary wound coverage in the early stage.

After adequate wound care and observation for eight days, we took a surgical approach with further debridement and autologous split-thickness skin graft because we noted profound tissue damage involving the full thickness of the skin and the underlying fascial layer over the first web space of both hands. However, a patch of grafting loss and eschar formation were found later over the first web space of the left hand. The residual wound defect was then covered with a split-thickness skin graft, and the contracture scar over the first web space of the left hand was released one month after the initial frostbite injury. The skin take was good after three days with the retained dressing (Fig. 2). The patient was treated with anticontracture

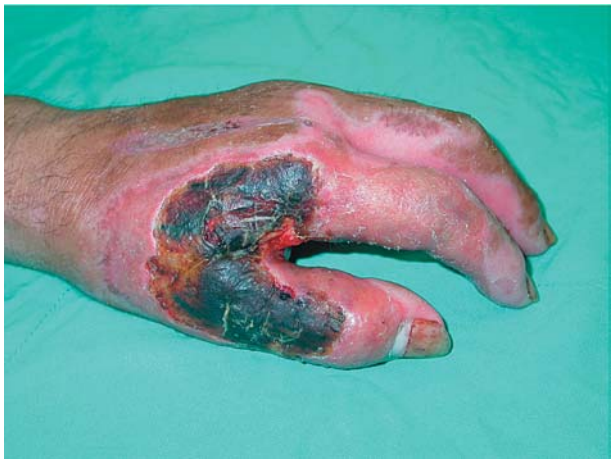

Fig. 2 A patch of grafting loss with eschar formation was found over the first web space of left hand after the prior skin graft surgery.

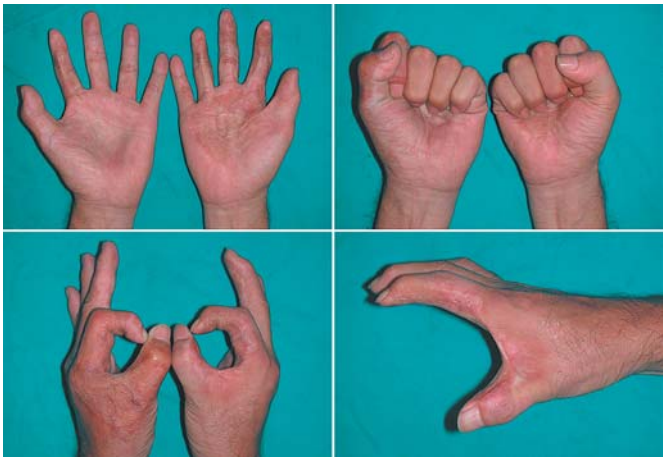

Fig. 3. Complete wound healing with moderate contracture scar formation over the first web space of right hand was noted at 9 months follow-up.

prosthetic splinting and active rehabilitation therapy during the following recovery stage. On the 39th day after the frostbite injury, he was discharged with complete wound coverage. The hand functions of grip and grasp were restored completely except for moderate contracture scar formation over the first web space of the right hand at the nine-month follow-up examination (Fig. 3).

DISCUSSION

Frostbite injury caused by liquid oxygen has been reported rarely. Our patient experienced frostbite injury caused by direct contact with a large leak of liquid oxygen from a cylinder in a hospital because of incorrect instructions in the manual about keeping the air valve open. The exposure caused severe deep-tissue injuries to both hands. Oxygen is usually stored in a cylinder container as a liquefied gas under very low temperature (-183.17^{o}C or 90 ^{o}K) and high pressure, and is an important and usually harmless medical air supply in the hospital. Liquid oxygen itself is a pale blue, extremely cold, and cryogenic liquid, whose normal boiling point is below -183.17^{o}C. Once vaporized, liquid oxygen increases in volume and takes heat from the surrounding environment, which can cause frostbite injury to the local tissue even with a short exposure[6].

Direct cold injury of skin can be caused by direct cellular damage or by indirect cellular effects resulting from microvascular changes, such as vasoconstriction, microvascular occlusion, and increased blood viscosity, which may lead to thrombosis and ischemia. Intracellular and extracellular ice crystal formation, cell dehydration, abnormal intracellular electrolyte concentration, thermal shock, and denaturation of lipid–protein complexes are some of the cellular changes described[7-11,25]. Frostbite, the most severe type of direct cold injury, can be divided into three distinct states. The prefreeze state is characterized by increased viscosity, tissue cooling, and the capillary constriction–dilatation cycle. The frozen state is characterized by intracellular dehydration and hyperosmolarity, intracellular or extracellular ice crystal formation, and fluid shifts across the cell membrane. The ischemic and vascular complications state is characterized by endothelial leakage, reperfusion injury, leakage of destructive prostaglandins and oxygen free radicals, coagulation resulting from vascular stasis, vasoconstriction and arteriovenous shunting, necrosis demarcation, and gangrene[7-11].

Acute compartment syndrome occurs because of elevated pressure within a confined musculofascial space. The increased pressure compromises the muscle and neurovascular structures residing within this space, and capillary perfusion is compromised and reduced below the threshold of pressure necessary for tissue viability. Acute compartment syndrome occurs frequently after vascular insults, including ischemia–reperfusion injury. Frostbite can cause significant vascular injury, including diminution of blood flow to the affected extremity. As noted by Velmahos and Toutouzas, ischemia can lead to significant edema, which can lead to further ischemia, a self-perpetuating cycle that can lead to compartment syndrome. Reperfusion of the extremity produces toxic oxygen free radicals, which can increase tissue damage and edema, further contributing to the development of compartment

syndrome[12-15,32,33]. The signs of compartment syndrome are defined clinically as the 5Ps–pain, pallor, pulselessness, paralysis, and paresthesia–which may occur in frostbite injury of a limb and may lead to tissue ischemia and necrosis. The local area injured by frostbite may be anesthetic, pulseless, pale, mottled, severely swollen, and immobile in the early stage; and the combined symptoms or signs of pain, pallor, pulselessness, paralysis, and paresthesia may appear later. The normal resting intramuscular pressure is estimated as 0-8 mmHg, and the symptoms of pain and paresthesia may first appear at pressures between 20 and 30 mmHg. In cases where the pressure is >30 mmHg, fasciotomy should be undertaken to release the intracompartmental pressure (ICP)[9,12-15]. However, continuous clinical assessment of ICP is still the first choice to detect progressive compartment syndrome, and assessment must be repeated at frequent intervals (30 min to 2 h) to allow early surgical intervention if needed and avoiding the possibility of distal limb necrosis. Even though we did not monitor ICP during early resuscitation in this patient, we performed the fasciotomy because of the symptoms and signs of progressive pallor, intractable pain, severe swelling, and cold with numbness over the distal fingers of both hands. The symptoms and signs were relieved significantly after management.

The policy for treatment of frostbite injury is controversial and only a few principles appear to be universally accepted. Treatment is usually divided into three phases: (1) prethawing, (2) rewarming, and (3) postthawing, which may continue for several weeks or months. Before evaluation by a qualified burn specialist, care of the patient should focus on protecting the involved part from mechanical trauma and avoiding thawing until definitive rewarming can be performed, because cyclic thawing and cooling or inadequate rewarming can worsen the cellular injury[7,25]. Systemic hypothermia may be resolved by resuscitation with warm intravenous fluids in an attempt to elevate the core temperature. Insulation with warm blankets or other clothing or materials should be used to prevent further cooling, but no other treatment should be initiated during transport. In general, the patient should be transferred to a specialist unit as soon as possible, and the affected areas must be kept in a warm-water bath (40-42 ^{o}C) for 10-30 min until the distal extremity becomes pliable and erythematous. The general recommendations for wound management are to debride clean blisters, leave hemorrhagic blisters intact, dress the injured area and blisters with aloe vera cream, elevate the affected parts with splinting as indicated, and begin daily hydrotherapy[7-10,15-21]. Early diagnosis may help in cases of adequate wound treatment and provide a better outcome. However, Satybaldyev et al reported that early diagnosis of the depth of extremity frostbite has little effect without further examination (e.g., 39.9%-43.6% of correct findings at the pre- and intrahospital stages of medical care), and the best timing of decision making for definite surgical management is still controversial[22]. A recent support suggests that a policy of active surgical treatment has significant advantages over an expectant surgical policy, as evident from the shorter treatment time and number of postoperative pyonecrotic complications (21.7% vs. 78.4%)[23].

Although frostbite is treated traditionally by awaiting tissue demarcation before surgical intervention, early debridement of the wound prevents infection and stimulates reepithelialization of the skin[7,8]. Removal of all clean and hemorrhagic blisters caused by burns or cold injury allows for the early and accurate diagnosis of superficial or deep tissue damage, and therefore makes early surgical wound treatment possible. In this patient, the timing for definite debridement and autologous split-thickness skin graft was decided early because of the initial findings of deep-tissue necrosis over both hands, including the first web spaces, on day 8 after injury. However, a piece of skin graft loss was still found over the first web space of the left hand, which may have resulted from progressive tissue necrotic change or inadequate wound debridement. Final wound coverage was achieved by split-thickness skin graft again at the 28th day after injury. In contrast, despite achieving early successful wound coverage of the right hand, the progressive formation of the contracture scar over the first web space reflected progressive ischemic changes of the underlying deep tissue layer leading to fibroplasia. In addition, the observation of less contracture scar formation over the first web space of the left hand may have contributed to the successful surgical outcome, adequate postoperative splinting, rehabilitation, and possibly the procedure of early web space release during the second skin graft operation.

A protocol of early surgical intervention for frostbite based on reliable radiographic studies appears prudent. Technium-99 (Tc-99) bone scanning has become the standard imaging study used to assess tissue perfusion and viability within the first several days of tissue damage and should be carried out after medical treatment to help the surgeon define salvageable tissue before surgical intervention[2,7,25-29]. Angiography may be used to define the limits of vascular injury, but it is invasive and expensive and it dose not allow imaging of vessels at the arteriolar or capillary levels. Thrombosis of large-caliber vessels is rare following frostbite injury. Doppler ultrasonography and

digital plethysmography may be used to assess local blood flow after frostbite, but neither is able to assess the microcirculation or capillary function[7,25]. Magnetic resonance imaging (MRI) or magnetic resonance angiography (MRA) appear to be superior to Tc-99 bone scanning by allowing direct visualization of occluded vessels and imaging of surrounding tissues, and by showing a more clear cut line of demarcation of ischemic tissue. MRI or MRA may also allow early intervention in cases of severe frostbite, thus preventing secondary infection and increased cost[2,25,26].

Hyperbaric oxygen (HBO) is useful in limiting tissue damage and enhancing the healing process after frostbite by counteracting tissue hypoxia and reducing tissue edema. HBO improves the chances of survival through one or more of the following mechanisms: relief of tissue hypoxia or ischemia, diminished metabolic disturbances in the ischemic–hypoxic tissues, improved microcirculation and reduced platelet aggregation, increased number and size of blood vessels within the microvasculature, counteracting free radical-mediated reperfusion injury, and accelerating the formation of healthy granulation tissue over bone. HBO also promotes wound healing, prevents infection, and eventually helps to demarcate the necrotic area from the viable area so that a greater part of the involved tissue can be salvaged and restored to function. HBO is indicated when there is evidence of ischemia that is refractory to other measures and where surgery is considered to cover soft tissues defects. Good results have been reported in using HBO to treat problem wounds and frostbite and might be effective for treating late changes in the growing bones. This conservative treatment helps to reduce hospital stay, allows early return to work, and protects from late skeletal changes. Because of its low risk and its potential therapeutic efficiency, HBO should be recommended as adjunct therapy in the treatment of deep frostbite[18,19,30,31,34].

The late sequelae of frostbite injury include hyperhidrosis, pain, numbness, color changes in the integument, vasospastic attacks, intrinsic muscle atrophy, tissue loss, and joint stiffness, which occur in relation to the degree of initial cold injury and the success of treatment[10,24]. Our patient complained less of these symptoms and returned to work earlier than would be expected, and he had complete restoration of hand function at the nine-month follow up.

In summary, the unusual case of hand frostbite injury reported here helps highlight the importance of correct early evaluation of deep tissue injury, the benefits of an aggressive surgical approach, including early web space release once involved, and the importance of safety programs for medical staff working in hospitals.

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