Lightning injury: A review and case presentations

Jerome Edelstein MD, Walter Peters PhD MD FRCSC, Robert Cartotto MD FRCSC
Ross Tilley Burn Centre, Wellesley Hospital, Division of Plastic Surgery, University of Toronto, Toronto, Ontario

Lightning injuries are relatively uncommon, although thousands are affected each year. Many patients are left with permanent sequelae. This type of injury is very different from other high voltage electrical injuries. Lightning burns are usually superficial and only about one quarter of those struck by lightning will actually die. The number of deaths can be reduced if proper early resuscitative methods are used. Two lightning-strike victims are presented. The first patient suffered transient neurological abnormalities, eye and ear injuries including permanent sensorineural hearing loss, and superficial burns over 70% of his body surface area. The second patient, who wore bilateral arm prostheses, was struck by lightning while driving his motor boat. The lightning welded one of his prostheses to the steering wheel of the boat. He sustained a 10% partial thickness burn, but had no permanent sequelae. A review of the current literature is presented, including the pathophysiology, multisystem clinical features and treatment of lightning injury.

Key Words: Electrical injury, Lightning burn

Foudroiement : Revue et présentation de cas

RÉSUMÉ : Les lésions causées par la foudre sont relativement rares. Bien qu’accompagnées d’un taux de mortalité de 25 % seulement, elles laissent des séquelles permanentes chez de nombreux patients et sont très différentes des lésions produites par une électrocution de haut voltage. Les lésions provoquées par la foudre sont habituellement superficielles et environ le quart seulement des victimes de la foudre en mortront. Le nombre de décès peut être réduit si des méthodes de réanimation adéquates sont promptement instituées. Deux cas de foudroiement sont présentés ici. Le premier patient a présenté des anomalies neurologiques transitoires, des lésions à l’œil et à l’oreille, y compris une perte sensorielle permanente de l’ouie, et des brûlures superficielles sur plus de 70 % du corps. Le deuxième patient, porteur de deux bras artificiels a été frappé par la foudre alors qu’il manœuvrait son hors-bord. La foudre a touché l’un des ses prothèses au volant de l’embarcation. Il a subi un épaississement partiel de 10 % par brûlure, ce qui ne lui a par ailleurs laissé aucune autre séquelle. La littérature récente est passée en revue, y compris la physiopathologie, les caractéristiques cliniques plurisystémiques et le traitement des blessures infligées par la foudre.

Each year in the United States, about 1800 people are struck by lightning. About 450 of these patients die (mortality rate approximately 25%). These figures are small, when one considers that lightning strikes the earth 100 times per second (eight million times per day). A tall free standing structure such as the Empire State building in New York is struck thousands of times every year (1).

Lightning injuries usually occur during the summer months when people are outdoors, between the hours of noon and 6 pm. Common activities include waiting under a tree, camping, jogging, water sports, golfing, working around farm or construction equipment, and using the telephone. In a third of cases, there are two or more victims (2).

PHYSICS
Lightning involves a transfer of electrical charge, developing when warm, low pressure air rises through colder, higher pressure air. Complex interactions occur to create an electrical potential between the negatively charged inferior aspect of a cloud and the positively charged ground. When the voltage difference exceeds atmospheric resistance (approximately 30,000 volts), lightning results (3).

The initial discharge from the cloud is called the stepped leader. It is negatively charged, moves downwards in discrete steps, and is not visible. When the stepped leader approaches within 100 m of the ground, a stronger, positively charged return stroke moves upwards to meet it. With contact, visible lightning occurs (4).

The downward stroke is slower so that the observer perceives the strike to be from the cloud to the ground. Thunder is heard as the explosive heating and expansion of air produces a shock wave (4).
TABLE 1: Mechanisms of lightning injury

| Direct injury | Contact | Splash | Ground current | Blunt injury |

TABLE 2: Differences between standard electrical and lightning injuries

<table>
<thead>
<tr>
<th>Standard electricity</th>
<th>Lightning</th>
</tr>
</thead>
<tbody>
<tr>
<td>Current type</td>
<td>Direct current</td>
</tr>
<tr>
<td>Voltage</td>
<td>Up to 1 billion volts</td>
</tr>
<tr>
<td>Duration</td>
<td>0.0001 - 0.001 s</td>
</tr>
<tr>
<td>Tissue pathway</td>
<td>Skin ‘flashover’</td>
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</tbody>
</table>

PATHOPHYSIOLOGY

Lightning injures people in five possible ways (Table 1). Determining the type of strike in a given case is often impossible. A direct strike, as the name implies, occurs when lightning hits the victim directly. This is the most dangerous of all injury mechanisms. Contact occurs when lightning strikes an object the victim is in contact with (e.g., metallic jewelry, golf club). Even an object as small as a hairpin has been shown to increase significantly the risk of being struck by lightning.

The most common mechanism for lightning injury is splash. In this situation, lightning strikes an object first and then jumps to a nearby person of lower resistance. With ground current, the bolt strikes the ground, radiating outward to the victim. Injury is then inversely proportional to the distance from the strike. Blunt injury results from the shock wave (e.g., acoustic trauma, contusion of internal organs) or from being thrown. The violent propulsion of some lightning victims is secondary to tetanic muscular contraction or the acquisition of charge. When struck by lightning, the victim becomes highly charged and may be strongly attracted to or repelled by an adjacent object (5,6).

Standard electrical injuries are different from those due to lightning (Table 2).

The alternating current of technical electricity fixes a victim to the current source by tetanic muscle spasm. The longer contact causes skin to break down and injury to deeper tissues even though voltage is lower. In contrast, lightning contact is instantaneous with less time to cause injury. Skin ‘flashover’ occurs with the majority of electrical energy flowing over the victim’s body rather than through it, similar to electrical current flowing along the outside of a metal conductor.

The cardiorespiratory system is more sensitive to alternating current, which acts as a fibrillatory stimulus. This induces ventricular fibrillation whereas the direct current of lightning injury depolarizes the myocardium only once causing temporary asystole, similar to a defibrillatory countershock. The

asystole is only temporary as the heart’s inherent automaticity eventually causes conversion to normal sinus rhythm (5).

CASE PRESENTATIONS

Case 1

A 20-year-old male was camping with his girlfriend at a national park. During an early morning walk he was struck by lightning, the contact point being a metallic chain he wore around his neck (Figure 1). His girlfriend was also electrified as they were holding hands at the time of the incident. At the scene, the patient was found to be comatose. A cervical collar was applied and he was transferred onto a spine board. Intravenous access was obtained and tetanus toxoid was administered. He was evacuated to hospital by military transport.

Upon arrival to hospital, his vital signs were normal. He was alert and oriented, his level of consciousness having improved over a 2 h period. There was, however, lower limb paraparesis evident on neurological examination and he was amnesic for the event. He complained of decreased vision in his left eye, vertigo, a total inability to hear, and a painful cervical spine. Physical examination of the left eye revealed...
a subconjunctival hemorrhage (Figure 1), corneal abrasion, miosis, and decreased visual acuity to finger counting only. The right tympanic membrane was perforated, and blood was evident in the canal. His cervical spine was tender over C6.

Second degree burns were present around the neck and unevenly distributed over the right arm and leg. There were confluent first degree burns over most of his body surface and extensive scorching of body hair. There was no evidence of any fractures or motting of the extremities.

A nasogastric tube was inserted. Excellent urinary output was obtained following catheter insertion, with no evidence of discoloration. Testing for myoglobinuria was negative. Electrocardiogram (ECG) and chest radiographs were normal.

During the patient’s first two days in hospital, his lower limb weakness resolved spontaneously. C-spine radiographs were normal and the spine was cleared after neurosurgical consultation. Ophthalmic injuries responded to 24 h treatment with antibiotic ointment and eye patching. Otologic consultation documented profound, bilateral conductive and sensorineural hearing loss. His vertigo resolved spontaneously. Standard wound therapy was applied, including polysporin to the face and jelonet/saline gauze dressings to the right upper extremity.

The patient was discharged home on day 5. Dressings were continued at home by a visiting nurse. At follow-up two weeks later, his wounds were healed.

Unfortunately, severe sensorineural hearing loss persisted and arrangements had to be made for a hearing aid. He also developed recurrent episodes of iritis involving his left eye. In addition, he developed numbness of his lower limbs which was diagnosed as a cervical myelopathy.

Case 2
A 34-year-old man was driving a power boat during a rainstorm. Following a severe farming accident as a teenager, he lost both arms and therefore wore a bilateral arm prosthesis (Figure 2). Lightning struck the steering wheel of the boat, welding it to the hook portion of his prosthesis.

On admission to hospital, the patient was alert but amnestic for the event. He had a deep partial thickness burn to his neck, at the site of the metallic buckle of his arm prosthesis unit (Figure 3). He also had scattered areas of second degree burns on his trunk. A complaint of decreased hearing in the left ear revealed a small perforation of the tympanic membrane.

The patient was treated in hospital with topical antibiotic therapy (silver sulphadiazine) and regular dressings (saline gauze) for three days. Dressings were continued at home by a visiting nurse, and his wounds healed spontaneously within three weeks. At follow-up one month after the event, there was no evidence of tympanic membrane perforation and hearing was normal.

CLINICAL FEATURES
Although only about 25% of lightning victims die, as many as 75% of survivors have permanent sequelae (Table 3). The clinical spectrum of potential involvement is quite diverse. Some patients present with multisystem involvement, while others show little evidence of injury (Table 4). Poor prognostic indicators include cardiorespiratory arrest (76% mortality), cranial burns (37%) and leg burns (30%) (7).
TABLE 3: Long term sequelae of lightning injury

<table>
<thead>
<tr>
<th>Condition</th>
<th>Outcome</th>
</tr>
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<tbody>
<tr>
<td>Hearing loss</td>
<td>Burn scars</td>
</tr>
<tr>
<td>Cataracts</td>
<td>Paresis</td>
</tr>
<tr>
<td>Psychiatric illness</td>
<td>Neuritis with painful neuralgia</td>
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TABLE 4: Types of lightning injury

<table>
<thead>
<tr>
<th>System</th>
<th>Injury</th>
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<tbody>
<tr>
<td>Central nervous system</td>
<td>Change in level of consciousness, amnesia, seizures, paresis, neuritis, subdural hematoma</td>
</tr>
<tr>
<td>Cardiac</td>
<td>Arrhythmias, ECG changes, myocardial infarction</td>
</tr>
<tr>
<td>Skin</td>
<td>Partial to full thickness burns; feathering, linear, punctate, thermal</td>
</tr>
<tr>
<td>Ophthalmic</td>
<td>Corneal damage, hyphema, uveitis, retinal hemorrhage or detachment, optic neuritis and atrophy, cataracts</td>
</tr>
<tr>
<td>Otologic</td>
<td>Tympanic membrane rupture, ossicular disruption, vertigo, middle ear or mastoid hematoma</td>
</tr>
<tr>
<td>Psychiatric</td>
<td>Sleep disturbance, fear of storms, anxiety, depression</td>
</tr>
<tr>
<td>Pulmonary</td>
<td>Contusion, edema, hemopneumothorax</td>
</tr>
<tr>
<td>Renal</td>
<td>Acute tubular necrosis (hypoperfusion or myoglobinuria)</td>
</tr>
<tr>
<td>Gastrointestinal</td>
<td>Gastric atony, ileus</td>
</tr>
<tr>
<td>Musculoskeletal</td>
<td>Fractures</td>
</tr>
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</table>

Nervous system

The nervous system is quite sensitive to lightning injury. More than half of those struck by lightning display some form of neurological involvement. In the majority of cases this involvement is transient, lasting less than 24 h. Injury is often ascribed to 'short circuiting' of the electrical system, but concussive injury sustained in the initial fall or hypoxia secondary to cardiac arrest or vascular injury may also be involved.

A change in level of consciousness ranging from disorientation to frank coma is often noted and retrograde amnesia is almost universal (similar to electroconvulsive therapy). When the patient's level of consciousness improves, transient weakness most often involving the lower extremities may be present. Seizures may also occur. Less than 5% of patients suffer from late sequelae such as hemiplegia or painful neuritis (7,8).

Cardiac system

It is estimated that one third of lightning victims suffer primary cardiac arrest. The massive DC countershock of lightning stroke results in temporary asystole, which converts spontaneously to normal sinus rhythm due to the heart's inherent automaticity (7).

Secondary cardiac arrest is the critical mortality factor. Seventy-five percent of those who die as a result of electrification by lightning are thought to have suffered secondary arrest. Demonstrated experimentally, the arrest is caused by primary respiratory centre depression with resultant hypoxemia leading to ventricular fibrillation or asystole (9). The asystole in this case is much more resistant to spontaneous conversion (10).

Other cardiovascular complications include transient hypertension, tachycardia, and a variety of arrhythmias (eg, atrial fibrillation). Nonspecific ECG changes are often present, ST elevation and T wave inversion being the most common. These changes usually resolve within 24 h and only require close monitoring (11). Myocardial infarction is uncommon with lightning injury. Serum creatine kinase-MB can give a false positive result because of the electrical interaction with skeletal muscle. Serum lactate dehydrogenase (LDH) is a better indicator of myocardial damage in this situation (12).

Skin

Lightning burns are usually superficial (partial thickness). Less than 5% of patients suffer full thickness burns. The superficial nature of lightning burns is due to the brief flashover effect of the electrical energy. Deeper burns occur under metallic objects (jewellery), with ignition of clothing, or at entrance/exit points (rare).

There are four patterns of lightning burns. Feathering is highly characteristic of lightning. It is a nonblanching, reddish-brown fern pattern which disappears in several days. If seen in an unaccompanied, comatose patient, lightning injury may be presumed. Linear burns tend to occur in areas where sweat accumulates (eg, axilla) and appear to be due to steam production by the electrical flashover. Punctate lesions are small, cigarette-like burns that occur in clusters, while thermal burns are caused by ignition of clothing or heating of metallic objects (4,13).

Vascular

Intense vasomotor instability, possibly due to autonomic dysfunction, has been repeatedly documented in these patients. Arterial spasm leads to cold, mottled, pulseless extremities. This is usually a temporary finding, resolving spontaneously within hours. The physician must keep this in mind whenever considering the need for fasciotomy in these patients (rare) (14,15).

Ophthalmic

More than half of all lightning victims suffer some form of ophthalmic injury, most commonly involving the cornea. Damage is the result of a combination of electrical energy and intense brightness. Cataracts are the single most common long term sequelae of lightning injury, occurring in approximately 5% of patients. They can occur months or even years after the initial insult. Therapy is delayed since a percentage resolve spontaneously over time. Other findings include uveitis, hyphema, retinal hemorrhage or detachment, and optic neuritis leading to atrophy (16).

It is extremely important to remember that autonomic dysfunction is common after lightning strikes, possibly resulting in mydriasis, loss of pupillary reflex, anisocoria, and
Homer's syndrome. As such, fixed dilated pupils do not imply brainstem injury and should not lead to discontinuation of cardiopulmonary resuscitation (CPR) (17).

Otologic
Tymppanic membrane rupture is the most common otologic abnormality, occurring in more than half of patients secondary to barotrauma. Oscillatory disruption, vertigo and sensorineural hearing loss can also be evident. Treatment of these injuries is reserved for debilitating vertigo, oscillatory disruption or nonhealing (longer than six to 12 months) tympanic membrane rupture (18,19).

TREATMENT
The immediate (pre-hospital) resuscitation is the most important factor with respect to outcome. At the scene of a lightning strike, medical attention should be directed towards those 'apparently dead' as the living will almost always survive. This is in contrast to the normal triage of a disaster scene where attention is focused initially on the living. CPR seems to have a higher success rate in this situation even after prolonged elapsed time (20). The popular explanation is that metabolism ceases instantly following lightning contact, delaying tissue degeneration. Although this has not been proven, Rovitch in 1961 (21) reported the case of an eight-year-old boy who was struck by lightning. Although the patient had no vital signs for approximately 13 mins, his pupils were fixed and dilated, and he did not bleed when a thoracotomy was performed; he did respond to cardiac massage and intracardiac adrenaline injection and went on to survive with no apparent sequelae. Hospitalization is required for those displaying poor prognostic indicators (cardiorespiratory arrest, cranial or leg burns), a history of loss of consciousness, residual neurologic deficits, or changes on ECG.

Upon arrival to hospital, initial therapy involves the ABCs of trauma care including C-spine immobilization and prolonged CPR if necessary. Tetanus prophylaxis must be ensured. An ECG should be obtained. Cardiac monitoring and serum LDH are indicated if there are any ECG changes or a history of loss of consciousness. Arrhythmias should be treated in standard fashion. Patients usually do not require vigorous fluid resuscitation due to the superficial nature of their injuries. In fact, fluid replacement should be tempered by the possibility of cerebral edema.

Depressed level of consciousness should be treated in standard fashion. If residual neurologic deficits are present, a computerized tomographic (CT) scan of the head is useful to rule out hematoma. Cerebral edema and seizures are treated as in other settings. Ophthalmic and otologic consultations are recommended. A nasogastric tube should be inserted if ileus is suspected. Urinalysis is indicated to detect myoglobinuria. Because burns seldom involve deep tissues, myoglobinuria is rare, as is the need for hydration and diuresis, or for fasciotomies to correct compartment syndrome. The pulseless, mottled extremity is best approached with patience, allowing the vascular spasm to resolve spontaneously. In the majority of cases, the overall management of lightning victims is conservative and supportive, awaiting spontaneous resolution of their injuries.

REFERENCES