Neuronal desertification after a direct lightning strike: a case report

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Case Report

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Abstract

Background:

Lightning strike is a rare but dramatic cause of injury to patients admitted to Intensive Care units (ICU). High mortality and significant long-term morbidity are frequently related to a direct brain injury or an induced cardiac arrest (CA).

Case presentation:

We describe the case of a 50 years old man struck by lightning resulting in immediate CA. After the initial restoration of spontaneous circulation and admission to ICU, the patient died in hospital due to neurological injury. The computer tomography scan (CT scan) revealed a massive loss of grey-white matter differentiation at the fronto-temporal lobes bilaterally. Somatosensory evoked potentials demonstrated bilateral absence of the cortical somatosensory N20-potential and the electroencephalogram (EEG) recorded minimal cerebral electrical activity. The patient died on day 10 and a post-mortem study revealed a widespread loss of neurons.

Conclusion:

This study report a severe brain injury by a direct lighting strike presenting with an impressive microscopic pattern of neuronal desertification.

Background

Lightning occurs nearly 50 times per second worldwide[1] and, although injuries secondary to lightning strike are rare, they carry significant long-term morbidity and high mortality. Many of these fatalities result in immediate death due to fatal arrhythmias or respiratory failure. Lightning injuries determine critical alterations in the cardiovascular, respiratory, and central nervous systems.[2] Resuscitation attempts may have higher success in lightning casualties compared with other etiologies of cardiac arrest, leading to a return to spontaneous circulation (ROSC) and in rare cases good functional outcome. We report the case of a middle-aged man struck by lightning, followed by a review of the literature on the pathophysiology of lightning-induced injury. In addition to previous reports, we describe a pattern of diffuse cerebral neuronal desertification revealed at the post-mortem study and directly related to the injury.

Case Presentation

During a summer rainstorm, a 50-years-old man riding a bicycle in the forest surrounding the city of Trieste, Italy, was struck by lightning resulting in immediate cardiac arrest as reported by some bystanders. Basic life support was not initiated by the lay bystanders, but only a few minutes later by a physician hiking nearby, and thereafter continued by the medical emergency system personnel, arrived at the scene 15 min after the event. Rhythm of presentation was asystole, downtime before ROSC was 16

minutes. After ROSC, the patient remained unconscious with a Glasgow Coma Score (GCS) of 3, non-reactive mydriatic pupils, stable blood pressure and oxygenation values once intubated. While performing the secondary survey at hospital admission, was noted a cutaneous lesion in the occipital area described as singed hair with an important burning smell, linear in shape, with a small round black eschar, presumably compatible with the first contact with lightning. (Figure 1)

FIGURE 1

The presence of burnt plastic at the level of the occipital slot of the helmet was consistent with such hypothesis. Similar erythematous cutaneous lesions with singed hair were discovered at the suprapubic level, probably the output area in proximity to the metal core of the bicycle frame through the saddle. (Figure 2)

FIGURE 2

In addition, the right tympanic membrane was perforated. Ophthalmological assessment was negative. Total body CT scan showed a modest right apical and temporal subarachnoid hemorrhage, millimetric petechial hemorrhages in the cortical-subcortical junction of the fronto-parietal lobes and modest signs of initial edema (Figure 3); an abundant hemoperitoneum associated with a bleeding hepatic angioma, was present.

FIGURE 3

The patient was subjected to damage control surgery with abdominal packaging to control the bleeding and then admitted to our ICU. Blood samples depicted an elevation of biomarkers of myocardial and hepatic injury (high-sensitivity troponin I 90879 ng/L, creatine kinase 1131 U/L; aspartate and alanine transaminase 361 and 421 (U/L respectively), and blood lactate (11.36 mmol/L). As the EKG showed sinus tachycardia with minimal nonspecific ST elevation in inferior leads and the cardiac ultrasound showed normokinetic ventricles, no further cardiological investigations were undertaken at that time. Owing to the prolonged time to admission, target controlled temperature management was not established, and the subsequent appearance of fever (from day 2 after ICU admission, mean central body temperature of 38.2°C with only one peak at 38.8°C) was managed with ice packs, a short period of diclofenac infusion and then acetaminophen on demand. No prophylactic therapy against vasospasm or brain edema was instituted, since serum hyperosmolality was persistently elevated (319 mOsm/L). Following surgery, the patient remained in cardiogenic shock with profound haemodynamic instability and lactic metabolic acidosis. Increasingly high norepinephrine doses were needed to support arterial pressure, peaking 0.95 mcg/kg/min on day 2. On day three, after adequate goal-direct volume expansion, terlipressin infusion was added (peaking 1.28 mcg/kg/hr). Cardiac function restored to normal, as demonstrated by a further ultrasound assessment. Acute kidney injury due to rhabdomyolysis (depicted at the ICU admission by the elevation in serum creatinine, blood urea nitrogen, serum creatine kinase (1131 U) and oliguria were managed with volume expansion with crystalloids, urine alkalinization with sodium bicarbonate boluses, and diuretics. A brain CT scan was repeated on day 2 revealing a further

reduction of the cortico-subcortical differentiation bilaterally in the frontal-parietal and temporo-occipital areas compatible with anoxic edema, a marked flattening of the cortical convolutions, and the obliteration of the supratentorial peri-encephalic liquor spaces and at the level of basal cisterns. On day 3 sedation was interrupted to allow for neuroprognostication. A neurological clinical assessment evidenced a persisting GCS 3 and the absence of pupillary and corneal reflexes. Somatosensory evoked potentials demonstrated bilateral absence of the cortical somatosensory N20-potential (ERB- and N13-potential were present) and the EEG recorded minimal cerebral electrical activity with very low voltages. On day 4, haemodynamic stability was obtained, allowing for tapering of vasopressor support and fever ceased. On day 5, the patient remained unconscious with a GCS 3 and only weak cough reflex and respiratory drive. A new EEG revealed an isoelectric trace. A cerebral CT angiography revealed a regular opacification of the arterial circulation with normal venous drainage. Direct scanning highlighted further reduced cortical-subcortical differentiation and marked flattening of brain convolution. (Figure 4)

FIGURE 4

The patient died on day 10. A post-mortem evaluation revealed signs of multi-organ hypoperfusion, subendocardial and transmural cardiomyocytes suffering. Cerebral sections confirmed CT findings of petechiae, and subarachnoid hemorrhage previously located along the electrical current and as a result of direct damage to brain vasculature. Brain tissue softening and edema was associated with the direct effects of electrical passage. The cerebral parenchyma sections revealed an abundant, non-selective loss of neurons. (Figure 5)

FIGURE 5

Discussion And Conclusion

Lightning injuries are responsible for an average of 300 injuries and 100 deaths per year in the United States [3][4][5]. It is likely that lightning-related fatalities and hospitalizations are underestimated [6]. Approximately 30% of those struck by lightning die and up to 74% of survivors may have permanent disabilities [7][8][4] Two-thirds of lightning-associated deaths occur within one hour of injury and are generally due to a fatal arrhythmia or respiratory failure [9][10] Asystole or ventricular fibrillation represent the underlying mechanism [11, 12]. Whereas direct strikes more probably result in asystole due to sudden depolarization of the heart[2][11], ventricular fibrillation or tachycardia could be more frequent [13] but they are more likely associated with indirect strikes[11]. This case report describes the dramatic neurological consequences of a patient surviving to a direct strike by a lightning. Of note, we present the pathology findings determined over time by the effects of electrical current through the brain, namely massive neuronal loss leading to cerebral neuronal desertification. In the brain parenchymal sections explored, pathologists reported a non-selective depletion of neuronal cells.

These alterations are common findings in Alzheimer disease which occurs over years due to the progressive formation, diffusion and thickening of diffuse and senile plaques. Spongiosis is the result of the death of the neuron, which leaves on the brain section an empty halo. Gliosis represents the reactive

proliferation of the common glia in deneuronization situations. Perivascular halo are observed, resulting from the retraction of all the brain substance and the neuropil and from gas bubbles also to electrolytic processes. What usually happens in months in severe cerebral ischemia and in years in dementia has been condensed into a few days and can be explained only by an external event that exerted a dramatic influence on cell tropism, and consequently, cell death. The combination of both cardiac arrest and direct electrical damage might have thus resulted in a quickly complete cerebral crushing in this specific case.

In our patient, beside the damage due to the global cerebral ischemia developed during the no flow and low flow phases after the cardiac arrest, a direct lightning strike injury might have played a significant role. A sudden extremely high warming-up of the brain due to the lightning route through the head have indubitably contributed to an immediate neuronal death and a subsequent increased injury in the re-perfused "warm" brain. Indeed, physiologically neurons transmit to each other with chemical and electrical synapses; the powerfulness of the electrical discharge of lightning may immediately alters both processes, and in particular the electrical inter-neuronal communication pathway. [14] Lightning strikes cause injuries through different mechanisms. Direct strike is a bolt that hits directly the victim as it happened in this case we report. Another type is the so-called "side splash", a bolt that reaches the victim leaping from a struck object without any contact between the object and the person, contact injury is a consequence of the contact between the person and the struck object. Ground current refers to a strike that hits the ground disseminating to the victim. [1–15] In an upward streamer a person is hit by a current coming from the ground but without a lightning strike hitting the surrounding ground. [15] Half of the lightning strikes are ground currents, while side splash accounts for a third of them. The presence of cardiac arrest survivors after a direct lightning strike is a rare event. The severity of an electric injury relates to the intensity of the current that crosses the victim's tissue [16], which is dependent on the electrical resistance of the different tissues. Bone offers the highest resistance, while muscles, vessels and nerves the lowest. The skin is the main barrier against penetration of the electrical current in the body, but its resistance is significantly impaired by the presence of moist, sweat or water on its surface. [2] Injuries due to electricity occur by four mechanisms: a) light; b) direct effect of electrical current on body tissues; c) conversion of electrical energy to thermal energy, resulting in deep and superficial burns; d) blunt mechanical injury from lightning strike, muscle contraction, or as a complication of a fall after electrocution. [17] Light component can injury the eye causing delayed damage such as cataracts (the most common finding) but also more serious and acute issues like macular holes or edema, optic nerve neuropathy and retinal detachment. Conversely, temperatures that rise up to 8,000°C for a few milliseconds are responsible for skin and hair burns. Electric component is accountable for the damage to the nervous system, to the heart and for the Lichtenberg figures and tip-toe sign. Neuronal and cardiomyocytes death is believed to occur through the cellular mechanism of electroporation: lightning can cause the reorganization of the lipid layer with the generation of pores due to membrane potential's modification. Therefore, cells die from the progressive depletion of energy spent to maintain ion gradient[8]. In the case of direct strike, tissues are severely damaged by the consistent amount of electric charge absorbed by the victim.[18–19] As suggested by Cherrington et al, sometimes the generation of a magnetic field, not necessarily the direct striking, is responsible for detrimental effects to the body, such

as cardiac arrest, thus explaining the absence of signs of injury in some cases. Indeed, even when the current is not sufficient to cause Joule heating it is sufficient to cause nerve and muscle cell electroporation.[20] A classification of neural damage includes 4 types of syndromes: an Immediate and transient syndrome characterized by a loss of consciousness, amnesia, confusion, headaches, paresthesias, weakness and keraunoparalysis (a transient paralysis and anesthesia typically in lower extremities, specific to lightning victim and probably due to intense catecholamines release). As reported in this case, patients hit by lightning may present with pupils that are fixed and dilated or asymmetric due to autonomic dysfunction. As a result, fixed, dilated, or asymmetric pupils should not be used as a reason to stop resuscitation [9]. Motor system diseases and movement disorders belong to the delayed syndrome. The last pattern is secondary to other mechanisms which includes subdural and epidural hematomas and subarachnoid hemorrhage originated from trauma [8–21] Brain lesions are the most common finding at autopsy, whereas cerebellar injuries and injury to peripheral nerves are rare.[21] Hemorrhages of different types and ischemic encephalopathy are widely described. [22] Huan-Jui et al [23] reported a right frontotemporal infarction also involving the basal ganglia after low alternating voltage shock, Eriksson et al [24] reported pons infarction after lightning injury, nevertheless cerebral infarction is a rare finding, commonly linked to vascular damage. Gliomas and cerebral atrophy could also be permanent consequences of lightning shock [21]. There is only a small number of cerebellar syndromes in the literature [21] with a remarkable form of acute atrophy [26] secondary to extensive brain damage, abrupt cerebral salt wasting syndrome has been reported [8–17] The clinical manifestations of electrical injuries range from mild superficial skin burns to severe multiorgan dysfunction and death. The case we present is an example of severe multi organ involvement presenting with cardiac arrest with first monitored rhythm asystole, either due to massive myocardial depolarization or as a degradation of ventricular fibrillation due to several minutes of no flow downtime. Fixed dilated pupils, as exhibited in our case, are also a possible manifestation of electrical injury. The underlying mechanism has been attributed to autonomic dysfunction and may represent an important confounder for prognostication following central nervous injury or cardiac arrest, and should not be used as a reason to stop resuscitation [27] A thorough examination of the patient showed tympanic perforation, which should be sought as it has been reported in 50 to 80% of patients [28]. Rupture tympanic membranes are secondary to barotrauma determined by peak temperature within a bolt of lightning rises within milliseconds to 30,000 Kelvin, generating a shock wave of up to 20 atmospheres induced by the rapid heating of the surrounding air [9,36,37]. This shock wave then can be transmitted through the body and result in mechanical trauma. Thus, damage to internal organs, albeit uncommon, is possible as reported in this case and should be excluded. We also observed rhabdomyolysis that may result from massive tissue necrosis and that was complicated by pigment-induced acute kidney injury. We observed superficial thermal burns that were limited in extension and that went unnoticed at the primary survey. Burns are most common at the site of electrical contact and at places in contact with the ground at the time of injury. The degree of external injury cannot be used to determine the extent of internal damage. Indeed, seemingly minor surface burns may coexist with massive muscle coagulation and necrosis, as well as internal organ injury. Neurological injury was the cause of death of our patient. Vascular and parenchymal injuries of the central nervous system are often associated with peripheral and autonomic

nervous system dysfunction. In our case report we describe the consequence of a direct lightning strike on the neural cells. Cerebral multicentric necrosis, particularly of the basal ganglia, was associated with massive cerebral edema and large areas of absolute neuronal absence, described as desertification. The latter describes an impressive reduction in the number of neuronal elements, not selective by type of neuron, extremely similar to the rarefaction commonly seen in chronic vascular dementia[25]. These conditions would be secondary to the dramatic acute insult that the patient suffered both directly by lightning strike and secondary by edema due to the prolonged cardiac arrest. To date, acute atrophy is reported only in a small number of cerebellar syndromes secondary to lightning shock[24].

Conclusion

We present a fatal case of multisystem injury by direct lightning strike presenting with cardiac arrest followed by initial resuscitation and death due to severe direct neurological injury, with an impressive microscopic pattern of neuronal desertification.

Abbreviations

ICU	
intensive care unit	
CA	
cardiac arrest	
CT	
Computer tomography	
EEG	
Electroencephalogram	
ROSC	
Return on sustained circulation	
GCS	
Glasgow coma scale	
EKG	
Electrocardiogram	

Declarations

Ethics approval and consent participate

The request for approval was submitted and discussed to our local committee: Nucleo di ricerca clinica-Azienda sanitaria universitaria Giuliano isontina - statement n. 2-25.01.2022 . The need for approval was waived due to minimal risk.

Consent for publication

The patient arrived at the hospital in a coma and actually is long dead. The consent was obtained by the next of kin.

Availability of data and materials

All data generated or analysed during this study are included in this published article

Competing interest

The authors declare they have not competing interest

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Authors contribution

Roman-Pognuz Erik: conceived of the presented idea, contribute to the design and implementation of the research, to the analysis of the results and to the writing of the manuscript.

Moro Edoardo: contributed to research data

Macchini Elisabetta: contributed to research data

Di Paolo Edoardo: contributed to research data

Pesenti Kenneth: contributed to research data

Berlot Giorgio: involved in planning and supervised the work

Lucangelo Umberto: involved in planning and supervised the work

Bussani Rossana contributed to the design and implementation of the research, to the analysis of the results and to the writing of the manuscript.

Baratella Elisa contributed to the analysis of the results

Pellis Tommaso contributed to the analysis of the results

Ristagno Giuseppe aided in interpreting the results and worked on the manuscript

All authors read and approved the final manuscript

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Figures

Figure 1

a. Round black eschar. Presumably the entrance gate of the lightning in the body. b. The eschar from a different view.

Figure 2

Erythematous cutaneous lesions with singed hair at the suprapubic level due to the probably output area.

Figure 3

Axial non-enhanced brain TC demonstrates a loss of grey-white matter differentiation at the fronto-temporal lobes bilaterally with gyral effacement. Subtle subarachnoid hemorrhage (white arrows) is also noted.

Figure 4

CT scan performed 4 days later shows diffuse cortical hypodensity of both cerebral hemispheres with a more evident loss of grey-white matter differentiation with gyral effacement and compression of lateral ventricles.

Figure 5

Cerebral parenchymal sections with an abundant, non-selective loss of neuronal elements.