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CASE REPORT

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Lightning-strike induced acute injury: A case report

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Abstract: Lightning strike injury is a rare but a devastating life-threatening condition affecting virtually all tissues and solid organs of the body. This can be attributed to the various energy transmission mechanisms from lightning strikes. This include ground strike, contact voltage, side flash, wire-mediated lightning, direct strike and weak upper streamer. Acute lung injury following lightning strike is an uncommon presentation which can manifest as pulmonary oedema, alveolar haemorrhage, pulmonary contusion and acute respiratory distress syndrome. This is a case report of a 15-year-old female, who presented to the Emergency unit following a lightning strike injury. She presented in coma from the referral centre, markedly dyspnoeic, and eventually admitted into the intensive care unit on account of respiratory failure. Chest radiograph findings showed features in-keeping with non-cardiogenic pulmonary oedema. She was mechanically ventilated, placed on steroids, hydrated, and given antibiotics. Following treatment, respiratory distress resolved and she regained full consciousness. She was discharged home after 11 days of admission.

Keywords: lightning, acute injury, pulmonary oedema, non-cardiogenic

Introduction

Lightning strike is one of the leading causes of weather-related fatalities, although the odds of being struck by lightning in any given year is 1 in 500000.[1] It most often strikes people when they are outdoors with a third of deaths occurring in farms. Males are five times more likely to be struck by lightning than females.[1]Globally, an estimated annual deaths of 24,000 are associated with lightning strikes with cardio-respiratory arrest as the only known direct cause of mortality.[2,3]

Lightning may injure an individual in the following ways; direct strike, side splash from another object, contact voltage from an object that is struck and conducts electricity to the site of touch, ground current effect as energy spreads out across the surface of the earth when lightning hits a distance away from the person and blunt trauma if a person is thrown.[4] The most important characteristic features of lightning injuries are multisystem involvement, primarily neurologic and widely variable severity. Lightning can harm an individual through various mechanisms. These include the effect of electric current on/through body tissues, burns due to conversion of electrical current to thermal energy, mechanical trauma either being thrown due to a transmitted shock wave, direct strike or fall secondary to muscle contraction.

Respiratory injuries associated with lightning strike include pulmonary contusion, pulmonary oedema, pulmonary haemorrhage and acute respiratory distress syndrome.[5] Pulmonary oedema is defined as an abnormal accumulation of extravascular fluid in the lung parenchyma leading to diminished gas exchange at the alveolar level, progressing to potentially causing respiratory failure. It can be broadly classified into cardiogenic and non-cardiogenic pulmonary oedema.[6] Sudden death following lightning strike is usually due to simultaneous cardiac and respiratory arrest and is more common with direct strikes. Lightning may also cause serious injury to the central and peripheral nervous system. It has been found to induce intracranial bleed in some patients, mostly in the brainstem and basal ganglia. Burns ranging from superficial to full thickness are also associated with lightning strikes. Other common lightning strike induced injury include ocular injury from blurnt or blast trauma, cataracts, most often bilateral, hyphema,, vitreous haemorrhage, and optic nerve injury.

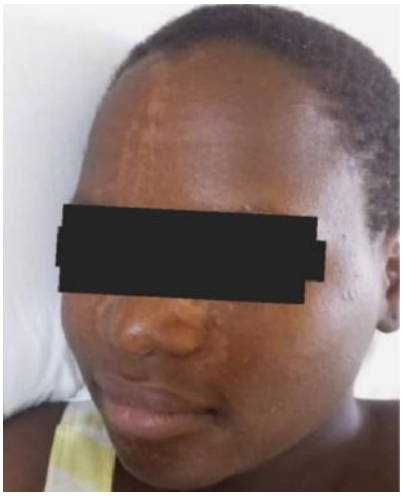
While most paediatric case reports[7,8] on lightning strike describe common injuries such as burns, blunt trauma, cardiac arrest, coma; pulmonary injuries in children who did not sustain severe cardiac injury or cardiac arrest is rare. Hence, the need to report this case, and also to emphasize the management interventions and the excellent outcome.

Case presentation

A 15-year-old girl was admitted into the Trauma unit of University of Benin Teaching Hospital (UBTH),

following a referral from a peripheral hospital, two hours after being struck by lightning. She was brought in unconscious. A bystander during the incident noticed she had been thrown of balance where she was standing just outside a local eatry at a motor park, as a result of the lightning strike. Vital signs at the referral hospital were as follows; pulse rate: 130b/m, blood pressure: 80/40mmHg, oxygen saturation: 40% in room air. She was commenced on intranasal oxygen, given intravenous dextrose saline at 100ml/kg/24hrs and referred to UBTH. At the trauma unit, initial examination showed blood pressure was 126/80mmHg, respiratory rate was 76c/m, and pulse rate was 116b/m. She was saturating at 50% in room air and 75% on oxygen with a face mask. Her Glasgow coma score was 8/15. There was no signs of burns on her skin or hair, however there was a hypopigmented area on her face located between the glabella extending down her nose and cheek area (figure 1).

Fig 1: Facial picture of the index patient with hypopigmented patches following lightning strike



Her respiratory system examination showed she was markedly dyspnoeic, and tachypnoeic, with wide spread crepitations heard on auscultation. Examination of the Cardiovascular system showed that the Apex beat was at the 5th Left intercostal Space on the mid clavicular line and her heart sounds heard where the 1st and 2nd only. Neurologic findings revealed equal pupils which were bilaterally reactive to light. There was no obvious cranial nerve deficit and her muscle tone and deep tendon reflexes were normal. Abdominal findings were not remarkable.

A chest radiograph done at admission (figure 2) showed the following; 'Wide spread fluffy opacities involving both lung fields with air bronchogram and obscuring the cardiac margins hence the heart size cannot be objectively assessed'. Radiologic conclusion was pulmonary oedema

Fig 2: Chest X-ray film on presentation

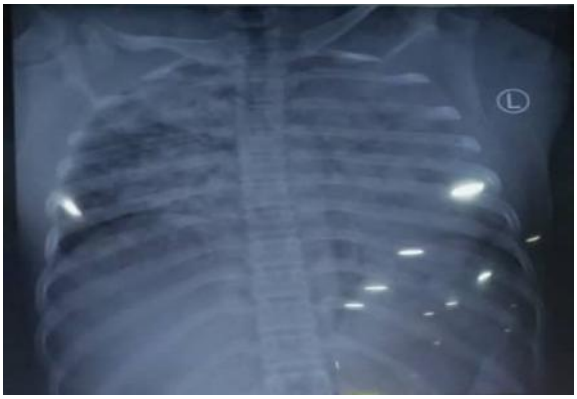
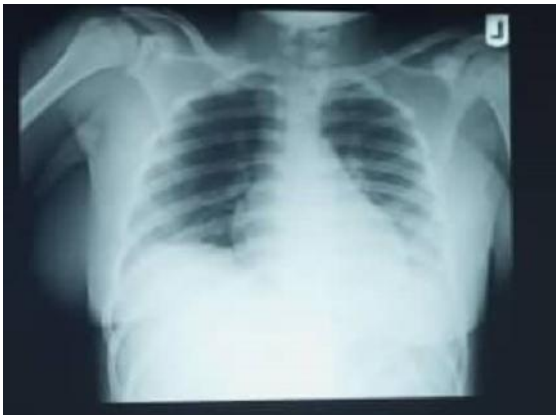


Fig 3: Repeat Chest X-ray film on the 9th day of admission



An Electrocardiograph done showed features of sinus Tachycardia. Laboratory investigations done at admission showed the following findings; Electrolytes showed sodium of 145mmol/l, Chloride of 107mmol/l, potassium of 3.2mmol/l, and bicarbonate of 23mmol/l. Her Urea was 125mg/dl and creatinine was 0.6mg/dl. Her packed cell volume was 45% with a total white cell count of 11400/uL, and platelets of 110000/uL

Arterial blood gases showed a PH of 7.377, PCO2 was 37.6mmHg, PO2 was 23mmHg, SO2 was 39% and HCO2 22.1, in keeping with a Type 1 Respiratory failure.

She was admitted into the intensive care unit of the hospital. She was intubated and placed on a mechanical ventilator. She was commenced on systemic steroids using iv hydrocortisone 4mg/kg 6hourly, and was also nebulised with dexamethasone, hydrated with intravenous fluids using 5%Dextrose saline at 100mls/kg/24hrs. She was commenced on antibiotics; iv Augmentin was used at 60mg/kg/day.

She spent a total of 6 days in the ICU, regained consciousness after 24 hours and made good recovery. She was extubated after 72 hours on admission and oxygen by face mask was discontinued 24 hours after. Respiratory distress resolved on the 5th day with good oxygen saturation of 94% in room air. Had repeat ECG which now showed normal sinus rythm, a repeat chest radiograph was done which showed marked improvement with clearing of the fluffy opacities and no lung disease. She had chest physiotherapy and was commenced on

incentive spirometry. She was subsequently discharged home after 11 days of admission.
She was seen in the paediatric respiratory clinic 3 weeks after on follow up visit. During the visit she gave a written consent for the publication of this case report. She had no complications and has since remained stable though now lost to follow up.

Discussion

Lightning strikes are rare complex event that may be fatal to human life through different mechanisms such as blunt trauma. Blunt trauma mechanism following lightning strike can occur in at least two possible ways.[4] The first mechanism suggests that victims are shot out due to massive opisthotonic muscle contraction caused by lightning strike, secondly it could be through the blast generated by nearby lightning, explosively hitting the air close to the victim which results in the victim being thrown off balance and the subsequent loss of consciousness possibly from cerebral contusion. We postulate that the index patient was possibly injured by one of the 2 ways of blunt trauma mechanism following lightning strike. The hypopigmented facial lesion seen in the index patient on presentation, could be a postinflammatory skin hypopigmentation; which is the partial or total loss of skin color (depigmentation) due to damage of the melanocytes following skin injury.

Non- cardiogenic pulmonary oedema is caused by direct lung injury with a resultant increase in pulmonary vascular permeability leading to the movement of fluid, rich in proteins, to the alveaolar and interstitial compartments.[9] in non-cardiogenic pulmonary oedema type, the oedema is typically patchy and peripheral, demonstrating the presence of ground-glass opacities and consolidations with air bronchogram.[6] It is therefore postulated that in the index patient, the direct blast effect on the lungs following lightning strike was responsible for the non-cardiogenic pulmonary oedema as evidenced by the chest X-ray report and the ECG tracing that showed no significant changes. Dhawan *et al*[10] reported a chest x-ray finding of bilateral upper and middle lobe pulmonary oedema with complete lower-lobe sparring following a lightning strike which was quite dissimilar to the Chest x-ray findings of the index patient. This they attributed to a myocardial injury, evidenced by the echocardiographic finding of ventricular dysfunction.[10]
The exact mechanism of cardio-respiratory arrest following lightning strike injury is unknown however, It has been theorized that lightning depolarizes the entire myocardium at once, causing a single systolic contraction followed by a variable period of asystole. Cardiac activity may resume spontaneously, initially at a lower heart rate and then slowly increases in speed (tachycardia). This rhythm may deteriorate from apnea and hypoxia resulting from paralysis of the respiratory center in the medulla.[9] Thus pulmonary oedema usually occur in association with severe cardiac damage, however in our index patient it was a non-cardiogenic pulmonary oedema. Increased capillary permeability and changes in pressure gradients within the pulmonary capillaries and vasculature are mechanisms for which non-cardiogenic pulmonary edema occurs. ECG done in our index case showed only sinus tachycardia, with no evidence of severe myocardial injury.
Sener *et al*[11] reported computed tomography (CT-scan) findings of pulmonary contusion and pleural effusion in their study, which they attributed to blast effect of the lightning. Oflu *et al*[2] also reported similar CT-scan finding of pulmonary contusion but with no associated pleural effusion. These findings were not established in our patient based on the chest X-ray report. Hence, this shows the varying manifestations of acute lung injury following lightning strikes and the need for physicians to evaluate victim of lightning strike holistically and equip themselves with myriads of presentations of acute lung injury following lightning strike.[12]

Conclusion

Lightning strike injury is a rare but devastating condition, characterised by varying systemic manifestations depending on the mechanism of injury. Non-cardiogenic pulmonary oedema, an uncommon manifestation of lightning induced direct lung injury should be considered by the physician treating lightning victims with respiratory symptoms.

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