Electric Shock-Induced Pulmonary Hemorrhage – A Rare Phenomenon

Sir,

Acute pulmonary hemorrhage due to electric shock is a rare phenomenon. A 25-year-old male, electrician by occupation, accidentally was exposed to a 220 voltage electric shock while fixing a domestic power supply. After the impact, he fell down and had a brief 10–20 s of tonic movement of the upper limbs with uprolling of eyeballs. The seizure ceased abruptly, and there was a short period of postictal confusion lasting another 1–2 min according to the fellow worker. He was brought to the casualty of this hospital. In the casualty, his vitals were stable. He was conscious and oriented to time, place, and person. Pulse was 96/min and regular, blood pressure (BP) was 138/84 mmHg in the right arm, and the oxygen saturation by pulse oximetry (SpO_2) was 96% while breathing ambient air. There was no evidence of any trauma to chest in the form of bruise, ecchymosis, or fracture rib. Examination of other systems was normal. There were no focal neurological deficits. The electrocardiogram (ECG) and electroencephalogram were normal. The patient was not willing for admission and observation and was discharged from the hospital.

Seven hours later, the patient was again brought to us by his relatives with complaints of coughing up of approximately 10 ml of bright red blood twice. There was no history of chest pain, dyspnea, orthopnea, palpitations, abdominal pain, or vomiting. His pulse rate was 88/min, regular, respiratory rate 18 cycles/min, BP 120/80 mmHg, and SpO_2 on room air was 94%. Auscultation of the chest revealed bilateral scattered crepitations. Chest X-ray (CXR) showed bilateral heterogeneous opacities [Figure 1]. High-resolution computerized tomography scan of the thorax showed bilateral perihilar ground-glass opacities and thickening of interlobular septa with thickening of the bronchial wall [Figure 2].

Arterial blood gas analysis was normal, serum creatine phosphokinase – 600 U/L, lactate dehydrogenase was 266 U/L, creatine kinase MB 27 U/L, serum urea 38 mg%, serum creatinine 0.9 mg%, and urine myoglobin was negative. The serum N-terminal pro b-type natriuretic peptide-148 pg/ml (normal <300 pg/ml). The platelet count and coagulation profile (bleeding time, clotting time, prothrombin time, and partial thromboplastin time) were within normal range. Repeat ECG and 2-dimensional echocardiogram were normal.

A provisional diagnosis of electricity-induced lung injury with pulmonary hemorrhage was entertained. He received intravenous tranexamic acid 500 mg thrice daily for 3 days and intravenous fluids. He had 3 additional although minimal amount of hemoptysis in the hospital. On day 3, his hemoptysis stopped. A repeat CXR performed on the 5th day showed complete resolution of the opacities, [Figure 3], and the patient was discharged.

Very few cases of electric shock-associated lung injury have been reported.[1-5] One was ventricular fibrillation-induced pulmonary edema;[1] two reports documented focal consolidation on lung imaging following high voltage (1000 V) shock.[2,3] Another report was of focal lung injury in an electrician after he got a low-voltage shock (380 V),[4] and finally, pulmonary hemorrhage in an agricultural worker following low voltage shock.[5] Our patient received a shock by low-voltage electrical current.

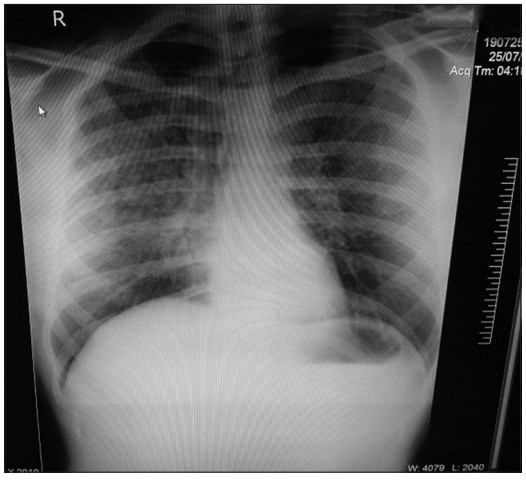

Figure 1: Chest X-ray showing bilateral pulmonary infiltrates

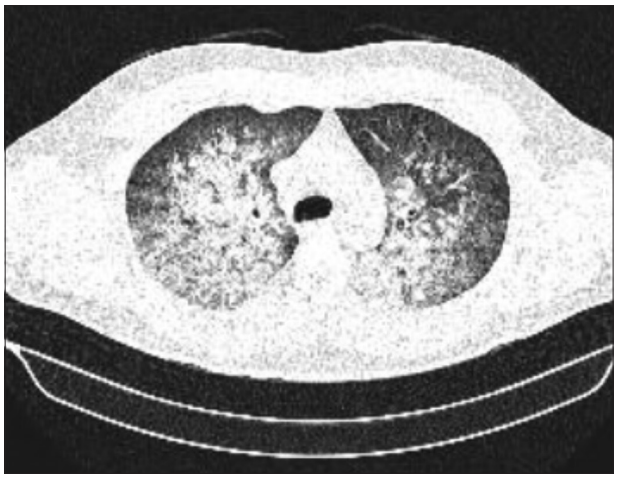

Figure 2: High-resolution computed tomography thorax showing bilateral opacities suggestive of pulmonary hemorrhage

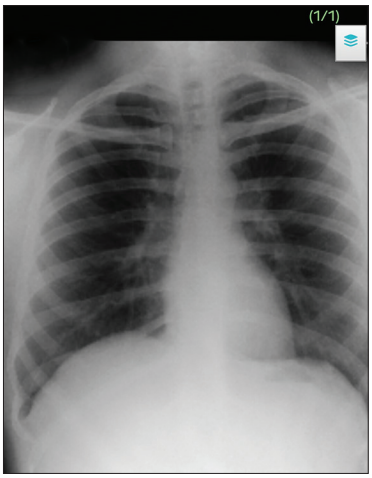

Figure 3: Chest X-ray showing clearing of infiltrates

Electric shock can cause pulmonary edema and/or hemorrhage by either cardiac arrest or as consequence of direct lung injury. Patients can present as hemoptysis, respiratory distress, and respiratory failure. The main pathologic manifestation of electrical lung injury is usually a coagulative necrosis. The mechanisms attributed are direct tissue damage which alters resting membrane potentials of the cells, conversion of electrical energy to thermal energy (Joule heating) that causes tissue destruction and necrosis, and finally, direct trauma from violent contraction of muscles. In addition, electrical energy can also cause electroporation, in which conformational damage of channel proteins occurs because of reorientation of polar amino acid residues.[6] All these mechanisms finally cause cell membrane dysfunction, injury, rupture, and necrosis. Although there exists guidelines for the management of electrical burns according to the advanced trauma life support, advanced burn life support, and advanced cardiac life support,[7] there are no specific guidelines for management for electric shock-induced pulmonary hemorrhage.

Our case is second to report bilateral pulmonary hemorrhage without left ventricular failure after electric shock.

Informed consent was obtained from the individual for publication.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given his consent for his images and other clinical information to be reported in the journal. The patient understands that name and initials will not be published and due efforts will be made to conceal identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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