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High-Voltage Electrical Lung Injury

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A 23-year-old male was admitted to the intensive care unit (ICU) with high-voltage electrical injuries. During construction work, he touched a cable carrying 20,000 V and lost consciousness. He was brought to the ICU intubated, unconscious, with a Glasgow Coma Scale of 7-8, hypotensive, in cardiogenic shock, and presented massive hemoptysis. Vital signs indicated a heart rate of 120 beats per min, a temperature of 37 °C, arterial blood pressure of 90/60 mmHg, and peripheral capillary oxygen saturation (SaO_2) of 80%. He had no significant previous medical history. Physical examination revealed auscultatory crackles and crepitations, with occasional wheezing in both lungs. The electric current entered on the left shoulder and exited on the right, resulting in minor first- and second-degree burns (Figure 1a, b). Chest radiography showed heterogeneous infiltrates, especially on the right (Figure 1c), and a lung computed tomography (CT) scan revealed ground-glass opacities predominantly on the right (Figure 1d). Laboratory results indicated a decrease in hemoglobin (95 g/L) and hematocrit (0.28 L/L), along with elevated transaminases (aspartate aminotransferase 166 IU/L, alanine aminotransferase 231 IU/L), lactate dehydrogenase (613, IU/L), and amylase (555 U/L). Serum troponin and N-terminal pro-B-type natriuretic peptide levels were normal. Platelet count, prothrombin time, and partial thromboplastin time were also normal. Urine myoglobin was negative, and there were no abnormalities on the electrocardiogram or echocardiogram. Treatment included supportive oxygen, intravenous hydration, antibiotics, systemic steroids, and daily bronchoaspiration, up to four times daily. Initially, hemorrhagic discharges were collected, followed by a thick, viscous secretion that acted as a medium for bacterial development. *Acinetobacter* was isolated from bronchoalveolar lavage and was sensitive to imipenem and colistin. The patient received antibiotic therapy according to the antibiogram. Ten days post-injury, he was weaned from ventilator support, with an SaO_2 of 96% on room air and adequate acid-base status. He was conscious and exhibited no residual neurological deficits. He was discharged with clinical and radiological improvement, and a follow-up CT scan of the lungs showed no pathological findings.

Electrical lung injuries are rare, occurring when electricity damages lung tissue. Both high (≥ 1000 V) and low-voltage (< 1000 V) can cause lung damage,[1-3] either isolated or alongside other injuries.[4-7] Although they represent a small percentage of mechanical injuries, they are significant due to their association with high morbidity and mortality.[5] Types of electrical lung injuries include acute pulmonary edema and focal lung parenchymal damage.[1-6] Electric current can cause conformational changes in protein channels of the cell membrane, leading to electroporation of the cell.[4] Injuries to organs and tissues are not only a result of the direct action of electricity but can also of accompanying thermal and mechanical effects. The most

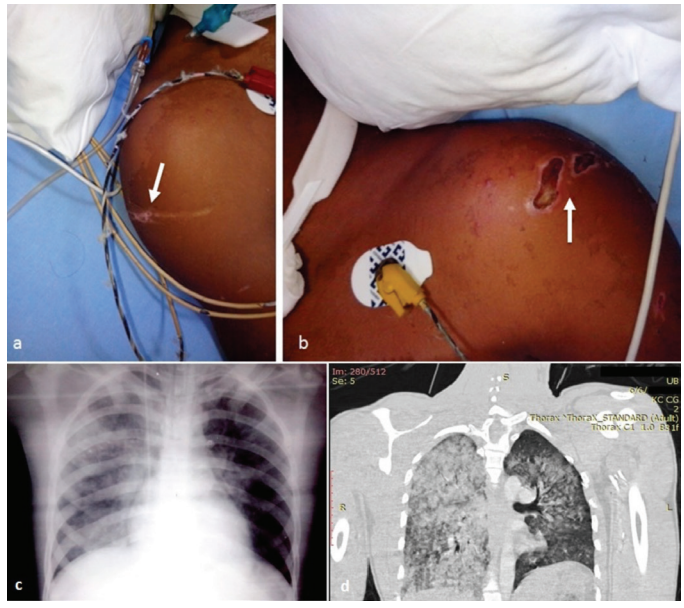

FIG. 1. Minor first- and second-degree burns on the right (a) and left shoulders (b). Chest radiograph demonstrates heterogeneous opacities, particularly on the right side (c). Lung computed tomography scan shows ground-glass opacities, primarily on the right side (d).

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serious injuries are caused by lightning, followed by high- and low-voltage injuries.[8] Thus, increasing voltage correlates with increased injury severity. Tissue resistance plays an important role; high skin resistance results in a significant energy loss at the skin level, leading to burns, but may protect internal organs. Conversely, low skin resistance can allow more electricity to reach internal organs.[3] Therefore, the extent of skin injuries does not necessarily correlate with the degree of internal organ damage. Electrical injuries can elevate thromboxane A2 levels, causing vasoconstriction and thrombosis, particularly in small nutrient arteries, contributing to tissue necrosis.[7-9] Elevated catecholamines secretion can raise pulmonary vascular resistance and damage the alveolar wall, leading to pulmonary edema.[6]

Due to their rarity and limited data in the literature, diagnosing electrical lung injuries is challenging. Consequently, we believe that every patient who sustains an electrical injury should be screened for potential lung injury, as timely diagnosis and treatment increase the likelihood of survival and can facilitate complete recovery without major consequences.

Informed Consent: Written informed consent was obtained from the patient.

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