Case Report

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Acute Lung Parenchymal Injury Caused by High-Voltage Electrical Injury: A Case Report and Literature Review

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High-voltage electrical injury (HVEI) causes devastating injury with mortality rate ranging from 3% – 15%. However, direct injury to the internal organ was only 1.7% and isolated lung injury is even rarer. Up to now, only eight cases of HVEI associated lung injury were reported and six of them involved direct injury to the lung. We report a fireman, who suffered from a 12,000 V HVEI presenting with blood-tinged sputum. Although chest X-ray showed diffuse infiltration, chest computed tomography (CT) demonstrated posterior lobe consolidation suggestive of acute lung parenchymal injury due to HVEI. He received endotracheal intubation and mechanical ventilation. The blood in the sputum disappeared five days after HVEI and weaning with extubation was successful 24 hours later. We report this case and review the literature about the etiology, diagnosis and treatment published in English literature. Our case highlighted the importance of an early correct diagnosis as well as the role of whole body CT in assessing the nature of lung injuries and ruling out hidden visceral injuries, to which all emergency and burn care physicians should be alert when encountering patients diagnosed with HVEI.

Key words: high-voltage electrical injury, lung parenchymal injury, whole body computed tomography, blood-tinged sputum, chest X-ray

Introduction

Electrical injury is categorized as high-voltage (> 1,000 V) or low-voltage (< 1,000 V).[1] High-voltage electrical injury (HVEI) causes devastating injuries such as neuromuscular injuries, cardiac arrhythmia/arrest, pulmonary injuries, renal failure (myoglobinuria), respiratory arrest, cerebral edema,[2] arterial thrombosis, and spinal injury.[3] In France, the mortality rate ranges from 3% – 15%.[4] Occupational HVEI is the fifth leading cause of death in the United States.[5] The severity of the electrical injury is determined by the voltage of current, duration of contact to the source,[6] and the tissue resistance to the electric current in the transmission pathway.[1] Injury to the internal organ is only 1.7% and isolated lung injury is even rarer.[1,2] Here, we report

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a case of HVEI with acute lung parenchymal injury and a review of the literature regarding the etiology, diagnosis, and treatment.

Case Report

A 35-year-old male fireman suffered from an electric injury on August 24, 2012. A typhoon attacked Taiwan, and a 12,000 V cable was broken by the strong wind. As the on-duty fireman, he was responsible for managing the situation. At the time, he was wearing a standard fire-fighting helmet and coat. At the scene, the fireman tried to cut the damaged cable with a wire cutter to stop the end of the cable from whiplashing and sparkling on the ground. Unfortunately, he received an electric shock because the power had not been cut off by the electric company. He lost consciousness. His colleague started cardiopulmonary resuscitation immediately. When the ambulance arrived at the scene four minutes after the call, his heartbeat had returned and the fireman was conscious. Upon arrival at the emergency room (ER), he reported pain. The fireman also became confused. His Glasgow coma score was E4M1V5. His vital signs were as follows: body temperature, 35.5°C; pulse, 160 beats/min; respiration rate, 35/min; blood pressure, 135/68 mmHg; and oxygen saturation (SaO_2), 88% in room air. Due to confusion and low SaO_2, emergency intubation was performed. After intubation, his SaO_2 increased to 98%. Physical examination showed a third-degree burn wound measuring 15 cm × 15 cm on the occipital-parietal area, a fourth-degree burn wound on the right forearm with wrist and finger flexion contracture, fourth-degree burn wounds on the right leg, right foot, left calf with muscle exposure and necrosis and the dorsal of the left big toe, one third-degree wound on the right buttock, and multiple third-degree spotted burn wounds on the back (Fig. 1). Chest examination showed coarse breath sounds with rhonchi and rales, but no chest tenderness. The abdomen had neither tenderness nor rebound pain. The four limbs had no bony deformity, and the cervical spine and pelvic bones were stable. Laboratory data showed a serum creatine phosphokinase (CPK) level of 14,827 U/L, creatine kinase-MB (CKMB) level of 103.3 ng/mL, troponin-I < 0.01 ng/mL, and myoglobin level of 35,500.25 ng/mL. Arterial blood gas analysis under room air showed a pH of 6.988, PaO_2 of 54.9 mmHg; PCO_2 of 58.9 mmHg and HCO_3- of 13.8 mM/L. The chest X-ray (CXR) showed ill-defined opacities in the middle zones of both lungs and elsewhere in the right lung (Fig. 2). The axial view of the

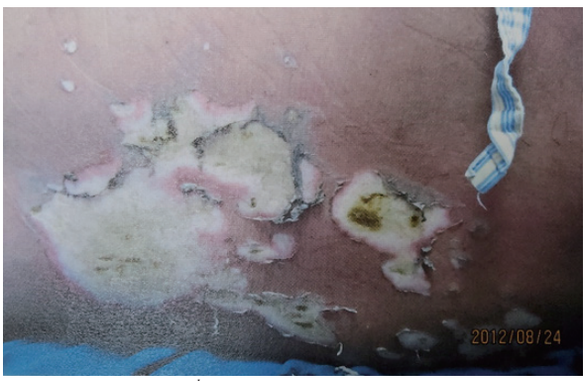

Fig. 1 Multiple 3rd degree spotted burn wounds on the back. The cephalic side was on the right side of the photo.

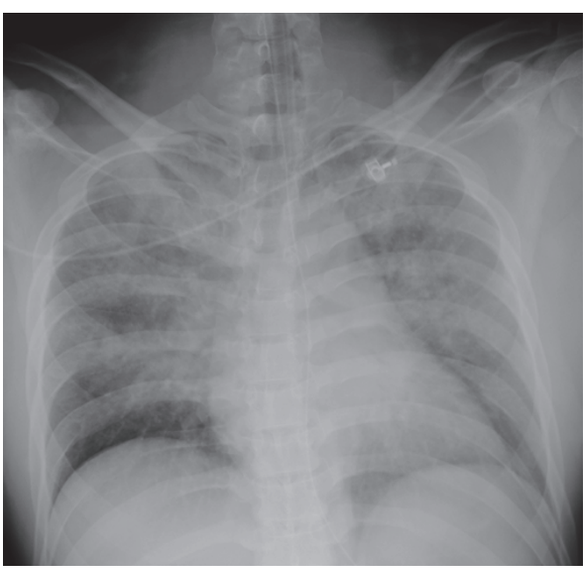

Fig. 2 The chest X-ray on 8/24 at ER showed ill-defined opacities at middle zones of both lungs, right lung predominantly.

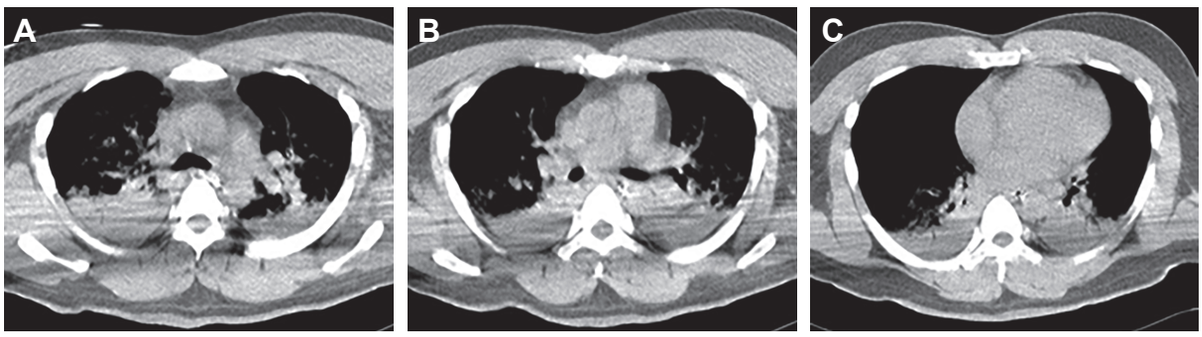

Fig. 3 The axial view of chest CT at ER showed relatively symmetric consolidation at dependent zones of both lungs. However, the consolidation of right upper lobe seemed larger than that of left upper lobe. (A) Chest CT axial view at primary bronchus level. (B) Chest CT axial view at inferior lobar bronchus level. (C) Chest CT axial view at segmental branches level of inferior lobar bronchus.

chest computed tomography (CT) showed relatively symmetric consolidation at dependent zones of both the lungs, but the consolidation of the right upper lobe seemed larger than that of the left upper lobe (Fig. 3). The coronal view showed diffuse symmetric consolidation of both lungs, but there was a spared area in the basal portions (Fig. 4A). The sagittal view showed skin thickening and increased subcutaneous infiltration of the back, corresponding to lung consolidation (Fig. 4B). Electrocardiography (ECG) showed sinus tachycardia without ST-segment elevation. A nasogastric tube, Foley catheter, and central venous catheter were inserted. He was sent to the burn center (BC) for specialized care.

In the BC, fluid resuscitation with lactated Ringer's solution according to the Parkland formula was given. The urine output was kept at 1 – 2 mL/kg/hr. Alkalinization of the urine with sodium bicarbonate was also performed to prevent the renal tubule from obstructing myoglobinuria. Blood in the sputum was noted during suction through the endotracheal tube. An acute lung parenchymal injury due to HVEI was suspected. Positive pressure ventilation, broad-spectrum antibiotics (tazocin), and aggressive chest care were administered. Albumin was infused 24 hours after burn injury. Overhydration was also prevented by carefully monitoring the urine output and using pulse contour cardiac output (PiCCO) to monitor the central venous pressure, cardiac index (CI), and extravascular lung water index (EVLWI). On August 25 (post-burn day 1 [PBD1]), the lung field became clearer, but blood was still present

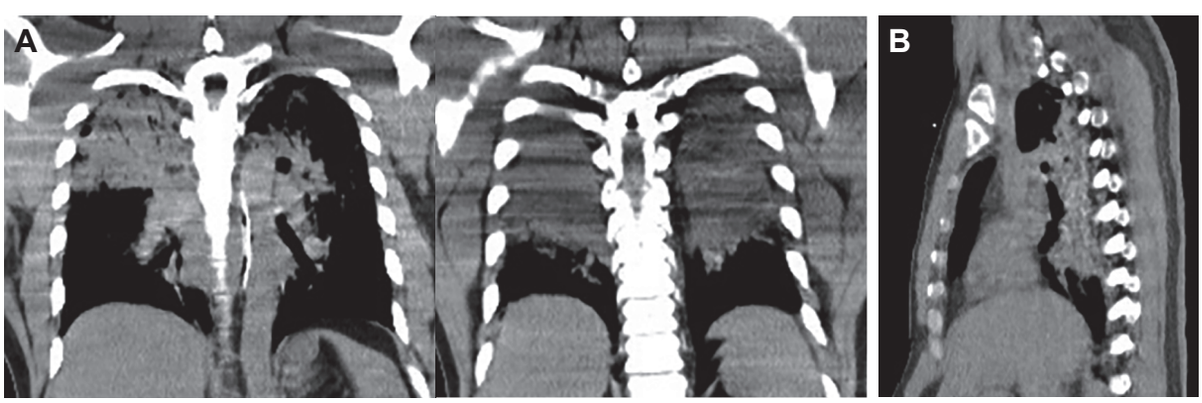

Fig. 4 (A) The coronal view of chest CT at ER through the costovertebral junction of upper thoracic spine revealed diffuse symmetric consolidation of both lungs but spared area in the basal portions of both lungs. (B) The sagittal view of chest CT at ER through right paraspinal area demonstrated skin thickening and increased subcutaneous infiltration of the back corresponding to the lung consolidation.

in the sputum. The condition of viscous sputum with blood persisted until August 29 (PBD5). On August 30 (PBD6), the CXR was much clearer (Fig. 5) and the sputum was yellowish without blood. The endotracheal tube was smoothly removed after successful weaning. The CXR showed complete resolution on September 4 (PBD11) (Fig. 6). The myoglobin level dropped to 1,580 ng/mL on PBD3.

After emergency fasciotomy and a series of debridement, the right leg and right forearm were amputated. The exposed scalp bone was covered with a free anterolateral thigh flap. The back and buttock wounds healed by secondary intention, and the left leg, the left foot, and the right knee amputation stumps were covered with a split-thickness skin graft. The fireman underwent an aggressive rehabilitation program after stabilization of his wound condition during his hospital stay. He was discharged on PBD77 without any sequelae of the heart, lung, kidney, or neuromuscular system.

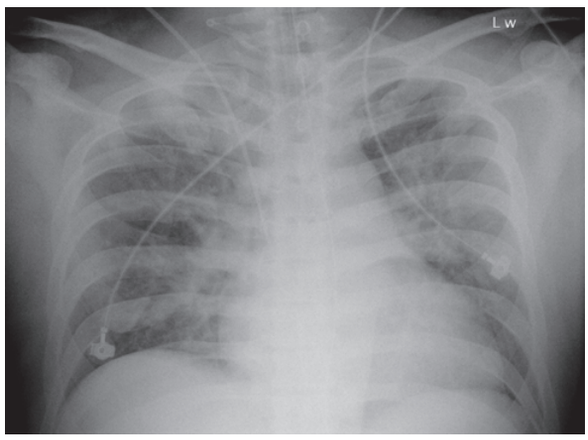

Fig. 5 The chest X-ray on 8/30 (PBD6) showed interval resolution of bilateral lung consolidation with residue at left perihilar zone.

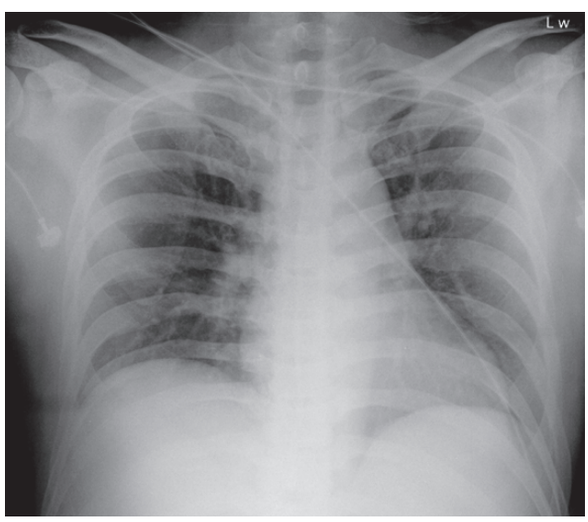

Fig. 6 The chest X-ray 9/4 (PBD11) showed no obviously abnormal opacity in the lung field, which implied complete resolution of initial bilateral lung consolidation.

Discussion

Electrical injury damages the human body in three ways. First, thermal injury can result from tissue's resistance to the electric current according to Ohm's law. Second, electroporation may cause dipoles to form pores on the cell membrane. Third, electricity may trigger denaturation of the voltage-gated channel proteins on the cell membrane.[3] Consequently, tissue necrosis results due to an increased permeability of the cell membrane.[1,2,7] HVEI-associated lung injury is very uncommon. From 1990 to 2021, there were only eight case reports of HVEI-related lung injury in the English literature, six of which were direct injuries to the lungs.[1,2,4-7] The first case was HVEI-induced pulmonary edema reported by Schein et al. in 1990.[8] Our case is the ninth case. Lung injury can be either primary or secondary. Primary lung injury (non-cardiogenic) is caused by direct electric injury to the lung parenchyma, causing lung tissue necrosis,[4,5] pulmonary hemorrhage with hemoptysis[1,7] without special complaint.[2,6] Secondary lung injury is a sequela of other organ injuries, such as cardiogenic shock-induced pulmonary edema[8] or neurogenic pulmonary edema.[9]

The initial loss of consciousness may be due to temporary cardiac arrest or diaphragm contracture in a tetanus state, which is more often associated with alternating current injury because of continuous muscle contraction. In this situation, the lung injury is secondary to cardiac injury and the patient is unable to

breathe. Temporary ventricular dysfunction or arrhythmia may not cause cardiac arrest but still lead to pulmonary edema.[7] In a study of the Army Brook Military Center, of 195 HVEI, 32.3% had loss of consciousness with or without cardiac arrest and 7.2% of victims with consciousness loss sustained apparent cardiac arrest right after contact with the electric source.[3] Initial loss of consciousness is common and accounts for 45% of patients with HVEI, while 79% regained, consciousness without neurological complications on arriving at the hospital.[3] Our patient lost consciousness and had cardiac arrest, then the heartbeat returned after cardiopulmonary resuscitation (CPR). This is a typical course for HVEI-induced cardiac arrest or arrhythmia.

In the literature, the physical findings associated with lung injuries have been reported. Copious amounts of pink frothy sputum, bilateral wheezing, rhonchi, and rales are typical signs of pulmonary edema.[8] Hemoptysis due to hemorrhage indicates lung parenchymal injury.[1,7] The correlation between the extent of burn wound and the degree of visceral injury is low and deceiving.[1-3] Although devastating visceral damage may hide deep in a small burn wound, the physical findings of the inlet and outlet can predict the possibility of lung injury.[1] Our patient had third-degree burn wounds on the back (Fig. 1), which may indicate primary lung injury.

Laboratory studies may help to identify HVEI-induced cardiac, pulmonary, renal, and muscular injuries. Cardiac enzymes,[1] troponin-I, arterial blood gas, electrolytes, blood urea nitrogen and creatinine, myoglobin level, and CPK have been mentioned in the literature. Myoglobinuria is due to massive muscle necrosis and predicts the need for amputation and fasciotomy.[3] ECG for rhythm study and ST-segment elevation is also useful. In our case, the troponin-I was less than 0.01 ng/mL, creatine kinase (CK) was 14,827 ng/mL, CKMB was 60.9 ng/mL and ECG showed no ST elevation. Therefore, cardiac injuries could be excluded. The initial myoglobin level was 35,500.25 ng/mL. Eight hours later, it declined to 14,823.09 ng/mL. Later, the level dropped by 50% on each subsequent day. On PBD2, the myoglobin level was 1580.67 ng/dL, which is harmless to the renal tubules. The patient underwent right arm and left leg amputations. These procedures would have contributed to the rapid decline in the myoglobin level.

Image studies are helpful for diagnosing lung injuries. CXR may show opacification and interstitial edema[8] or infiltration of lung fields[1] and chest CT scan might show focal consolidation,[1,6] lung collapse,[4] pulmonary embolism,[5] or cavitation of lung parenchyma.[5] Currently, there is no consensus regarding the use of chest CT scans to exclude intrathoracic injury in suspected cases.[1] Chen et al. suggested non-contrast enhanced chest CT scan for abnormal CXR and CT angiography for massive pulmonary hemorrhage.[1] Brain CT is needed if the patient is unconscious.[2] If the heart is involved, cardiac echography may show depressed myocardial contractility and blood flow anomaly.[8] Invasive hemodynamic monitoring such as pulmonary artery catheter placement to measure pulmonary artery wedge pressure and CI may also help to clarify the origin of the heart-lung problem and check the fluid status to prevent significant volume overload.[8] Once lung injury is diagnosed, bronchoalveolar lavage of the injured lobe may show enhanced cellularity, especially in polymorphonuclear leukocytes, due to localized inflammation.[2] Bronchoscopy can also be performed to exclude or evaluate an inhalational thermal lung injury. In some cases, the absence of electric burn on the chest or entry/exit points cannot rule out lung or other visceral injuries.[2,6] Significant discrepancies between the burn wound and the underlying visceral injuries[7] are possible and may result in a missed diagnosis. Therefore, we suggest routine CT scans for the brain, chest, and abdominal regions at the emergency department. In our case, the patient's CXR showed diffuse

infiltration in both the lungs. This may lead to the diagnosis of pulmonary edema or pneumonia. However, these conditions seldom happen immediately after a burn injury except in those with a pre-existing lung disease. In addition, the absence of fever and leukocytosis excluded the possibility of pneumonia. The chest CT scan showed only posterior lobe consolidation but not diffuse infiltration. This indicated that the lung parenchymal injury was more likely than pulmonary edema, especially in the presence of viscous sputum with blood.

The treatment of HVEI-induced lung injury usually requires intubation and mechanical ventilation.[2,4,8-10] After supportive treatment, pulmonary edema will generally subside, but lung necrosis may need resection.[4,5] Hemoptysis due to hemorrhage can be treated with tranexamic acid[1,7] for five to seven days.[1,7] Although there is no medical treatment to prevent lung necrosis and limit microvascular thrombosis, pulmonary embolism requires anticoagulation therapy.[5] Resection of the injured lung is unavoidable if lung infarction or severe parenchymal injuries develop.[4,5] The pathological picture of liquefaction, coagulative necrosis, abscess, and lipid resorption into the alveolar lumen[4,5] all may indicate unsuccessful conservative treatment. For electrical injuries, despite fluid resuscitation with the Parkland formula to prevent dehydration, alkalinization of urine with sodium bicarbonate, forced diuresis with mannitol, and 12.5 g per liter of lactated Ringer's solution are common strategies for preventing renal tubular obstruction due to myoglobinuria.[3,10] The adequacy of fluid resuscitation should be monitored with urine output 100 – 150 mL/h without gross myoglobinuria[10] and 70 – 100 mL/h with gross myoglobinuria.[3] Early fasciotomy, amputation burn wound excision, and skin grafting are still the gold standards for burn wound treatment. Ongoing muscle necrosis due to ischemia-reperfusion injury makes it difficult to determine muscle viability at the time of inspection.[10]

Conclusion

HVEI-associated lung injury is not common. Direct injury to the lung parenchyma is also rare. Although history, physical examination, laboratory data, and imaging studies can diagnose HVEI-related lung parenchymal injuries, the clinical diagnosis is often made by ruling out other differential diagnoses related to the lung problems in critical care.[5] Emergency and burn care physicians should be mindful of possible visceral injuries when managing HVEI. A whole-body CT scan is strongly recommended as a routine examination in the emergency department to identify hidden injuries in the brain, spine, chest, and abdomen. A chest CT scan is also valuable for excluding pulmonary edema or pneumonia and further identifying the injured lobe and confirming any lung parenchymal injury. A timely diagnosis to guide appropriate treatment is life-saving in this clinical setting.

Author Contributions

Study Design, Li-Ren Chang; Image Interpretation, I-Chang Lin; Data Collection, Chien-Chung Chen; Case Discussion, I-Ting Tsai; Literature Review, Guang-Ming Feng; Manuscript Review, Seng-Feng Jeng. All authors have read and agreed to the published version of the manuscript.

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Conflicts of Interest

The authors declare no conflict of interest.

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