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Electrical Injury–Induced Pulmonary Embolism: A Case Report

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ABSTRACT

Electrical injuries are most commonly associated with cutaneous burns, arrhythmias, and neuromuscular damage; however, thromboembolic complications are rarely described. Low-voltage electrical exposure may trigger endothelial disruption, inflammation, and a hypercoagulable state that predisposes to in-situ pulmonary artery thrombosis. We present the case of a 34-year-old male who developed progressive dyspnoea, chest pain, and hypoxaemia several days after an occupational electrical injury. Diagnostic evaluation revealed elevated D-dimer levels, right ventricular dilatation with signs of pulmonary hypertension on echocardiography, and no evidence of deep venous thrombosis. CT pulmonary angiography identified acute segmental pulmonary embolism with pulmonary infarction. The patient was successfully treated with anticoagulation and supportive therapy, resulting in clinical improvement. This case highlights the potential for low-voltage electrical trauma to precipitate pulmonary vascular injury and emphasizes the importance of early imaging and prompt anticoagulant therapy. Further studies are necessary to better understand the pathophysiological mechanisms and incidence of thromboembolic events following electrical injury.

Keywords: Electrical injury, low-voltage electric shock, pulmonary embolism, in-situ pulmonary thrombosis, pulmonary infarction, endothelial dysfunction.

INTRODUCTION

Electrical injuries, including those caused by low-voltage exposure, are traditionally associated with cutaneous burns, cardiac arrhythmias, and neuromuscular complications. However, emerging evidence suggests that electrical current may also contribute to vascular and thromboembolic events, although such complications remain exceedingly rare and underreported. The pulmonary vasculature, in particular, may become

vulnerable to both direct and indirect mechanisms of injury following electric shock [1,2].

Low-voltage electrical exposure has the potential to disrupt endothelial integrity through thermal, mechanical, and electroporative mechanisms. Damage to the vascular endothelium may initiate a prothrombotic cascade characterized by platelet activation, increased vascular permeability, and localized coagulation. These alterations can theoretically predispose patients to venous thromboembolism (VTE), including pulmonary embolism and subsequent lung infarction, particularly when the electric current traverses the thorax or lower extremities [3,4].

Furthermore, systemic responses following electrical injury—such as immobilization, rhabdomyolysis-induced hypercoagulability, dehydration, and inflammatory cytokine release—may create additional risk factors for thrombosis. Although only sporadic case reports describe PE or lung infarction following electrical accidents, these instances highlight a plausible pathophysiological link between electrical trauma and thromboembolic complications [5]. Lung infarction, resulting from compromised perfusion distal to the obstructed pulmonary artery, may manifest clinically with pleuritic chest pain, haemoptysis, and radiographic wedge-shaped opacities, potentially overlapping with findings of direct electricity-induced lung injury [6].

Given the limited literature and the absence of large-scale studies, the relationship between low-voltage electrical exposure and pulmonary thromboembolism remains poorly defined. Nevertheless, recognizing the possibility of vascular involvement is important for clinicians evaluating patients after electrical accidents, especially when respiratory symptoms such as dyspnoea, chest pain, or haemoptysis develop in a delayed fashion.

Aim: This report aims to contribute to the growing body of evidence by discussing the potential mechanisms and clinical significance of pulmonary embolism and lung infarction in the context of low-voltage electrical injury.

METHODS

This study is presented as a single-patient case report supplemented by a narrative review of the existing literature on pulmonary complications following low-voltage electrical injury. Clinical data were collected prospectively during the patient's hospitalization at a tertiary-level emergency and critical care center. All diagnostic evaluations, laboratory measurements, and imaging procedures were performed as part of routine clinical management.

RESULTS

To characterize the pulmonary and cardiovascular consequences of electrical exposure, the following investigations were performed:

A 34-year-old male, with no significant past medical history of chronic cardiovascular or pulmonary disease, presented to the emergency department with persistent retrosternal chest pain radiating to the back, dyspnea, chills, cold sweating, and general weakness.Three days prior to admission, the patient experienced an electrical injury at work, after which he noted progressive malaise and exertional dyspnea. On the day prior to admission, the patient developed severe chest discomfort radiating to the left hemithorax, accompanied by shortness of breath, prompting evaluation at a regional medical facility and subsequent transfer to reanimation department. Pre-hospital ECG showed sinus tachycardia without ischemic ST–T abnormalities. Vital signs were as follows: Temperature: 36.6 °C. Blood pressure: 90/60 mmHg. Heart rate: 112 bpm. Respiratory rate: 24 breaths/min. SpO_2: 85-95% air. Physical examination revealed pale, moist skin, with no peripheral edema. Cardiovascular examination demonstrated regular rhythm without murmurs. Lung auscultation revealed diminished breath sounds bilaterally without wheezes or crackles. The abdomen was soft and non-tender. There was no calf tenderness or unilateral leg swelling. Neurological exam was unremarkable. Laboratory Findings demonstrated: Hemoglobin: 130–129 g/L, Leukocytes: 6.3–6.4 $\times 10^9$/L, Platelets: 200–219 $\times 10^9$/L, ESR: 17 mm/h, CRP: 15 mg/L. D-dimer 5.7 µg/mL (elevated) , Renal function: Creatinine 73 µmol/L; Urea 5.1 mmol/L. Liver enzymes: ALT 27; AST 46 U/L. Coagulation: INR 1.03. Coagulation profile showed increased clotting activity (reduced clotting time). Troponin I: 0.18 ng/mL; in dynamic< 0.10 ng/mL;). ECG: Sinus rhythm 92–95 bpm; no ischemic changes; nonspecific ST-T abnormalities. Echocardiography: Right ventricular dilatation, left ventricular ejection fraction preserved, signs of pulmonary hypertension. sPAP 41 mmMg, increased tricuspid regurgitation velocity, reduced RV free-wall motion.

Lower limb venous duplex ultrasound No evidence of deep venous thrombosis. These findings support the hypothesis of "primary in-situ pulmonary thrombosis", not embolic migration from

peripheral veins.

Contrast-enhanced CT pulmonary angiography (CTPA): Acute segmental pulmonary embolism of the right pulmonary artery. Areas of pulmonary infarction involving the right lower lobe. No pleural effusion was noted. Mediastinum and heart size remained normal.

The patient was diagnosed with: Primary: Acute pulmonary embolism (I26). Complication: Hypoxaemia and pleuritic pain associated with pulmonary infarct

He was initiated on therapeutic anticoagulation with Heparine 5000 ED 4 time a day, followed by transition to Rivaroxaban. Additional therapies included levofloxacin 100 ml, sildinafil, trombopol. By discharge, the patient was clinically stable, able to ambulate without dyspnoea, and was discharged with a plan for continued outpatient anticoagulation therapy, cardiology and pulmonology follow-up, and lifestyle modification.

DISCUSSION

This case describes a young male patient who presented with acute pleuritic chest pain, dyspnoea, and elevated D-dimer, ultimately diagnosed as acute pulmonary embolism (PE) with radiologically confirmed pulmonary infarction. Although pulmonary embolism is a common cardiovascular emergency, its presentation in otherwise healthy individuals without typical predisposing risk factors requires careful evaluation. In this patient, the absence of prior thrombotic events, immobility, recent surgery, malignancy, or hereditary thrombophilia raises the question of transient environmental or physiologic triggers.

Low-voltage electrical injury and acute stress—factors present in this patient's recent history—may contribute to a hypercoagulable state through vasoconstriction, hemoconcentration, and endothelial dysfunction. Several studies have also suggested that inflammatory activation and increased sympathetic tone may transiently elevate the risk of venous thrombosis even in individuals without chronic disease [7]. While the patient lacked deep venous thrombosis (DVT) on duplex ultrasound, approximately 20–30% of PEs occur without demonstrable lower-extremity clot burden, either due to complete embolization or clot origin in non-leg venous beds, such as pelvic or small muscular veins [8].

Radiologically, the presence of wedge-shaped peripheral opacities and pleuritic pain strongly supported pulmonary infarction, a finding seen in up to one-third of segmental or subsegmental PEs. His preserved cardiac biomarkers and normal echocardiographic parameters ruled out right ventricular strain and classified the patient as low-intermediate risk PE, consistent with his stable hemodynamic presentation [9].

The case also highlights the importance of integrating clinical suspicion with imaging modalities. While chest radiography is frequently nonspecific, CT pulmonary angiography (CTPA) remains the gold standard for diagnosis, allowing precise localization of thrombi and evaluation of infarction, parenchymal involvement, and alternative diagnoses [10]. The absence of infectious, cardiac, or structural pulmonary pathology further strengthened the diagnosis.

Therapeutically, early initiation of anticoagulation was essential to prevent clot propagation and reduce mortality. The patient responded well to low-molecular-weight heparin followed by direct oral anticoagulant (DOAC) therapy, consistent with current guideline recommendations. Notably, no bleeding complications were observed, and the patient demonstrated rapid symptomatic improvement.

This case contributes to regional clinical literature by illustrating that PE can occur in relatively young, active individuals without classical risk factors. The presentation also emphasizes the need for clinicians to maintain a high index of suspicion in patients with persistent chest pain and dyspnoea, especially when initial cardiac evaluation is unrevealing. Understanding subtle triggers and appreciating the spectrum of PE manifestations can help reduce diagnostic delays and improve patient outcomes.

CONCLUSION

This case demonstrates that low-voltage electrical shock can precipitate acute lung injury and pulmonary artery thrombosis via endothelial disruption, inflammatory activation, and microthrombotic mechanisms. Clinicians should maintain high suspicion for pulmonary complications in patients presenting with respiratory symptoms after electrical exposure. Early CT imaging and timely anticoagulation substantially improve outcomes.

Further research is needed to better characterize the incidence and progression of pulmonary complications following low-voltage electrical trauma.

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