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CASE REPORT
Coronary intervention

Acute inferior ST-elevation myocardial infarction mimicked by direct lightning strike: a case report

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Background Direct lightning strikes are rare, and multiple organ systems can be involved. Prognosis is dependent on the severity of the injury. Severe myocardial injury associated with transient electrocardiogram changes, which have been previously described, is a hazardous complication.

Case summary A 35-year-old man with no known past medical history presented unresponsive following a direct lightning strike while sitting in a portable toilet. High-quality cardiopulmonary resuscitation was started in the field, with return of spontaneous circulation (ROSC) after 1 h. Following ROSC, he received volume resuscitation and was maintained on multiple vasopressors. Electrocardiogram showed significant ST-elevations in inferior leads with elevated troponin I, consistent with inferior ST-elevation myocardial infarction. Labs revealed lactic acidosis, hyperkalaemia, acute kidney, and liver injury. Due to concern for plaque rupture, coronary angiography was performed and revealed no obstructive coronary artery disease. Vasopressor support and volume resuscitation were continued for extensive burns covering greater than 30% body surface area. The patient became progressively hypotensive, eventually precipitating pulseless electrical activity arrest. Emergent labs were notable for severe acidaemia. Despite aggressive interventions, he expired due to severe multi-organ failure.

Discussion Direct lightning injuries are rare with serious potential complications. Myocardial damage, either from direct electrical insult or from induced coronary vasospasm, can lead to multi-organ system failure.

Keywords Lightning strike • Lightning injuries • Direct lightning • Indirect lightning • Inferior STEMI • Lightning • Case report

ESC curriculum 3.2 Acute coronary syndrome • 7.2 Post-cardiac arrest • 7.3 Critically ill cardiac patient • 3.4 Coronary angiography

Learning points

(1) Lightning injuries occur through either direct or indirect strikes.
(2) Both types of strikes can result in serious cardiovascular complications ranging from benign electrocardiogram changes to death.
(3) Multiple mechanisms have been implicated to explain these cardiovascular manifestations including induction of coronary artery spasm, catecholamine-mediated effects, direct thermal damage, ischaemia secondary to arrhythmia, and coronary artery ischaemia as part of a generalized vascular injury.

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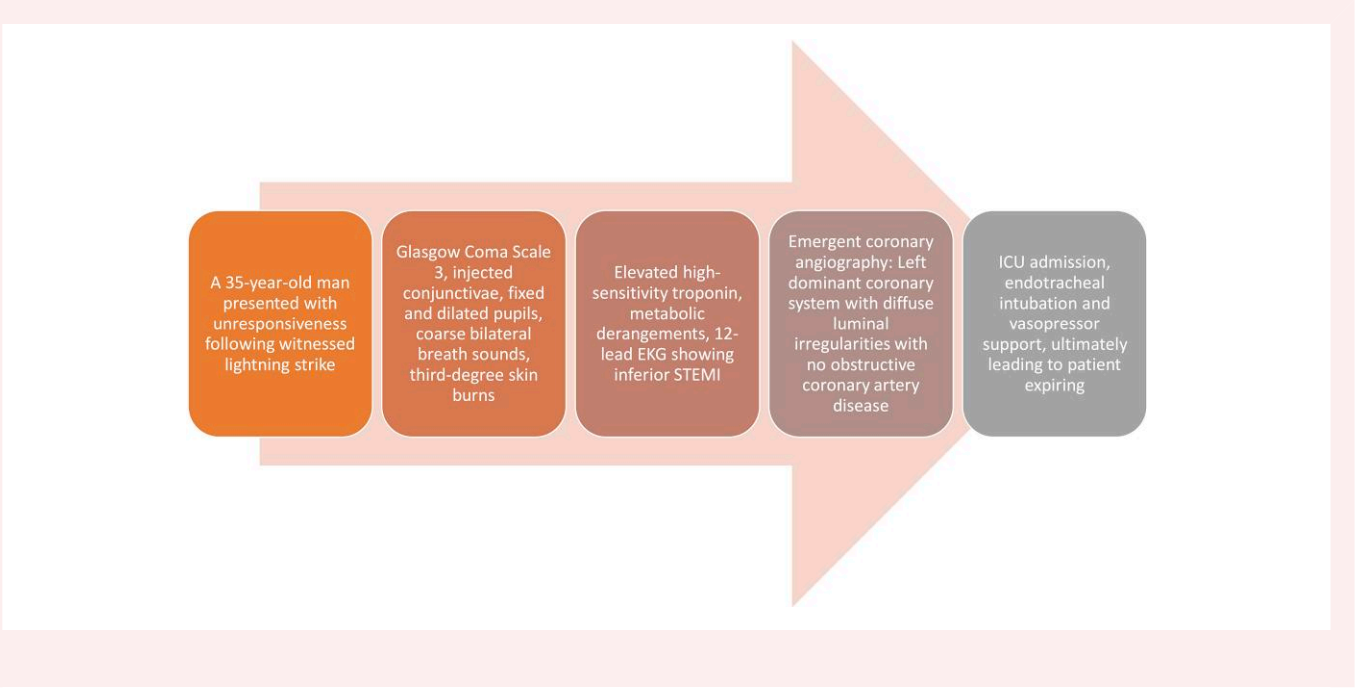
Introduction

Lightning strikes occur after the breakdown of excess positive and negative charges within clouds. Rarely, these lightning strikes make uninterrupted connection with a person on the ground, described as direct strikes. It is estimated in the USA that there are approximately 400 lightning injuries and 40 deaths annually.[1] The electrical current from lightning transfers through either direct or indirect strikes. Direct strikes occur when current passes over the surface body. Indirect strikes occur when a person comes into contact with an electrified area or the ground. Multiple organ systems may be involved with lightning strikes and the prognosis is dependent on the severity of the injury. Cardiac effects include arrhythmias, myocardial infarction, myocardial contusion, pericardial disease, aortic dissection, ventricular failure, and myocardial stunning.[2] We present an interesting case of electrocardiographic changes mimicking an ST-elevation myocardial infarction (STEMI) after a direct lightning strike.

Summary figure



Case presentation

A 35-year-old man presented unresponsive following a direct lightning strike while sitting in a portable toilet. He had no known past medical history. Emergency medical services were called and found the patient in asystole. A Lund University Cardiopulmonary Assist System device was placed. Advanced cardiac life support was continued as he was transported into the emergency department (ED) where the return of spontaneous circulation (ROSC) was achieved after 54 min.

He was noted to have a Glasgow Coma Scale score of 3 and was endotracheally intubated in the field. While in the ED, he was tachycardic (114 beats per minute), tachypneic (17 breaths per minute), low normotensive (92/54 mmHg), and saturating at 87% while intubated. Physical exam was notable for injected conjunctiva, fixed and dilated pupils at 8 mm bilaterally, and bilateral coarse breath sounds. Skin exam was notable for third-degree skin burns over the sternum with seared hairs and Lichtenberg figures across the abdomen and third-degree skin burning and blistering to the posterior aspect of the patient's left forearm and the posterior aspect of his left leg (*Figure 1*).

Initial laboratory diagnostics revealed elevated high-sensitivity troponin I (1590 ng/L; reference range: 0–53 ng/L), normal B-type natriuretic peptide (12.3 pg/mL; reference range 0.0–99.0 pg/mL), prolonged prothrombin time (21.8 s; reference range: 11.8–14.6 s), elevated international normalized ratio (1.9; reference range: 0.8–1.2), hypernatraemia (148 mmol/L; reference range: 137–145 mmol/L), hyperkalaemia (6.0 mmol/L; reference range: 3.5–5.1 mmol/L), hyperchloraemia (114 mmol/L; reference range: 98–107 mmol/L), low bicarbonate (18 mmol/L; reference range: 22–30 mmol/L), elevated creatinine (1.90 mg/dL; reference range: 0.66–1.50 mg/dL), elevated glucose (297 mg/dL; reference range: 65–140 mg/dL), elevated transaminases (aspartate aminotransferase 249 units/L; reference range: 0–33 units/L, alanine aminotransferase 227 units/L; reference range: 10–49 units/L), elevated creatinine kinase (266 units/L; reference range: 46–171 units/L), and elevated lactic acid (>15.5 mmol/L; reference range: 0.7–2.0 mmol/L). Venous blood gas was remarkable for pH < 6.8 (reference range: 7.32–7.42) and hypercarbia (>100 mmHg; reference range: 41–51 mmHg).

A plain film radiograph of the chest did not reveal any acute cardiopulmonary processes. Post-ROSC 12-lead electrocardiogram (ECG) demonstrated inferior ST-elevation myocardial infarction (*Figure 2*). Interventional cardiology was consulted and immediate left heart catheterization (LHC) was recommended, due to the elevated risk of thrombus following significant electrical injury. Prior to LHC, the patient was given a loading dose of aspirin and clopidogrel. Left heart catheterization demonstrated a left dominant coronary system with diffuse luminal irregularities with no obstructive coronary artery disease (*Figure 3*).

The patient was admitted to the intensive care unit for further management. He received volume resuscitation for extensive burns covering greater than 30% body surface area. He required multiple vasopressors due to persistent hypotension. During his hospital course, he became progressively hypotensive despite maximum vasopressor support, which eventually precipitated pulseless electrical activity arrest. High-quality cardiopulmonary resuscitation was started. Emergent laboratory diagnostics demonstrated worsening metabolic acidosis on arterial blood gas with pH

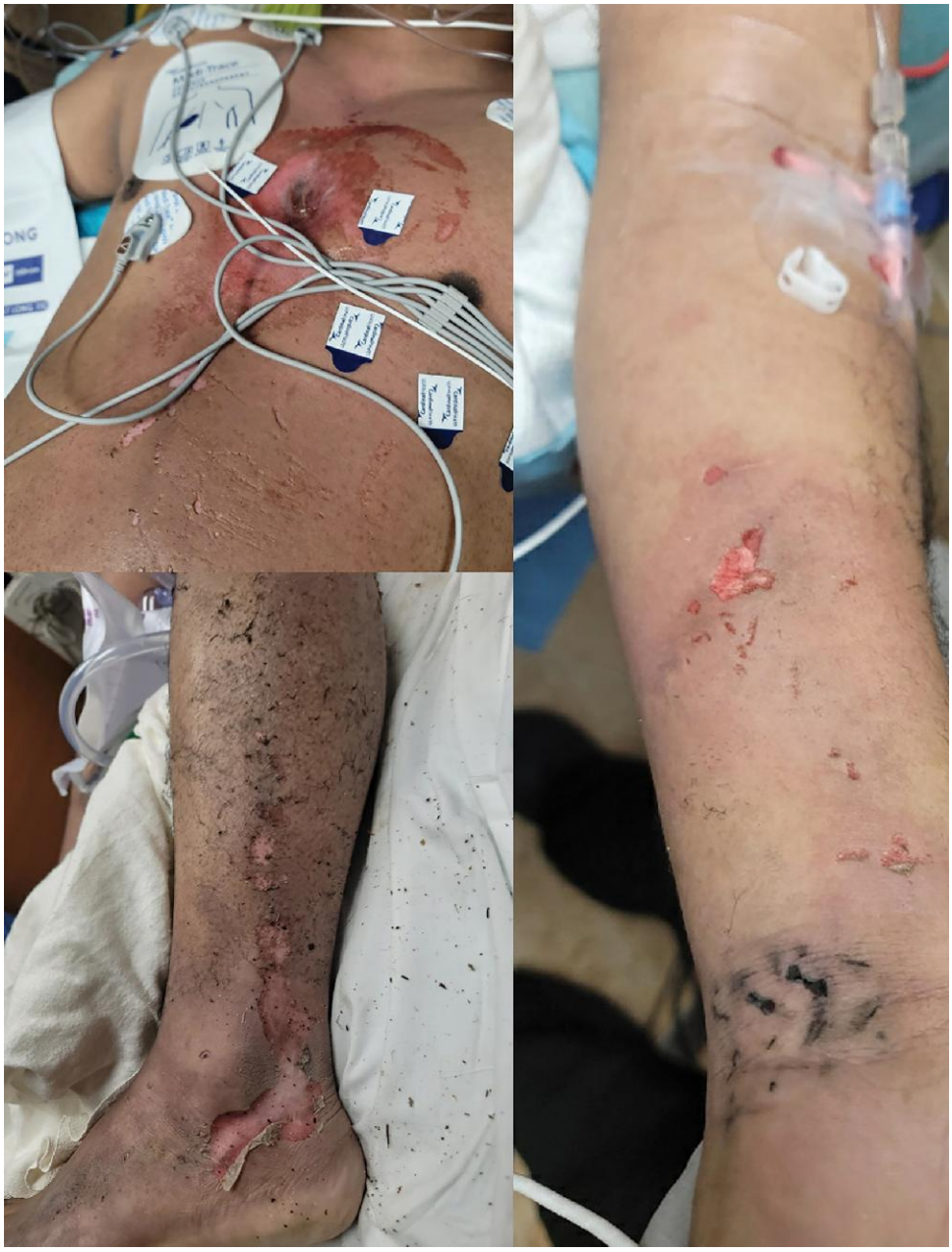


Figure 1 Third-degree burns on the anterior chest wall and upper and lower extremities.

6.96 (reference range: 7.35–7.45), low bicarbonate level (11.4 mmol/L; reference range: 21.0–28.0 mmol/L), and markedly elevated high-sensitivity troponin I beyond the maximum measurable value (>125 000 ng/L; reference range: 0–53 ng/L). Despite aggressive interventions, he remained haemodynamically unstable. Following goals of care discussions with his surrogate decision makers, the decision to proceed with comfort care was made, and the patient ultimately passed away.

Discussion

Lightning strikes have a greater incidence in areas with higher occurrences of thunderstorms. In the USA, most incidents occur between summer and early fall. It is important to note that unlike electrical injuries, lightning delivers a massive unidirectional impulse of current.[3,4] Several mechanisms are involved in lightning-induced injuries, which include the electrical current as it passes through body tissues, burns from conversion of electrical to thermal energy, and mechanical trauma from shockwave, direct strikes, and flying debris. Most major organ systems are involved with lightning strikes.

Lightning strikes, either direct or indirect, may have cardiac presentations ranging from arrhythmias, myocardial infarction, myocardial contusion, pericardial disease, aortic dissection, ventricular failure, and myocardial stunning. Dundon *et al.*[5] described the first report of a young woman who developed echocardiographic and angiographic evidence of Takotsubo cardiomyopathy and acute cardiogenic shock following a lightning strike. Our patient presented with ECG changes with STEMI in the inferior leads after a direct lightning strike.

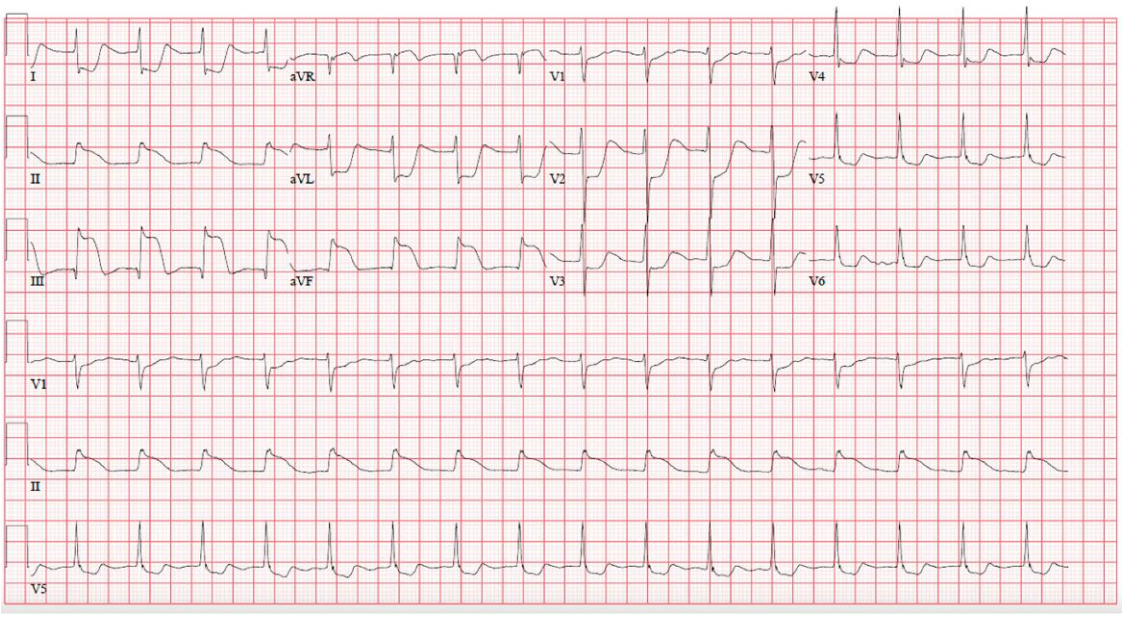


Figure 2 Twelve-lead electrocardiogram showing inferior ST-elevation myocardial infarction.

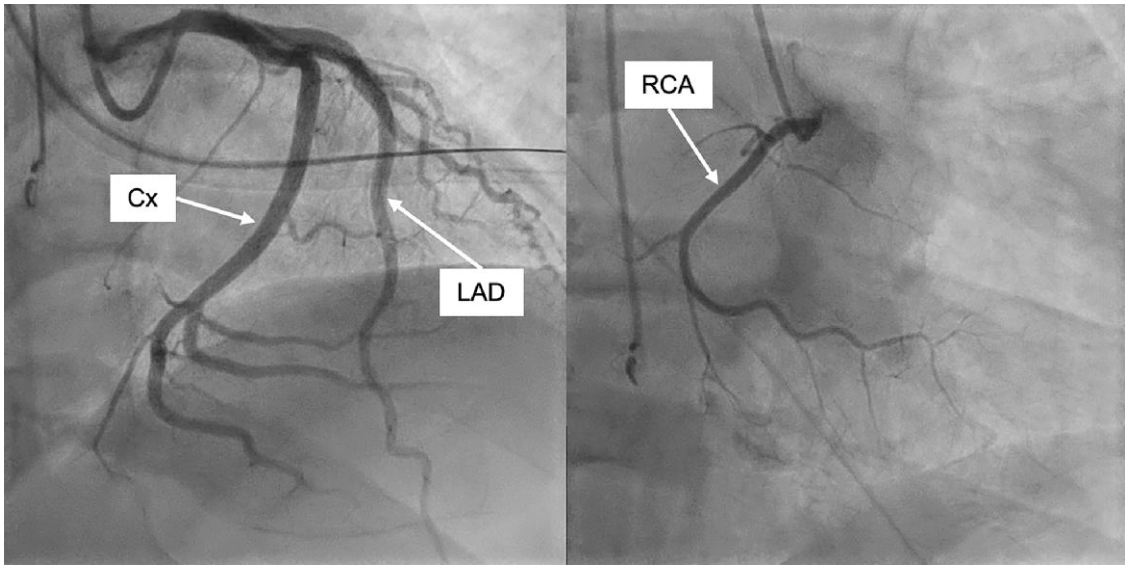


Figure 3 Left heart catheterization revealing luminal irregularities of the left anterior descending, circumflex, and right coronary arteries, with no obstructive coronary artery disease. Cx, circumflex; LAD, left anterior descending; RCA, right coronary artery.

Saglam *et al.*[4] and Aydin *et al.*[6] presented patients with similar ECG changes following direct and indirect lightning strikes, respectively. Other ECG changes that may be seen are QT prolongation, atrial fibrillation, and ventricular arrhythmias.[2,3]

Cardiovascular manifestations may be explained by different mechanisms: coronary artery spasm, catecholamine-mediated effects, direct thermal damage, ischaemia secondary to arrhythmia, and coronary artery ischaemia.[3] Sudden death is also commonly seen with direct strikes due to simultaneous cardiac and respiratory arrest. It is proposed that the asystolic arrest is due to an immediate and simultaneous depolarization of all myocardial cells.[1] Coronary artery vasospasm plays the underlying role in the reduction of coronary blood flow leading to myocardial infarction. However, some cases have shown evidence of thrombotic occlusions with endothelial damage and coagulation necrosis on histology.[2] Atherosclerotic processes that are common in typical presenting acute coronary syndrome do not play a major role in myocardial infarction due to lightning strike.[6] Our patient was thought to be at risk for thrombus occlusion and was immediately sent to the catheterization lab once stabilized. A case-based decision was made against continuing dual antiplatelet therapy and anticoagulation based on coronary angiography findings

of non-obstructive coronary artery disease. In agreement with previous reports, our patient did not have obstructive coronary artery disease.

Management of lightning-induced injuries is individualized to patient presentation and organ system involvement. Importantly for cardiovascular manifestations post-lightning strike, patients do not require mainstay therapies of acute coronary syndrome since myocardial necrosis is not due to atherosclerotic cardiovascular disease. Patients do not require anticoagulation upon admission despite ECG changes and elevated cardiac biomarkers. Percutaneous coronary intervention and antithrombotic agents may be beneficial if there is a high index of suspicion for thrombotic occlusion. Given the chance of a lightning strike is 1 in 10 000 in a lifetime, it is difficult to create evidence-based therapy and determine who would benefit from cardiac catheterization. Patients require hospitalization and close monitoring if suspected direct strike, loss of consciousness, focal neurological complaint, angina, dyspnoea, major trauma, and cranial or significant burns.[1] Patients would also benefit from discharge follow-up to track ECG changes such as QT prolongation.[2,3]

Lead author biography

Shaikh Iqbal is a resident physician at the MedStar Health Internal Medicine Residency Program in Baltimore, Maryland, USA. He graduated with his DO degree from Kansas City University College of Osteopathic Medicine. He is interested in pursuing a fellowship in Cardiovascular Disease.

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Consent: The patient reported in this case is deceased. Despite the best efforts of the authors, they have been unable to contact the patient's next of kin to obtain consent for publication. Every effort has been made to anonymize the case. This situation has been discussed with the editors.

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Data availability

No new data were generated or analysed in support of this research.

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