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CASUISTIC PAPER

ST-segment elevation in anterior leads secondary to electric shock – a diagnostic dilemma

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ABSTRACT

Introduction and aim. Electrical injuries can be life-threatening and prompt interventions can save lives. Cardiac complications like arrhythmias and sudden cardiac death are common after electric shock. Certain ECG abnormalities can persist after successfully reviving the patient which can mimic ST-Elevation occlusive myocardial infarction. This case report aims to inform the treating emergency physicians about this rare association of ST-Elevation in anterior leads after electric shock.

Description of the case. After obtaining proper consent from the patient, we describe here an interesting case of a 19-year-old boy who was presented to the emergency room with cardiac arrest after sustaining electrical injury. The patient was revived after cardiopulmonary resuscitation; ECG, as well as echocardiographic findings, were consistent with ST-elevation myocardial infarction of the anterior wall. A diagnostic dilemma was there between occlusive and non-occlusive causes of this condition. A coronary angiogram and conservative management of the patient helped in decision making and he was discharged with a Glasgow coma scale of 15/15 after recovery.

Conclusion. ST-Elevations in ECG can occur after electric shock injury and their cause is rarely due to occlusion of the coronaries. Hence thrombolysis in such cases is rarely needed and supportive management is required.

Keywords. cardiac arrest, electrical injury, non-occlusive MI

Introduction

Electrical injuries are very heterogeneous depending on multiple factors and their presentations can be varied ranging from small skin burns to extensive injuries of internal organs, which could be life-threatening.[1] Cardiovascular effects of electrical injuries can be arrhythmias or myocardial injuries. Arrhythmias are the most common cardiac complications of electrical injury but ECG pattern of myocardial infarction can also be present and lead to diagnostic challenges in the emergency room.[2]

Aim

Here we discuss a case of a 19-year-old male who was brought to the emergency room with cardiac arrest after sustaining electrical injuries and revived with post-return of spontaneous circulation (ROSC) ECG showing ST-segment elevation, a sign that could point to the diagnosis of occlusive myocardial infarction. This case report highlights the importance of the fact that most of the myocardial ischemic changes after electrocution injuries are related to vasospasm instead of thrombotic occlusion of the coronaries. Even ST-Elevations in the anterior lead in ECG, though rare, can

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be present without occlusive Myocardial infarction in this group of patients.

Description of the case

We report a case of a 19-year-old male, a resident of Jodhpur city in India, brought to the emergency room in an unresponsive state for 10 minutes following contact with an electric current at the workplace with a history of falling from a height of 5 feet. The patient was a handicraft factory worker and accidentally got shocked while fixing the power supply of a drill machine. The electric supply of the factory receives AC 220 Volt and 50 Hertz. There was no prior history of any comorbid conditions like diabetes, hypertension, ischemic heart disease, epilepsy etc. On examination, the carotid pulse was not palpable so code blue was activated and he was immediately shifted to the resuscitation bay. Cardiopulmonary resuscitation (CPR) was started according to advanced cardiac life support (ACLS) protocol. The initial rhythm on the monitor was ventricular fibrillation so defibrillation was done with 200 joules of energy. Defibrillation was repeated 2 times and an injection of amiodarone 300 mg intravenous was also given. After 2 cycles of CPR, ROSC was achieved and the airway was secured with a 7.5 mm endotracheal tube. ABG was suggestive of lactic acidosis with PH of 7.18, PCO_2 44 mmHg, PO_2 58 mmHg, Na^+ 132 mmol/L, K^+ 5.1 mmol/L, HCO_3 19 mmol/L and lactate levels were 6 mmol/L (Normal value 0.8–1.2 mmol/L) (Table 1). Post-ROSC vitals were stable with a blood pressure of 120/70 mmHg and a heart rate of 70/minute, regular. His GCS remained low, that is E1VTM1 with Bilateral pupils mid-dilated and non-reactive. On further examination, a wound mark was present on the left hand which was the entry wound, and the left foot was the exit wound (Fig. 1). Post-ROSC ECG taken after 30 minutes, showed ST segment elevation in leads V2 to V6,1, AVL (Fig. 2), and troponin I was 1.13 ng/mL (Normal is 0–0.03 ng/mL). The troponin I test was done by using a point-of-care Nanochecker machine, with 4-in-1 immunoassay-based kits for quantitative analysis of cardiac biomarkers. Bedside 2-dimensional Echocardiography (2D Echo) showed hypokinesia in the territory of the left anterior descending artery and E-FAST was negative. Blood gas showed Primary metabolic acidosis appropriately compensated by respiratory alkalosis. The patient was given loading doses of dual antiplatelets suspecting the possibility of acute ST-elevation myocardial infarction following electrical injury. Given a poor Glasgow Coma Scale (GCS) of 7/15 post-ROSC, a non-contrast CT head was done which was normal. The patient was shifted to the ICU where serial ECGs were obtained which showed persistently the same changes. Serial Trop levels were also done, which were decreasing and became normal within 3 days. Cardiologist consultation was taken for ECG changes and given no regional wall motion abnormality on 2D Echocardiography, persistently stable vitals, and improvement in the general condition of the patient, conservative management was advised. The patient was managed conservatively in the ICU for the next 3 days, after which he gained consciousness and was extubated. A coronary angiogram was done to rule out occlusive myocardial infarction but it was normal. The patient was monitored for complications and later was discharged with a cerebral performance score of 1 and GCS E4V5M6. Repeat ECG on day 5 of ICU admission showed biphasic t waves in lead V3, and V4, and the 2-dimensional echocardiography repeated was also normal.

Fig. 1. Entry wound in the left hand (black arrow) and exit wound in left foot (white arrow)

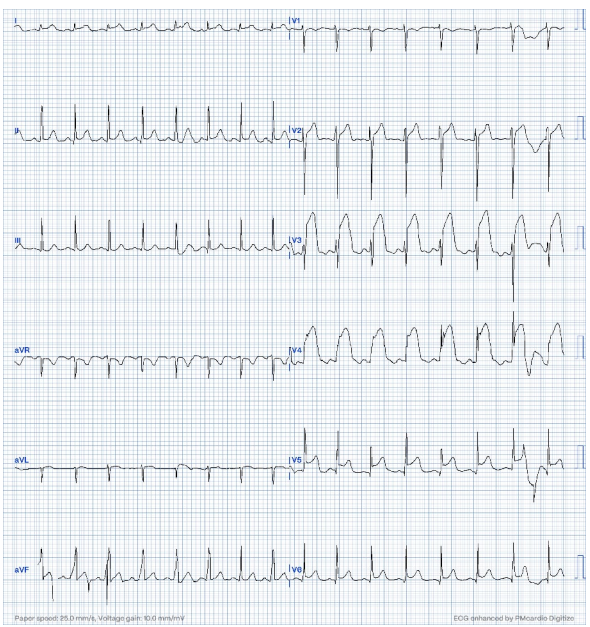

Fig. 2. ECG showing ST segment elevations in leads V2-V6 and I, aVL

Table 1. Investigations done in the Emergency Room

Parameter	Value
Total leukocyte count	$10\times10^3/mm^3$
Hemoglobin	12 g/dL
Platelets	$200\times10^3/\mu l$
Urea	39 mg/dL
Serum creatinine	1.28 mg/dL
$Na^+/K^+/Cl^-$	134/4.5/103 meq/L
SGOT/SGPT	113/194 IU/L
PT/INR	12/1.01
CT brain + C spine	Normal
Troponin I	1.13 ng/L (Positive)
CK NAC	3011 U/L (Elevated)

Discussion

The major mechanisms of electricity-induced injury are electrical injuries causing direct tissue damage, altering cell membrane potential, conversion of electrical energy into thermal energy, causing tissue destruction, and mechanical injury resulting from falls.[3] Factors that determine the nature and severity of electrical trauma include voltage, resistance to current flow, type of current (direct or alternating), duration of contact with the current source, current path through the body, and the magnitude of energy delivered.[4] When the chest is situated along the path that connects the entrance and exit points, the heart is often affected and can lead to complications like arrhythmias and myocardial tissue injuries. In most of the cases, patients who had myocardial infarction after electric shock have been reported to have normal coronary arteries and in the case of ST elevation MI; the aetiology is considered to be vasospasm.[5] Due to the proximity of the right coronary artery to the chest wall, it is the most frequently involved artery in electrical injuries. Therefore, ST elevations in inferior leads are more frequently observed.[6] But in our case, the ST elevations were present in the anterior leads which is reported rarely and caused the diagnostic challenge. Multiple mechanisms are proposed for these ECG changes which include coronary artery spasm, Direct thrombogenic effect on coronary arteries, direct thermal effect on myocardium, and ischemia secondary to arrhythmia-induced hypotension.[7] Due to the persistent elevations after resuscitation in anterior leads of ECG and regional wall motion abnormality in the same territory, the patient was administered a loading dose of aspirin and clopidogrel through the nasogastric tube. No heparin or thrombolysis was given considering the diagnostic uncertainty between occlusive and non-occlusive myocardial infarction. The patient improved with just ventilator support and conservative management. Therefore, this case signifies the importance of constant patient monitoring, including his vitals, ECG changes, echocardiographic abnormalities, and serial troponin levels after resuscitation from post-electrocution cardiac arrest in deciding the further course of management and the need for coronary interventions in these patients.

Conclusion

Myocardial infarctions after electrical injuries are rare and most commonly occur due to non-occlusive causes. Most of the ST segment changes occur in the inferior leads but involvement of anterior territory is also a possibility as described here in this case. Thrombolysis in these conditions is rarely necessary and only coronary angiogram can be used to differentiate between occlusive and non-occlusive conditions. Primarily managing the airway, breathing, circulation, and disability with good resuscitation efforts in these cases are the mainstay of treatment.

Declarations

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Author contributions

Conceptualization, A.K.R. and A.K.M.; Methodology, A.S.; Software, R.G.S.; Validation, A.K.R., M.S.R. and A.S.; Formal Analysis, A.K.R..; Investigation, A.K.M.; Resources, R.G.S.; Data Curation, A.K.R.; Writing – Original Draft Preparation, A.K.R.; Writing – Review & Editing, A.S.; Visualization, R.G.S; Supervision, M.S.R.

Conflicts of interest

All authors declare that they have no conflicts of interest.

Data availability

The data that support the findings of this study are available on request from the corresponding author.

Ethics approval

Written informed consent for publication was obtained from the patient. We complied with the policy of the journal on ethical consent.

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