CASE REPORT

Myocardial infarction due to lightning strike

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Abstract

Cardiac events due to lightning strike and their severity vary according to the strength of the electric current and the duration of exposure. The electrophysiological effects of lightning on the heart can result in ventricular fibrillation, asystole, QT prolongation, supraventricular tachycardia, and non-specific ST-T wave changes. In this report, a case of a patient who suffered myocardial infarction due to lightning strike is presented, which is a rare complication

Keywords: Myocardial infarction, Lightning strike.

Introduction

The incidence of lightning strikes worldwide is estimated to be 0.09-0.12/100,000 people. The mortality rate related with lightning strikes worldwide is 0.2-1.7 deaths/million/year.[1] The electric current occurring after a lightning strike usually passes from outside the body quickly. Therefore, burns with an entry and exit wound, and deep burns resulting in rhabdomyolysis, myoglobinuria and internal organ damage occur less frequently.[2,3] A lightning strike causes myocardial depolarisation and cardiac asystole by direct current.[4] Respiratory arrest may occur as a result of inhibition of the medullary respiratory centre in the brain and tetanic contraction of chest wall muscles and diaphragm.[4] Various cardiac anomalies such as dysrhythmias, ST-T wave changes and QT prolongation have been reported in cases surviving a lightning strike. Myocardial infarction (MI) may also occur as a result of conduction disturbances and endothelial damage to the coronary arteries.[5] In this report, a case of acute MI caused by lightning strike is presented together with a review of the related literature.

Case Report

A 44-year-old male was exposed to lightning strike while working in a field in June 2012 and was evaluated by the emergency team upon reaching the scene of the accident. Cardiopulmonary resuscitation (CPR) was performed on the patient, who was diagnosed with cardiopulmonary arrest. The patient responded to CPR and was admitted to the emergency department (ED). He was unconscious, and the physical examination revealed a Glasgow Coma Scale (GCS) score of 4, with respiratory rate of 25 breaths per min, temperature of 36.9°C, arterial blood pressure 180/110 mmHg, and pulse rate 150 beats per min; he was intubated and had spontaneous breathing. Second-degree (10%) burn from the arcus costalis bilaterally to the level of the umbilicus and on the front of both legs was observed. Examination of other systems were normal. The patient's medical history was unremarkable. In the 12-lead electrocardiogram (ECG), sinus rhythm, ST segment elevation in lead-II, lead-III and aVF leads and ST segment depression in V1-2 leads were detected (Figure). The patient's various laboratory tests results (Table) including cardiac enzymes were significantly elevated. In the echocardiographic (ECHO) evaluation, ejection fraction was 60%, the left ventricular wall motion, pericardium and aortic root were detected to be normal, and the inferior wall motion was found to be hypokinetic. Chest radiography and abdominal ultrasonography were normal. The patient underwent primary invasive coronary angiography on emergency

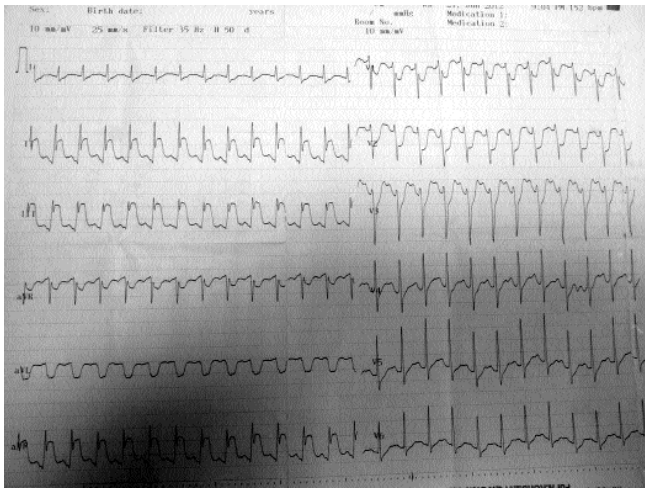

Figure: ST segment elevation in D2, D3 and aVF leads and ST segment.

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Table: Laboratory results of the patient.

	Value	Normal range
Haemoglobin		
19,3 g/dl	11-18	
Leukocytes	25,570 mm3	4000-11000
Thrombocytes	84000 mm3	150000-450000
Glucose	110 mg/dl,	65-95
BUN	55 mg/dl,	0-71
Creatinine	1,94 mg/dl,	07-1.3
Potassium	4,74 mmol/L,	3.5-5.1
Sodium	139mmol/L	136-145
ALT	222 U/L	0-41
AST	342 U/L,	0-31
LDH	1345 U/L,	240-480
Troponin I	> 50 ng/ml,	0-0.1
Myoglobin	>1000 ng/L	
CK-MB	300 U/L	0-24

BUN: Blood urea nitrogen. ALT: Alanine aminotransferase. AST: Aspartate aminotransferase. LDH: Lactate dehydrogenase. CK-MB: Creatine kinase-myocardial band.

basis due to ST segment elevation on ECG and implementation of CPR. Coronary arteries were normal in the right and left coronary angiography system. Fluid therapy, monitoring, and burn dressing were applied, and the patient was admitted to the intensive care unit (ICU). On the 2nd day of follow-up, negative T wave was observed in lead-II, lead-III and aVF leads. Hypoxic-ischaemic encephalopathy developed in the patient, who died in the ICU on the 5th day.

Discussion

Injuries and death occurring due to lightning strike are reported frequently.[6] Early recognition of lightning injury cases and management of complications may have better outcomes for these patients.[7] Those at the greatest risk include the age group of 10-29 years, and those working in occupations such as agriculture and construction.[6] In our case, the patient was a farmer working in the field.

Lightning strike results in death in 20%-30% of the injuries and the most common cause of death is cardiopulmonary arrest.[1,4] Cardiac events following lightning strikes and the severity of these events vary according to the electric current strength and the duration of exposure.[8] Four electrical mechanisms of lightning injury have been described: direct strike, contact, sideflash, and ground current. Mechanical injury may occur if the person falls or is thrown by muscular contraction.[2] Multiple mechanisms such as coronary artery spasm, catecholamine-mediated impact, direct thermal damage, myocardial ischaemia secondary to arrhythmia, and coronary artery ischaemia as part of a common vascular injury have been suggested to explain the cardiovascular events of lightning injury.[7]

Cardiac rhythm defect can be affected both directly and indirectly as a result of lightning strike.[7] In patients exposed to lightning strike, different ECG and clinical findings are seen,[8] but MI is rare.[8] There are no typical ECHO findings based on the electrophysiological effects of lightning on the heart, but ventricular fibrillation, ST elevation, asystole, QT prolongation, supraventricular tachycardia, and non-specific ST and T wave changes may be observed.[8] All of these may be related to changes in re-polarisation, which could be a direct result of cellular injury. These signs highlight the significance of ECG monitoring of such cases.[7]

In one case, emergency defibrillation was performed on a patient developing ventricular fibrillation after lightning injury.[8] Later, asystole developed, and ST elevation in inferior leads and ST segment depression in anterior leads were observed in the ECG taken after CPR. The coronary arteries were reported as normal in the emergency coronary angiography of that case. Another case has been reported in literature with acute inferior MI in the ECG after defibrillation,[9] while a different case has been reported with ST elevation due to direct lightning injuries.[5] Similarly, inferior MI was detected in the ECG in the case under review. The coronary arteries were observed to be normal on coronary angiography. It was thought that since our patient was exposed to a direct lightning current, MI occurred due to coronary artery spasm, and hypoxia occurred after respiratory arrest and thermal injury.

In a very short period after a lightning strike, high-voltage direct current causes disorder in the victim's general condition by causing ventricular fibrillation, asystole, or by damage to the respiratory centre. In an earlier case, ventricular fibrillation developed as a result of direct lightning strike injury and returned to normal sinus rhythm with electrical defibrillation within the first 15 minutes.[10] The authors concluded that the timely intervention in their case was the most important factor determining survival.[10] In the present case, cardiopulmonary arrest was detected when the medical teams reached the scene of the accident, and approximately 20 minutes had passed prior to the intervention. It is possible that the initial event was a ventricular arrhythmia only. As there was no CPR for 20 min, subsequent MI could have

resulted due to hypoxic insult in addition to the possibility of direct myocardial trauma and vasospasm.

Conclusion

Myocardial infarction can develop after lightning injury due to several mechanisms. Assessment of these patients should include history and physical examination, 12-lead ECG and value of baseline troponins. Early intervention influences morbidity and mortality.

References

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