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Case Report

Cardiac troponin I: A potent biomarker for myocardial damage assessment following high voltage electric burn

Arindam Bose, Chandra B. Chhabra[1], Shobha Chamania[2], Nanda Hemvani, Dhananjay S. Chitnis

Departments of Pathology, [1]Departments of Cardiology and [2]Burn Surgery, Choithram Hospital and Research Centre, Indore, Madhya Pradesh, India

Address for correspondence: Dr. Dhananjay S. Chitnis, Department of Pathology, Choithram Hospital and Research Centre, Manikbagh Road, Indore, Madhya Pradesh, India. E-mail: ds_chitnis@rediffmail.com

ABSTRACT

Myocardial infarction (MI) following high voltage electric burn is very rare, and its pathogenesis remains controversial. Electrical burns represent only 4% of all burns. Hence, clinical managements have taken a slow pace in developing. The recent guidelines laid down by the cardiology societies include cardiac troponin I (cTnI) as the gold standard marker for the assessment of myocardial damage assessment. Two patients were admitted to our hospital at the different time with the same kind of high voltage electric burn. Both patients had complained with chest discomfort during admission, and cardiac parameter assessment was done for both the patients. cTnI was also measured for both patients, and marked increase in the values was seen within 5 h of onset of myocardial damage and got into normal range within 72 h. Myocardial damage following electric burn needs to be suspected and assessed as early as possible. Hence, cTnI should be the valuable tool to detect the severity of myocardial damage incurred in the electric burn cases.

KEY WORDS

Cardiac troponin I; high voltage electric burn; myocardial necrosis

INTRODUCTION

In electric shock injuries, the true extent of injury is unlikely to be recognised immediately compared to the extent of cutaneous burns. Cardiac dysfunction needs to be suspected in the early stage of electric burn. A variety of cardiac and noncardiac abnormalities have been described following low voltage (≤1000 volts) alternate current household (220–240 volts) electric shock.[1] Among cardiac abnormalities, most lethal is sudden death due to asystole or ventricular fibrillation. An electrocardiogram (ECG) may show tachycardia, ST-segment changes, arrhythmia, rarely bundle branch block or complete heart block. Infrequently acute infarction is also noted.[2] Myocardial infarction (MI) after electrical injury is not a common event, and its

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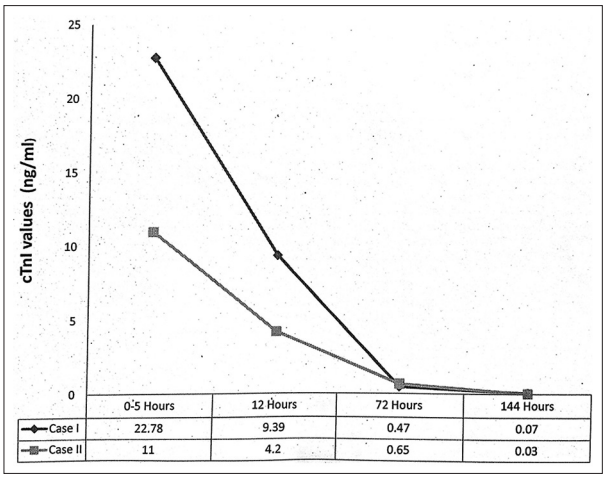
pathogenesis remains controversial. Although electrical currents can damage the wall of the coronary arteries, they may have a direct thrombogenic effect.[3] ECG is the most valuable indicator to detect MI, but ECG changes may not be obvious in all cases of acute MI (AMI). Hence, the Joint European Cardiology/American College of Cardiology Committee included cardiac troponin I (cTnI) as a marker for acute cardiac syndrome even in the absence of ECG findings. Reports on cTnI levels following thermal injuries are scanty, but mild elevations in cTnI levels have been documented.[4] These elevations of cTnI did not correlate with overt cardiac morbidity or mortality among thermal burn cases and point out that subtle degree of cardiac injury was present following severe thermal burn in spite of hyperdynamic cardiac function during resuscitation. There is virtually no data on cTnI levels for cases of electric burn. We present here two cases of electric burn admitted to our hospital with the finding on ECG, Echocardiogram and cTnI.

CASE REPORTS

Case 1

A 32-year-old man with 21% total body surface area (TBSA) burn by high voltage electric (≥1000 volts) shock, admitted to the Burns Intensive Care Unit on 10th September 2014. The burn area involved was chest and mainly right side of the body. The patient was asymptomatic from anginal pain but ECG at the time of admission showed 'J' point elevation. He was not shifted to Intensive Cardiac Care Unit (ICCU), but cardiac monitoring was done in Burns Intensive Care Unit. After 5 h of hospital admission, first cTnI levels were assessed, and the result showed marked elevation (22.78 ng/ml) using ARCHITECT i1000SR, Abbott system based on Microparticle Enzyme Immunoassay. cTnI measurement was repeated at 12 h, 72 h and 144 h [Figure 1]. The echocardiographic findings showed the normal-sized left ventricle with fair systolic function and without any regional wall motion abnormality (RWMA) at rest. The patient had left ventricular ejection fraction (LVEF) approximately 60%. Cardiac valves and pericardium were normal. In echocardiograph, no vegetative clots or intracardiac mass was seen. The patient had tachycardia (≥112 bpm), but with normal blood pressure (100/70 mmHg). The patient had no acute coronary syndrome history, and he was nondiabetic and nonhypertensive. The patient underwent 10% TBSA tangential excision and split skin autografting. During this surgery, no cardiac decompensation was felt by the patient.



	0·5 Hours	12 Hours	72 Hours	144 Hours
Case I	22.78	9.39	0.47	0.07
Case II	11	4.2	0.65	0.03

Figure 1: Graphical representation of the cardiac troponin I levels in two different high voltage electric burn cases

Case 2

The second case was a 25-year-old young man who was admitted to the Burns Intensive Care Unit on 6th February 2015 with 23% TBSA electric burn (≥1000 volts electric shock) injury. Accidentally, the hand of the patient, which held an iron rod, touched a live high tension electric wire (≥1000 volt). The patient was brought to the hospital within an hour of the accident. He had noticeable burns in the anterior trunk portion (13%) and had a tightening sensation on the chest region, but no anginal pain was felt by the patient. ECG findings during hospitalisation showed ST elevation along with 'T' wave abnormalities. ECG was analysed, and the patient was shifted in ICCU for supervision of cardiac conditions. To evaluate the myocardial damage, cTnI level was assessed after 5 h of hospitalisation. The elevated cTnI blood level (11.0 ng/ml) in serum was reported. A serial time point study of cTnI was conducted also in this case and which shows the cTnI level in serum dropped to 0.65 ng/ml after 72 h of hospitalisation. During ICCU monitoring, there was no noticeable cardiovascular discomfort or complications reported and ECG was also normal. Echocardiograph findings enumerated that left ventricle was in normal size with 73% of LVEF without RWMA rest. The cardiac valves and pericardium were normal. This patient had also tachycardia and like the patient in Case 1. This patient also was nondiabetic and nonhypertensive. The burn wounds were excised, and autograph was done on post-burn day five. Right-hand wound which was holding the iron rod had a full thickness burn wound on the grip side. This was later covered with autograph. Wounds healed well and were discharged with usual instructions for rehabilitation.

The value of the cTnI reached its peak within first 5 h of the onset of myocardial damage due to high voltage electric shock in both the cases and came down to the normal in circulation after 72 h. The recovery in both the cases was uneventful without any cardiac problems during follow-up of more than 4 weeks.

DISCUSSION

Serum cTnI level is a well-established marker for cardiac muscle necrosis in association with acute cardiac syndrome, especially during MI. cTnI is being used as a diagnostic and prognostic marker for MI. Reports on cTnI levels among burn cases are very scanty, but low level rise for cTnI reported following thermal burn.[3] The muscle necrosis including that of cardiac muscle is expected to be high following electric burns and was reflected with high cTnI level in the present cases. The Joint European Cardiology/American College of Cardiology Committee and the National Academy of Clinical Biochemistry proposed cTnI as the appropriate markers for a definitive AMI diagnosis.[5,6]

Injuries caused by exposure to 1000 volts or greater are defined as high-tension electrical burns.[7,8] High-tension wires can carry up to 100,000 volts.[8] Both the patients presented here had sustained high-tension electrical burns with associated secondary flame burns, which is documented to be a common accompaniment of high-tension electric burns.[7]

The electrical burns are not so common in the burn cases admitted to major burn centres. Mortality rates are significant with these types of injuries, as reported in the literature to be as high as 59%;[9] the most common cause being secondary to an acute arrhythmia at the time of the burn injury.[10]

The cTnI level was measured at various time points for both the cases. The cTnI level was at the peak at the time of admission. The cTnI level dropped to <1 ng/ml in 72 h in both the cases. The overt cardiac morbidity is not commonly seen following thermal burn. The available data appear scanty for electric burn, but a case of electric burn following electric shock through diathermy equipment has been reported to have had MI.[11] A silent anterior wall MI was discovered from incidental ECG in a 55-year-old male following high-tension electric current.[12] The initial ECG in both the present cases showed 'J' point elevation without symptomatic angina, but with a marked rise in cTnI. Data on cTnI levels are virtually nonexistent for electric burn cases, and it is felt that one should look for myocardial damage among them and more information needs to be generated on the cTnI levels among them.

This appears to be the first report to show a marked increase of cTnI levels following electric burn. Myocardial damage, acute or silent needs to be suspected following electric burn and cTnI levels should be assessed within 6 h of the accident, to reveal myocardial necrosis.

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Conflicts of interest

There are no conflicts of interest.

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