A Rare Complication of Electric Shock: Myocardial Infarction

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ABSTRACT

Myocardial infarction is one potential outcome after an electric shock although it is seen relatively rarely and its pathogenesis remains controversial. Coronary arteriography is the most helpful investigation in assessing the physiopathology of the rare event. These patients merit a careful scrutiny due to an increased death rate because of cardiopulmonary arrest. Here, we reported a man with inferior myocardial infarction following electrical shock. Although he had frankly normal coronary arteries by coronary angiography, myocardial infarction was objectively evident by cardiac enzymes, electrocardiography and echocardiography. Amputation was performed on his left wrist from the elbow and fasciotomy was performed on his right wrist due to compartment syndrome. Electrocardiography changes returned to normal level within 6th hours of electric shock. He was discharged in good health after stabilization.

Keywords: Electric shock, electrocardiography, coronary angiography, high voltage

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ÖZET


Anahtar Kelimeler: Elektrik çarpması, elektrokardiyografi, koroner anjiyografi, yüksek voltaj


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INTRODUCTION
Electrically shocked patients typically are young and male, and electrical shock to adults mostly occurs in an occupational setting (1, 2). Widespread and conflicting data is available in terms of myocardial injury after an electric shock. Despite the controversy, some mechanisms have been proposed to account for myocardial injury after electrical shock. These are: coronary artery spasm; direct thrombogenic effect on coronary arteries; direct thermal effect on the myocardium; ischemia secondary to arrhythmia-induced hypotension; coronary artery ischemia as part of a generalized vascular injury and direct contusion during cardiopulmonary resuscitation with subsequent coronary artery injury (3-7). Also, hypoxic conditions after respiratory arrest might possibly contribute to myocardial injury.

CASE REPORT
A 43-year-old construction worker was admitted to our emergency department following accidental contact with a high voltage (>1000 volt) electricity power line. Loss of consciousness was experienced for a short time, and the Glasgow Coma Scale was 15. There were no pre-existing medical problems. On admission, blood pressure, heart rate and O₂ saturation were 135/95 mm Hg, 95 beats per minute and 98% respectively. Cardiovascular and respiratory examinations were unremarkable. The entry points of the electric current were present in both palms and exit points were in the soles. The left radial and ulnar artery flows could not be sensed with physical examination nor detected with Doppler ultrasound. The pulses were sensed on the right wrist, but the finger activities were limited and painful. A total of 15% of third (both wrists with palms and both soles) and 25% of second degree burns (anterior right and left arms, a half of left back, anterior right leg, posterior left leg) were present.

Electrocardiograph (ECG) revealed sinus rhythm with >1 mm ST segment elevation in inferior leads (D2-D3-aVF) and ST depression in anterior leads (V1-V2-V3), suggesting evolving inferior myocardial infarction (Fig. 1). Transthoracic echocardiography (TTE) demonstrated normal global systolic function (ejection fraction=60%) and hypokinesia of the left ventricular

Figure 1. Admission ECG (one hour post electric shock)
inferior wall. Serum UREA, Creatinine, creatine kinase, CK-MB, AST, ALT, and troponin I levels were markedly high [51 (19-44 mg/dL), 1.85 (0.72-1.25 mg/dL), 90130 (25-200 U/L), 2177 (0-25 U/L), 473 (5-34 U/L) and 0.366 (0-0.01 ng/mL) respectively]. After stabilization, he underwent coronary angiography. Both left and right coronary arteries were free of any occlusive lesions. However, left ventriculography complied with ECG findings in detecting hypokinesia of the inferior wall. Anti ischemic (Acetylsalicylic acid, beta-blocker and angiotensin converting enzyme inhibitor) therapies were started. The right wrist was amputated from the elbow and fasciotomy was performed to the left wrist due to a compartment syndrome. At the end of the 6th hours following the event, the changes in ECG had returned to normal levels, no arrhythmia developed elsewhere (Fig. 2). After one month there was no sequel of myocardial injury and systolic function was normal by TTE. He was discharged from hospital after treatment for electrical burns.

DISCUSSION

Electrical shocks can cause cardiac abnormalities, ranging from dysrhythmias to myocardial infarction. These usually occur at the time of shock; however, some studies suggest that they may develop in the post-shock period (8-10). The primary cause of death from electrical shock is cardiopulmonary arrest. Myocardial infarction is a potential, though rare, consequence of electric shock (11, 12). It was reported that only five cases of myocardial infarction following high voltage injury were detected in four series out of a total of 344 patients (9).

In essence, coronary angiography is the first choice for the detection of the underlying mechanism of myocardial injury after electrical shock. Lesions are categorized as obstructive or non-obstructive. Demonstration of normal coronary arteries evokes a non-obstructive mechanism. Coronary artery spasm (3), a direct thermal effect on myocardium (3), ischemia secondary to arrhythmia-induced hypotension (5), direct contusion during cardiopulmonary resuscitation with subsequent coronary artery injury (7) and hypoxic conditions after respiratory arrest (8), could all have contributed to myocardial injury in the current case. Celebi et al. (12) reported similar causes for their case also. Abnormal ECG findings of patients following an electrical shock were detected in Arrowsmith et al. (9) and Al et al. (13) studies in approximately 3%, and 67.9% respectively. In some other studies ECG abnormalities range from 14% to 54% (9, 10, 14), the highest percentages arising from a series of high voltage injuries. Non-specific ST segment changes and sinus tachycardia are the most commonly reported ECG findings (13, 15, 16). Other ECG findings include QT prolongation, bundle branch block, atrial and ventricular fibrillation, atrial and ventricular premature contractions (12, 15, 16). Given the non-transmural nature of necrosis, ST segment elevation rarely occurs due to electrical
Shock (17). ST elevation of inferior derivations is, however, observed more often, as in our case. As we know from the literature, inferior myocardial infarction is the most common injury caused by electric shock. This seemingly higher predominance is explained by the right coronary artery’s close proximity to the chest surface during its course, which makes it vulnerable to electrical shock (6). These notable ECG changes can normalize and tend to be totally reversible in long-term survivors (15). In contrast, Celebi et al. (12) reported that abnormalities of ECG in their patient had persisted even after one year. Herein, ECG abnormalities in our case recovered within 24 hours. Both CK and CK-MB can markedly increase, owing to concomitant skeletal muscle injury and cardiopulmonary resuscitation. It is unclear to what extent skeletal muscle injury contributes to this increase. This can cause a spurious diagnosis of myocardial infarction after electrical shock. Therefore, CK and CK-MB are suggested to be less specific markers for myocardial injury (12, 18). The abovementioned condition may have tarnished the value of CK and CK-MB in identifying myocardial infarction after electrical shock. Elevation of troponin I is more likely to increase in the event of myocardial injury rather than skeletal muscle injury. Seen in this light, specific troponin I should unquestionably be the preferred cardiac enzyme investigated (12). Assessment of left ventricular systolic function after electrical shock is clinically relevant. Echocardiography can be beneficial in determining the presence of myocardial injury and its severity after electrical shock. It may reveal diffuse hypokinesia of the myocardium, as well as regional hypokinesia. Echocardiographic findings may markedly improve in the follow-up (11, 19). Additionally, echocardiography may provide confirmation of the induced myocardial injury, as happened in the present case and also in the Celebi et al. case.

Celebi et al. (12) did not detect a history or evidence of cardiopulmonary resuscitation, hypoxic condition or arrhythmia-induced hypotension. Therefore, coronary artery spasm and direct thermal effect seemed the likeliest explanations for their case. The results of our case supported that idea. The optimal management of myocardial injury after electrical shock may be challenging since there is no consensus as to the best management of ST segment elevation myocardial infarction after electrical shock. Contraindications for fibrinolysis such as prolonged resuscitation, trauma or hematoma may unfortunately accompany electrical injury. So, coronary angiography with subsequent percutaneous coronary intervention may be better than fibrinolytic treatment as an initial reperfusion strategy (12). On the other hand, myocardial injury might occur due to a non-occlusive mechanism, as in the present and Celebi et al. cases (12). Therefore, there may be no need for either medical or mechanical reperfusion. Thus, coronary angiography is of central importance and may clearly guide the therapy. Co-existing tachyarrhythmia can mostly be handled by anti-arrhythmic drugs (19). Angiotensin converting enzyme inhibitors are as reliably effective as angiotensin II receptor blockers at protecting against remodel-