

A Rare Complication of Electric Shock: Myocardial Infarction

Elektrik Çarpmasının Nadir Komplikasyonu: Miyokart Enfarktüsü

**Behçet Al¹, Pınar Yarbil¹, Hasan Orhan Özer², Selve Aslan², Cuma Yıldırım¹,
Vedat Davutoğlu²**

¹Department of Emergency, Faculty of Medicine, Gaziantep University, Gaziantep
²Department of Cardiology, Faculty of Medicine, Gaziantep University, Gaziantep

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

Received: 30.06.2011

Accepted: 28.07.2011

ÖZET

Patogenezi tartışmalı olmasına ve nadiren görülmesine rağmen, miyokart enfarktüsü bir elektrik çarpmasından sonra potansiyel bir sonuçtur. Bu nadir olayın patofizyolojinin değerlendirilmesinde koroner arteriografi en iyi yardımcı araştırma yöntemidir. Bu hastalar kardiyopulmoner arresten dolayı artan bölüm oranlarından dolayı ciddi bir bakımı hak ediyorlar. Burada elektrik çarpmasını takibe miyokart enfarktüsü gelişen bir vaka bildirdik. Koroner anjiyografisi tamamen normal olmakla beraber miyokart enfarktüsü kardiyak enzimler ve elektrokardiyografi ile objektif olarak kanıtlandı. Sol el bileği dirsekten itibaren ampute edildi ve sağ el bileğine kompartman sendromundan dolayı fasiyotomi uygulandı. Elektrik çarpmasının altıncı saatinde elektrokardiyografideki değişiklikler normale döndü. Hasta stabilize edildikten sonra şifa ile taburcu edildi.

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

Başvuru Tarihi: 30.06.2011

Kabul Tarihi: 28.07.2011

Corresponding to/Yazışma Adresi:

Behçet Al
Gaziantep Üniversitesi Tıp Fakültesi, Acil Tıp Anabilim Dalı,
Gaziantep, Türkiye
GSM: 0533 810 76 90
e-mail: behcetal@gmail.com

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

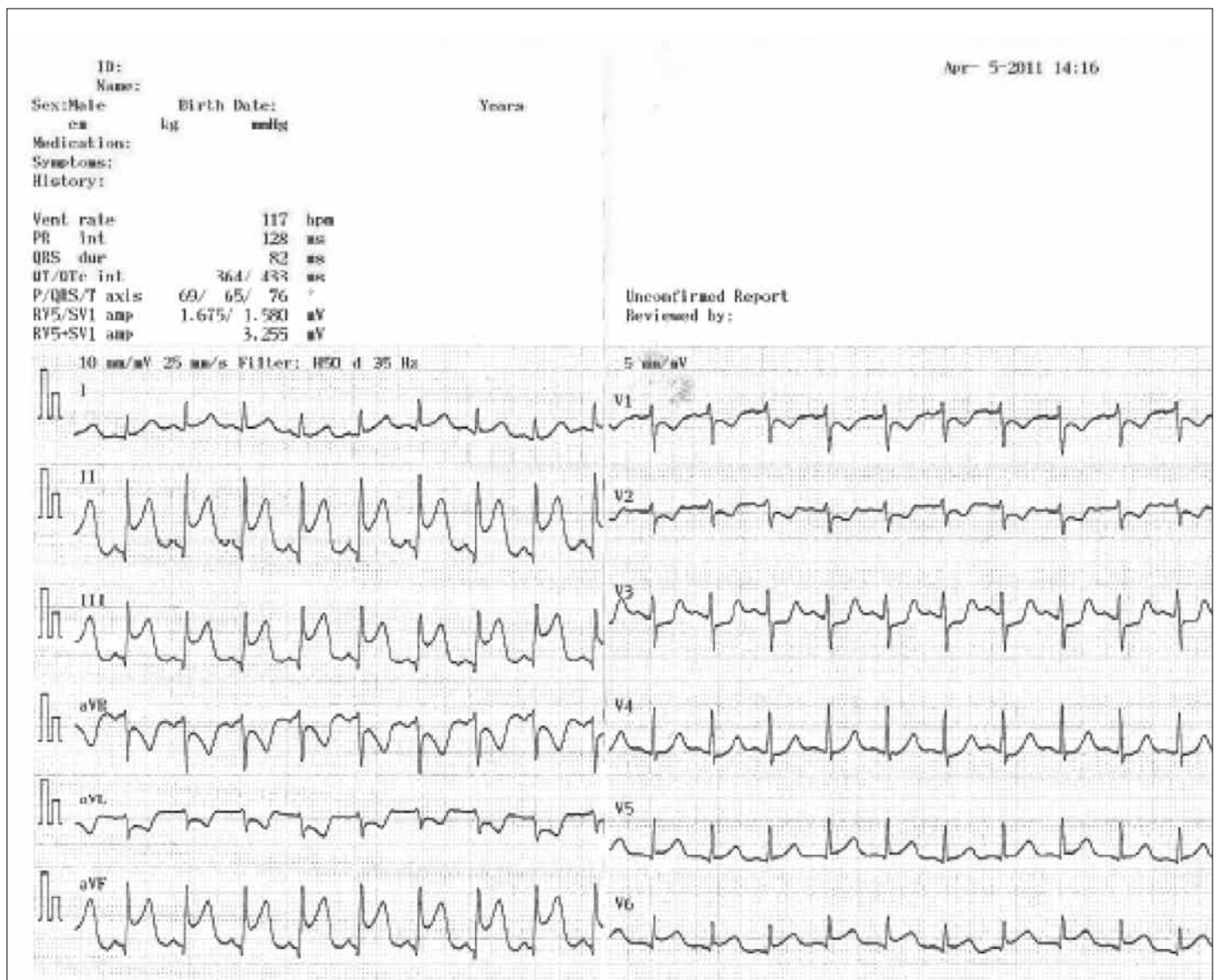


Figure 1. Admission ECG (one hour post electric shock)

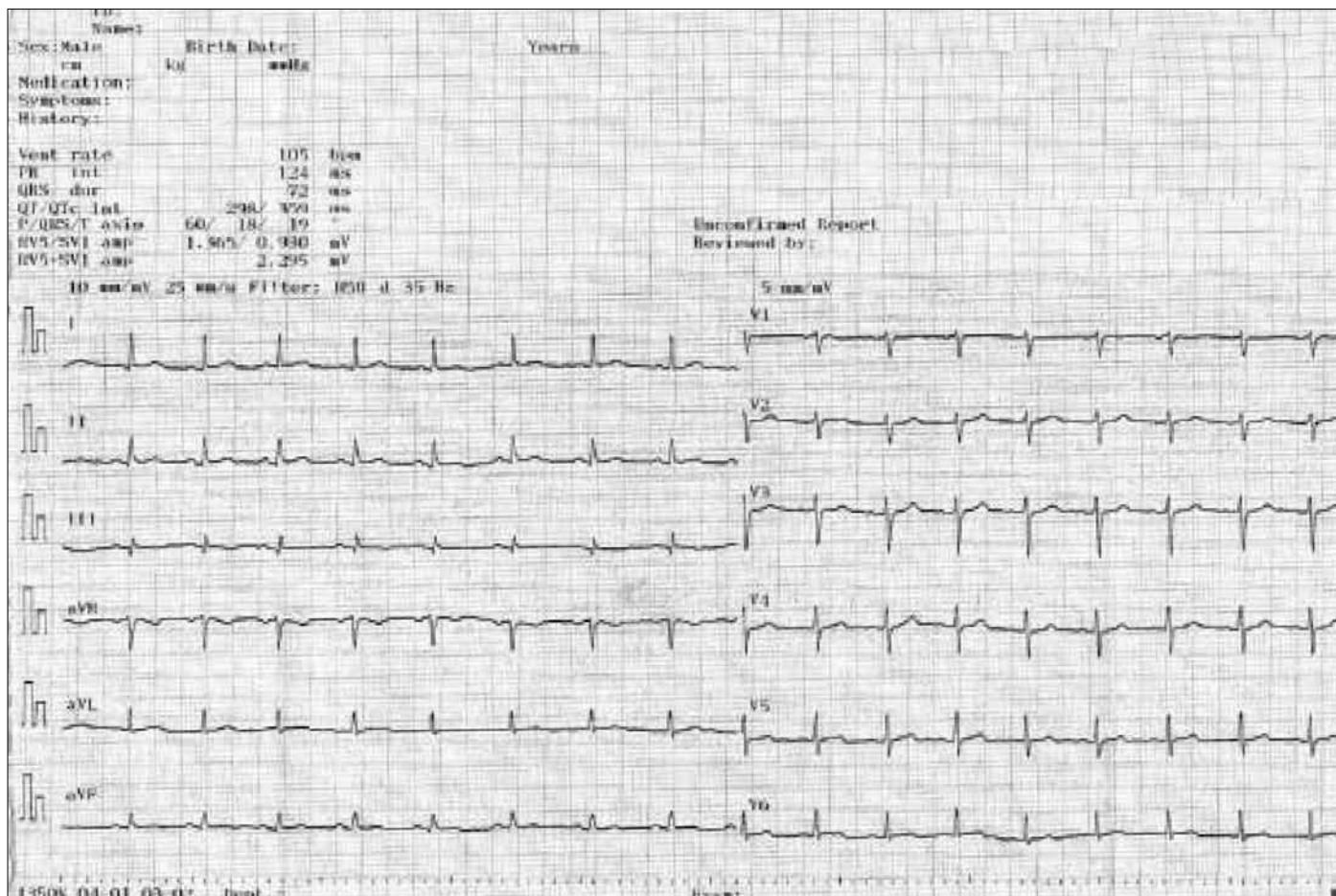


Figure 2. Control ECG (Six hours post electric shock)

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), 236 (3-55 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-

ing (19). Cardiac complications are managed similarly to other myocardial infarction causes and require follow-up evaluation (20). In addition to these, patients should be managed in exactly the same way as following trauma.

CONCLUSIONS

In conclusion, troponin I and echocardiography should be the primary considerations in order to detect myocardial injury after electrical shock. Despite the uncertainty of therapeutic options and lack of guidelines, coronary angiography can help determine whether myocardial injury is of occlusive or non-occlusive origin, thereby assisting a more tailored treatment. The patients with loss of consciousness, documented dysrhythmia in the field, and abnormal ECG on admission should ideally be observed closely during hospitalization and after discharge.

Acknowledgements

The authors do not report any conflict of interest regarding this work.

REFERENCES

1. Purdue GF, Hunt JL. Electrocardiographic monitoring after electrical injury: Necessity or luxury. *J Trauma*. 1986; 26: 166-7. [\[CrossRef\]](#)
2. Cooper MA. Electrical and lightning injuries. *Emerg Med Clin North Am*. 1984; 2: 489-501.
3. Xenopoulos N, Movahed A, Hudson P, Reeves WC. Myocardial injury in electrocution. *Am Heart J*. 1991; 122: 1481-4. [\[CrossRef\]](#)
4. Baubion N, Metzger JP, Heulin A, Grosdemouge A, De Vernejoul P, Vacheron A. Myocardial infarction caused by electric injury. Value of coronarography. *Ann Med Interne (Paris)*. 1985; 136: 659-62.
5. Oltman CL, Clark CB, Kane NL, Zhang Y, Gutterman DD, Dellsperger KC, et al. Coronary vascular dysfunction associated with direct current shock injury. *Basic Res Cardiol*. 2003; 98: 406-15. [\[CrossRef\]](#)
6. James TN, Riddick L, Embry JH. Cardiac abnormalities demonstrated postmortem in four cases of accidental electrocution and their potential significance relative to nonfatal electrical injuries of the heart. *Am Heart J*. 1990; 120: 143-57. [\[CrossRef\]](#)
7. Oliva PB, Breckinridge JC. Acute myocardial infarction with normal and near normal coronary arteries. Documentation with coronary arteriography within 12 1/2 hours of the onset of symptoms in two cases (three episodes). *Am J Cardiol*. 1977; 40: 1000-7. [\[CrossRef\]](#)
8. Koumbourlis AC. Electrical injuries. *Crit Care Med*. 2002; 30: 424-30. [\[CrossRef\]](#)
9. Arrowsmith J, Usgaocar RP, Dickson WA. Electrical injury and the frequency of cardiac complications. *Burns*. 1997; 23: 576-8. [\[CrossRef\]](#)
10. Solem L, Fischer RP, Strate RG. The natural history of electrical injury. *J Trauma*. 1977; 17: 487-92. [\[CrossRef\]](#)
11. Homma S, Gillam LD, Weyman AE. Echocardiographic observations in survivors of acute electrical injury. *Chest*. 1990; 97: 103-5. [\[CrossRef\]](#)
12. Celebi A, Gulel O, Cicekcioglu H, Gokaslan S, Kututcularoglu G, Ulusoy V. Myocardial Myocardial infarction after an electric shock: a rare complication. *Cardiol J*. 2009; 16: 362-4.
13. Al B, Aldemir M, Güloğlu C, Kara IH, Girgin S. Epidemiological characteristics of electrical injuries of patients applied to the emergency department. *Ulus Travma Acil Cerrahi Derg*. 2006; 12: 135-42.

14. Butler ED, Gant TD. Electrical injuries, with special reference to the upper extremities. A review of 182 cases. *Am J Surg.* 1977; 134: 95-101. [\[CrossRef\]](#)
15. Housinger TA, Green L, Shahangian S, Saffle JR, Warden GD. A prospective study of myocardial damage in electrical injuries. *J Trauma.* 1985; 25: 122-4. [\[CrossRef\]](#)
16. Carleton SC. Cardiac problems associated with electrical injury. *Cardiol Clin.* 1995; 13: 263-6.
17. Chandra NC, Siu CO, Munster AM. Clinical predictors of myocardial damage after high voltage electrical injury. *Crit Care Med.* 1990; 18: 293-7. [\[CrossRef\]](#)
18. Hammond J, Ward CG. Myocardial damage and electrical injuries: Significance of early elevation of CPK-MB isoenzymes. *South Med J.* 1986; 79: 414-6. [\[CrossRef\]](#)
19. McGill MP, Kamp TJ, Rahko PS. High-voltage injury resulting in permanent right heart dysfunction. *Chest.* 1999; 115: 586-7. [\[CrossRef\]](#)
20. Kirchmer JT Jr, Larson DL, Tyson KR. Cardiac rupture following electrical injury. *J Trauma.* 1977; 17: 389-91. [\[CrossRef\]](#)