

Delayed ST-Segment Elevation Due to Electrical Injury Mimicking Acute Myocardial Infarction

Akut Miyokard Enfarktüsünü Taklit Eden Elektrik Çarpması Sonrası Geç Gelişen ST Segment Elevasyonu

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Abstract

Electrical injury can cause cardiac problems and various electrocardiographic (ECG) changes. It has been suggested that an electrical current may permanently damage the cardiac conduction tissue and predispose to late dysrhythmia. Less serious transient ECG changes have also been described after electrical injury, such as ST segment elevation. Electrical injuries can cause delayed cardiac problems. In the emergency department, a patient who has chest pain and ECG changes should be investigated through his history about any previous electrical injury, so that we can differentiate myocardial infarction and other cardiac problems mimicking it. (*JAEM 2013; 12: 222-4*)

Key words: Electrical injury, ST elevation, electrocardiographic changes, chest pain, rhythm disturbances

Özet

Elektrik çarpması kardiyak problem ve birçok elektrokardiyografik değişikliğine neden olur. Elektrik akımının geçici olarak kardiyak ileti dokularına zarar verdiği ve geçici disritmilere neden olduğu tahmin ediliyor. Daha az ciddiyete sahip olan geçici ST segment elevasyonu elektrik çarpması sonrası gözlenebiliyor. Elektrik yaralanmaları gecikmiş kardiyak problemlere neden olabiliyor. Acil servise gelen göğüs ağrılı ve elektrokardiyografisinde ritm anormalliği olan hastaların hikayeleri geniş detaylı alınmalıdır. Böylelikle miyokardiyal enfarktüs ile onu taklit eden diğer kardiyak problemleri ayırt edebiliriz. (*JAEM 2013; 12: 222-4*)

Anahtar kelimeler: Elektrik çarpması, St elevasyonu, elektrokardiyografik değişiklikler, göğüs ağrısı, ritm bozukluğu

Introduction

Electrical injury can cause cardiac problems such as dysrhythmias, myocardial/valvular rupture, structural changes in coronary arteries, pericardial effusion, and various electrocardiographic (ECG) changes. It has been suggested that an electrical current may permanently damage the cardiac conduction tissue and predispose to late dysrhythmia (1). The heart in particular is liable to damage by electrical injury. Less serious transient ECG changes have also been described after electrical injury (2). In our report, a case of late onset of chest pain and ST elevation in ECG after electrical injury is presented.

Case Report

A 22-year-old patient suffering from chest pain was referred to our hospital. He had a history of exposure to 220 volt alternative current electricity with a wet wood stick 6 days earlier. During the following first days he had no cardiologic problem such as chest pain,

ECG changes and cardiac enzymes. His cardiac enzyme levels and ECG were totally normal. Six days later his chest pain began and his ECG changes appeared without cardiac enzyme changes.

On physical examination his appearance was healthy, with a normal body structure. His vital signs were as follows: pulse, 45 beats per minute; respiratory rate, 17 per minute; blood pressure, 120/80 mm Hg; O₂ saturation 98% in room air; body temperature, 36.5°C. His Glasgow coma scale was 15. Pupils were 4 mm reactive. His lungs had equal and good air entry without any pathological sounds. He was bradycardic with normal heart sounds and a regular rhythm; peripheral pulses were of good volume and with brisk capillary refill, and no pulse deficit was noted. In his left arm, hand and left foot there were burn scars (Figure 1a-c). Other systemic examinations were normal.

His complete blood count, chemistry, coagulation profile and cardiac enzymes were normal. His standard 12 lead ECG showed sinus bradycardia with 45 heart beat per minute, 2 mm ST segment elevation in D2 D3 and AVF (early repolarisation) (Figure 2), there was no hemodynamic instability. Echocardiographic assessment revealed



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a normal left ventricular size and function, no valvular pathology, and no wall motion abnormality.

Because of bradycardia, 1 mg atropine was injected, after that heart beat increased to 65. Patient was given a consultation in the cardiology department and he was hospitalized. After admission and follow-up in the cardiology intensive care unit, the patient's symptoms resolved one day later. After normalization of the ECG changes, the patient was discharged from the hospital with complete recovery.

Discussion

In our report, there was a delay between electrical injury and the late onset of ST segment elevation and chest pain. In the emergency department, typical chest pain and ST segment elevations in D2, D3 and AVF derivations are related with acute inferior myocardial infarction in high percentage. However, in our case there were no abnormalities in cardiac enzymes such as creatinin kinase MB, myoglobin and troponin I. There was no abnormality in echocardiography.

The heart is one of the most vulnerable organs to electricity. Various myocardial manifestations develop at the time of injury. These include asystole, ventricular fibrillation, which may cause immediate death, QT-prolongation, right bundle branch block, complete AV block, valvular or myocardial rupture, CK-MB elevations caused by myocardial injury, structural changes in the small coronary vessels, and pericardial effusion (3). In our report, the patient referred to our hospital with ECG problem such as ST segment elevations in D2, D3 and AVF derivations and typical chest pain six days after the injury.

Carleton recommended that all patients injured by electrical energy should be followed because of the risk of developing cardiac manifestations for at least one year, and any cardiac operation should be avoided during the first six months (4). There are many reports about late onset cardiac problems after electric injury such as in our case. We were interested in this case to find out why transient

ST elevation developed after alternative current electrical injury without myocardial injury. Transient ST-segment elevation without myocardial infarction immediately following direct current cardioversion is a clearly documented occurrence in some of cases. Cantor et al. (6) reported two cases of "intermittent" and transient ST-segment elevation after cardioversion. Transient ST segment elevation was reported by Chun et al. (5) after elective DC cardioversion. Van Gelder et al. (7) found post-shock ST elevations in 19% in a larger series of patients undergoing electrical cardioversion. Also, Lichtenberg et al. (8) demonstrated on ECG by ST segment elevation the early effects of lightning without myocardial injury. However, to our knowledge ST segment elevation after alternative current electric injury has not been reported previously. According to Chun et al. their patients showed that transient ST-segment elevation after DC cardioversion does not necessarily indicate myocardial injury. Similarly, myocardial infarction is not seen in our patient; but our patient was exposed to alternative current. We think that our patient also showed that transient ST-segment elevation after alternative current injury does not necessarily indicate myocardial injury.

There is little data regarding the possible mechanisms of ST-segment elevation following electrical cardioversion (9). Cantor et al. hypothesized this electrocardiographic observation that the electroshock induced a timing difference in the action potential between the epicardium and endocardium. This may be a possible mechanism for our case.

Conclusion

Electrical injuries can cause delayed cardiac problems. In emergency departments, such patients should have cardiac monitoring for a minimum of 24 hours, and followed by cardiologist after discharge. Also, in emergency departments, a patient who has chest pain and ECG changes should have his history investigated about previous electrical injury, so that we can differentiate myocardial infarction and other cardiac problems.



Figure 1 a-c. Burn scars of patient as an input in left arm and hand, and output in left foot

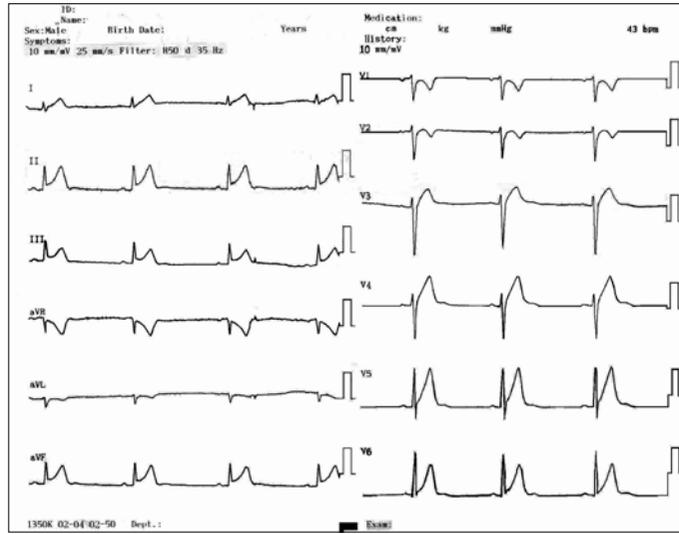


Figure 2. Electrocardiograph of a patient with 2 mm ST segment elevation D2, D3 and AVF and T-wave inversion in lead V1 and V2 with heart rate 43 beats/per minute

Conflict of Interest

No conflict of interest was declared by the authors.

Peer-review: Externally peer-reviewed.

Informed Consent: Written informed consent was obtained from patient who participated in this case.

Author Contributions

Concept - E.U., B.E.; Design - E.U., B.E.; Supervision - B.E.; Funding - E.U., B.E.; Materials - E.U., B.E.; Data Collection and/or Processing - E.A., B.E., M.Ö.; Analysis and/or Interpretation - E.U., B.E.; Literature Review - E.U., B.E.; Writer - E.U., B.E.; Critical Review - E.U., B.E.

Çıkar Çatışması

Yazarlar herhangi bir çıkar çatışması bildirmemişlerdir.

Hakem değerlendirmesi: Dış bağımsız.

Hasta Onamı: Yazılı hasta onamı bu olguya katılan hastalardan alınmıştır.

Yazar Katkıları

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