

Acute inferior myocardial infarction after electrical weapon exposure: case report and review of the literature

Elektrikli silah uygulaması ardından gelişen akut alt duvar miyokart enfarktüsü: Olgu sunumu ve literatüre bir değerlendirme

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Summary– The use of conducted electrical weapons (CEWs) by legal security forces and in civil society is rapidly increasing. While they are generally considered safe devices, and fatal complications are rare, it is possible to see a small number of complications. In the present case, we describe the detection of acute inferior myocardial infarction in a patient who experienced chest pain after being exposed to a CEW. In such cases, multiple factors should be considered, and the choice of treatment and follow-up should be decided accordingly.

Özet– Elektrikli silah kullanımı hem kanun güçleri çalışanları hem de sivil toplumda hızla artmaktadır. Genel olarak güvenli cihazlar olarak bilirse de minimal komplikasyonlar görülebilmektedir. Fakat ölümcül komplikasyonlar çok nadirdir. Bu yazıda, elektrikli silaha maruz kalmasıyla birlikte göğüs ağrısı başlayan hastada akut alt duvar miyokart enfarktüsü saptandı. Bu gibi durumlarda, birden fazla faktör göz önüne alınmalıdır, tedavi seçimi ve takip şeklimizi de ona göre belirlemeliyiz.

Conducted electrical weapons (CEWs) constitute a class of arms that aim to neutralize people by causing pain and continuous muscle contractions. While the experience is painful, proper use of the device is rarely associated with significant side effects. [1] CEWs are used either front-of-weapon and applied directly to the subject (drive stun), or with a pair of metal probes fired from the weapon (probe mode).

In the present case, we describe the detection of acute inferior MI in a patient who was admitted to the emergency service with chest pain after being exposed on the anterior thoracic wall to a CEW in probe mode, and the subsequent procedures performed.

CASE REPORT

A 37-year-old male patient with no prior cardiac anamnesis was in an altercation with the security personnel of a hotel, and collapsed after application of a CEW in probe mode to the anterior thoracic wall, with pain in

all parts of the body and spasms. The probes were removed at the scene of the incident. One of the injuries caused by the CEW dart was 1cm left

of the left midclavicular line on the sixth intercostal space, and the other was 2 cm right of the anterior axillary line near nipple level. While the patient was able to stand up with support, a crush-like pain in the thoracic cage persisted for approximately 30 seconds following CEW application. The patient was admitted to the emergency service due to the legal aspect of the incident, as well as the persistent chest pain. The patient's vital status was as follows: heart rate 109/minute, blood pressure 143/100 mm/Hg, respiratory rate 24 breaths/min, and body temperature 36.8°C. The patient was monitored, and electrocardiography showed elevated ST segment in D2, D3, AVF leads, and reciprocal ST depression in D1 and AVL leads

Abbreviations:

CAD	Coronary artery disease
CEWs	Conducted electrical weapons
CK-MB	Creatine kinase-MB
MI	Myocardial infarction

Received: September 21, 2014 Accepted: December 09, 2014

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(Figure 1). The patient was conscious, and anamnesis showed only chest pain, and revealed no known disease (e.g. diabetes mellitus, hypertension), and no use of medication, including narcotics. The patient did not have premature coronary artery disease (CAD) anamnesis in the family history. Physical examination showed no signs of traumatic injury or bleeding. The patient was transferred to coronary intensive care. The first laboratory tests showed normal glucose, electrolyte, urea, creatinine, hemogram levels, as well as normal pH, PaO₂, and PaCO₂ levels in the arterial blood gas. According to results we received afterwards, ethanol or cocaine were not detected in the blood. There was no possibility to study other potentially cardioactive substances in our emergency laboratory.

The patient was administered with acetylsalicylic acid, clopidogrel, isosorbide dinitrate, metoprolol, and diazepam (for sedation), and transferred to the nearest center for coronary angiography. Three hours elapsed between CEW application and coronary angiography. The chest pain had decreased by half at the beginning of angiography. Coronary angiography was normal, and complications such as arrhythmia, heart failure, and recurrent angina did not develop during

the patient's intensive care follow-up. The angina disappeared about 5 hours after CEW application. The patient recovered without any permanent finding in the ECG (Figure 1), while echocardiography showed minimal levels of inferior wall hypokinesia. During hospitalization, the patient's CK, CK-MB, and troponin I levels exceeded the normal values (Table 1). The patient was discharged with acetylsalicylic acid, beta blocker, and ACEI administration. Recurrent angina was not observed in the third month follow-up; ECG, echocardiography, and physical examination were normal.

DISCUSSION

Electrical injuries are frequently seen in work places, and among young men. Furthermore, increased use of CEWs leads to a certain level of concern. CEWs aim to neutralize the exposed subject by causing sudden pain and widespread muscle spasms. Localized skin problems are the most frequently associated with CEW use. CEWs are considered as a reason for death despite the lack of a clear-cut unifying pathophysiological hypothesis.^[2] Heart problems, which are a rare outcome of CEW application, are seen as a result of intervention in the anterior chest wall, as in our case.^[3]

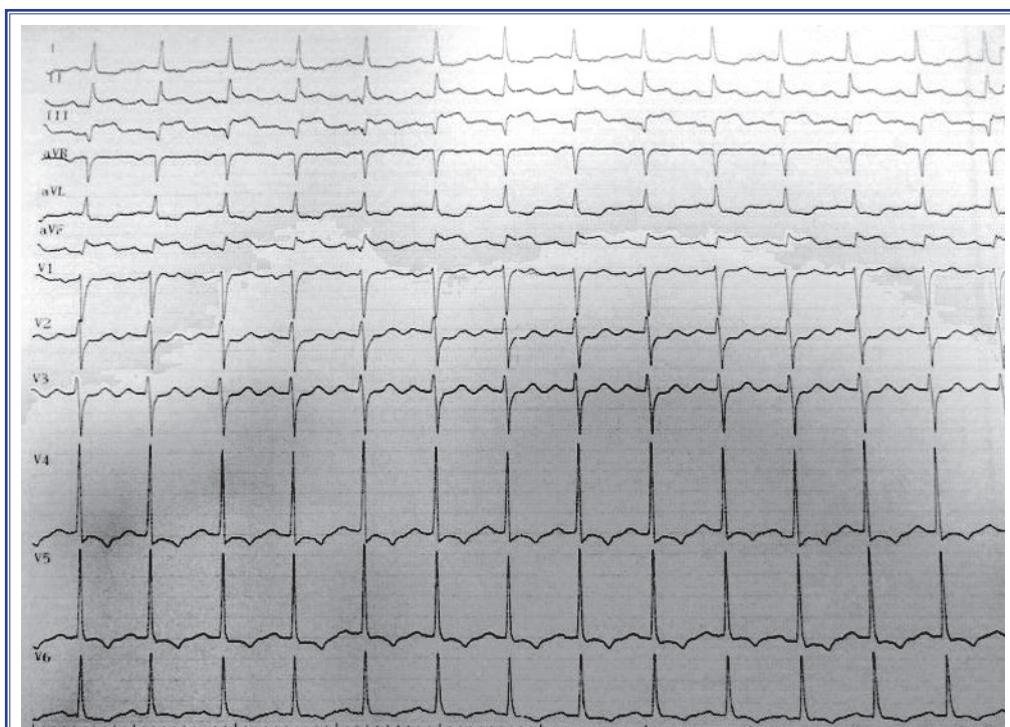


Figure 1. 12-lead electrocardiogram.

Table 1. The course of cardiac markers during hospitalization

	90 th min	12 th h	48 th h
Troponin I (ng/mL)	1.9	9	7
Creatine kinase-MB (U/L)	70	150	120
Creatine kinase (U/L)	620	1250	980
Aspartate aminotransferase (U/L)	34	59	110
Alanine aminotransferase (U/L)	29	31	33
Lactate dehydrogenase (IU/L)	65	93	155

Electricity-related cardiac problems generally emerge as rhythm abnormalities.^[4] Myocardial injury is explained by various mechanisms, which include coronary artery spasm,^[5] direct thrombogenic effects,^[6] direct thermal effects on myocardia,^[5] injury related to arrhythmia-induced hypotension, post-CPR injury (if there is cardiac arrest), and hypoxia. Segmentary arterial spasm has been seen with electricity application in animal experiments.^[7] Lee et al.^[8] showed that endothelial injury not only occurs because of a thermal effect, but also because of intracellular protein denaturation and changes in cell membrane permeability. Because of their high water content, blood and vascular tissue, particularly the inner endothelial layer, have a low resistance to electricity. They are therefore more vulnerable to the propagation of electricity than other tissues like muscle, fat, skin and bone. Accordingly, electrostatic energy can provoke vascular mediopathy and/or intravascular coagulation, even when the outward appearance of skin or soft tissue remains unchanged. Aside from vascular mediopathy, an increase of blood fibrin fragments and plasminogen activator inhibitor and a decrease of tissue plasminogen activator, decreased red blood cell deformability, platelet hyperaggregation, and increased circulatory endothelin are found in rabbits 72 hours after an electric shock^[9,10] These findings support an activation of coagulation even after a low-voltage electric hazard. This tissue-specific sensitivity reasonably explains a distant, isolated vascular thrombosis in organs remote from contact points after a high- or low-voltage electric shock, as in our patient. There are very limited data related to MI after CEW discharge. One case report has been published about a 20-year-old male who developed acute inferior MI after CEW discharge.^[11] In that case, which is similar to ours, an increase in cardiac enzymes, echocardiographically-inferior wall hypokinesia and angiographically-normal coronary

arteries were observed. In our case, normal coronary arteries might be related to the time lapse of 3 hours between CEW application and coronary angiography, and the effects of medications (acetylsalicylic acid, clopidogrel, isosorbide dinitrate, metoprolol, diazepam) on possible spasm and/or thrombus.

The incidence of ECG changes is 31% in electrical injuries. The most frequent changes are nonspecific ST segment changes and sinus tachycardia. In addition, long QT, branch block, atrial fibrillation, ventricular fibrillation, atrial extrasystoles and ventricular extrasystoles can be observed. ST elevation is rarely observed. The right coronary artery (RCA) is more prone to be affected since it is closer to the anterior thoracic wall compared to other vessels.^[12] The biomarkers used to diagnose MI are also used. However, given that assault resulting from arguments, falling, and trauma are common in electrical injuries, it is more suitable to use troponin rather than CK and CK-MB, as their levels might be elevated due to such events. Echocardiography, on the other hand, can be used to determine segmentary wall movement defects. Coronary angiography is more suitable than thrombolytic treatment since trauma and bleeding mostly accompany electrical injuries, and coronary artery obstruction is not often observed in its pathology. The use of antiarrhythmics, ACE inhibitors, angiotensin receptor blockers, and the follow-up and treatment of MI complications are performed in the same manner as those of other MIs.

Besides the direct effect of electricity, multiple factors should be considered and questioned. The effects of extreme stress should be considered as it generally occurs during argument or escape. A meta-analysis suggests that MI occurrence is strongly associated with acute experiences of anger, anxiety sadness, grief, and stress.^[13] The direct role of stress in CAD,

on the other hand, is explained by endothelial damage, elevated heart rate, coagulation, and increased fibrillation.^[14] Previous coronary artery disease, DM, or HT should be investigated since these conditions can facilitate MI. Similarly, cocaine or similar excitant substances, anabolic steroids, energy beverages, and alcohol consumption should be investigated, and adequate tests carried out. As in the present case, sudden and extreme stress should also be considered for patients who do not have most of the additional causes above, and drugs combating stress should be added to the treatment. Moreover, it may be suitable to guide the patient towards stress management techniques after discharge.

Data on the effects of CEWs is rapidly increasing on a parallel with increased use of these devices. While they are highly safe compared to other arms, their use in some circumstances can give rise to certain complications. Existing morbidities, medication usage, and the use of excitants such as cocaine and alcohol, acute extreme emotional disturbance should be queried in cases of cardiac complications developing after CEW use incidents. It is important to monitor CEW patients and to perform cardiac follow-up. ECG, biomarkers (especially troponin) and in certain cases, echocardiography, can be used for diagnosis.

Conflict-of-interest issues regarding the authorship or article: None declared.

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Key words: Conducted electrical weapon; myocardial infarction.

Anahtar sözcükler: Elektrikli silah; miyokart enfarktüsü.