Transient Cardiac Dysfunction After Suffering Electric Shock: Case Report

Bir Olguda Elektrik Çarpması Sonrası Gelişen Geçici Kardiyak Disfonksiyon

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ABSTRACT Temporary or sometimes permanent cardiac function disorders can develop due to myocardial ischemia or infarct in patients who suffer electric shock. We present an 8-year-old case who developed arrest after a short time from suffering electric shock. Ischemic changes have observed on electrocardiography. Hypotension developed in the follow-up and on the transthoracic echocardiographic examination, hypokinesia was detected in the whole septum, more apparently at the anterior and inferior parts of the interventricular septum. The case was put on acetyl salicylic acid, digoxin, enalapril, and spiranolactone treatment. In the follow-up, the electrocardiography showed that ischemic changes were reduced and echocardiography revealed that systolic dysfunction of the left ventricle was diminished. In these cases, the follow-up with serial electrocardiographic, echocardiographic, and cardiac enzymes and delivery of appropriate treatment in line with follow-up are important both in the prevention of certain complications that can develop and of the undesired outcomes of myocardial damage such as permanent ventricular dysfunction.

Key Words: Electric injuries; myocardial ischemia; myocardial infarction

ÖZET Elektrik çarpan olgularda miyokardiyal iskemi ya da infarkt nedeni ile geçici ya da kalıcı kardiyak fonksiyon bozuklukları gelişebilir. Biz bu yazıda elektrik çarpmasından kısa bir süre sonra arrest gelişen 8 yaşındaki bir olguyu sunuyoruz. Elektrokardiyografide bazı iskemik değişiklikler gözlendi. Takipde hipotansiyon gelişti ve transtorasik ekokardiyografik incelemede interventriküler septumun ön ve arka kısımlarında daha belirgin tüm septumda hipokinezi saptandı. Olguya asetil salisilik asit, digoksin, enalapril ve spiranolakton verildi. Takipde, elektrokardiyografi iskemik değişikliklerin azaldığını, ekokardiyografi sol ventriküldeki sistolik disfonksiyonun iyileştiğini gösterdi. Bu olgularda, seri elektrokardiyografik, ekokardiyografik ve kardiyak enzimlerle takip ve bu takibe göre uygun tedavinin verilmesi, gerek gelişebilecek bazı komplikasyonların önlenebilmesi açısından gerekse miyokardiyal hasarın kalıcı ventriküler disfonksiyon gibi istenmeyen sonuçlarının önlenebilmesi açısından önemlidir.

Anahtar Kelimeler: Elektrik yaralanmaları; miyokard iskemisi; miyokard infarktüsü

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Tachycardia, ST-T changes, arrhythmias, cardiopulmonary arrest and conduction disturbances are well-known cardiac complications in patients suffering injuries by electrical current.[1] Morever, temporary or sometimes permanent cardiac dysfunction can develop due to myocardial ischemia or infarction in patients who suffer electric shock.[2]

Many mechanisms have been proposed to account for the myocardial damage seen in electrical injury. These include the induction of coronary ar-

tery spasm, direct thermal injury, ischemia secondary to arrhythmia-induced hypotension, catecholamine-mediated injuries, and coronary artery ischemia as part of a generalized vascular injury.[3,4] Dysfunctions have also been reported in association with regions without direct electrofulguration. The exact mechanism of abnormal contractility in the absence of direct electrofulguration is unknown but may be explained by release of oxygen free radicals, proteolysis of the contractile apparatus, and cytosolic overload of intracellular calcium, followed by reduced myofilament sensitivity to calcium. These abnormalities are consistent with stunned myocardium.[5] We are presenting an 8-year-old case, in whom ischemia due to myocardial damage and ventricular dysfunction developed following electric shock and whose dysfunction completely recovered in the follow-up.

CASE REPORT

An 8-year-old child was brought to the emergency room by his family within ten minutes following the electric shock. When brought to the emergency service, the patient developed respiratory arrest. During the application of cardiopulmonary resuscitation, ventricular fibrillation developed, therefore the patient was defibrillated three times. Consequently, patient resumed sinus rhythm. The patient was extubated as the spontaneous respiration recovered following the short lasting mechanical ventilation.

ELECTROCARDIOGRAPHY AND ECHOCARDIOGRAPHIC FINDINGS

Some ischemic changes were observed in the 12-lead electrocardiography after defibrillation. Diffuse T wave inversion was detected (Figure 1). Hypotension developed in the follow up. The telecardiography was normal. In the transthoracic echocardiographic examination, hypokinesia being more prominent at the anterior and inferior parts of the interventricular septum. Spontaneous contrast of the left ventricle was detected (Figure 2A). In the M-Mode measurement, left ventricular end diastolic and end systolic diameters were within the normal limits for this age whereas fractional shortening was found to be 19% and ejection fraction was found to be 40% (Figure 2B). Hypokinesia was not detected at the right ventricular free wall.

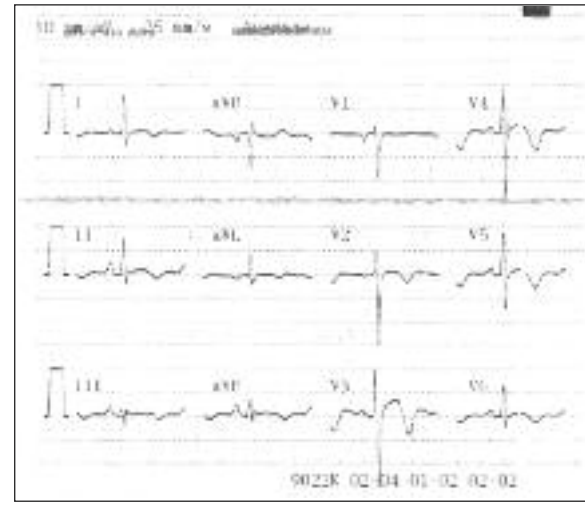

FIGURE 1: Ischemic changes in the 12 lead electrocardiography after defibrillation.

OTHER LABORATORY FINDINGS

An arterial blood gas analysis showed a pH of 7.1, a Po2 value of 158 mm Hg, a Pco2 value of 38 mm Hg, a serum bicarbonate value of 40 mmol/L, a base excess of 14 mmol/L, and an O_2 saturation of 98.3% on a fraction of inspired oxygen of 100%. The CBC count revealed a WBC count of 16 900/mm^3, hemoglobin of 12.1 g/dL, hematocrit of 36.7%, and a platelet count of 369 000/mm^3. The serum analysis was documented as below: glucose 147 mg/dL (70-105), urea nitrogen 34 mg/dL (5-18), creatinine 0.68 mg/dL, aspartate transaminase 387 IU/L (0-37), alanin transaminase 144 IU/L (0-50), creatine phosphokinase-MB fraction 64 IU/L (0-24), lactic dehydrogenase 894 IU/L (120-300), troponin I 9.7 ng/mL (0-0.06), sodium 141 mEq/L, potassium 3.2 mEq/L, chloride 105 mEq/L, and calcium was found 9.3 mg/dL (8.8-10.8). In the urine examination, density and pH were found as 1030 and 5.5, respectively. The case was put on acetyl salicylic acid, 5 mg/kg in a single dose a day, digoxin, 5 μg/kg in twice daily, enalapril, 0.2 mg/kg/day in twice daily and spiranolactone, 2 mg/kg in twice daily. Systemic blood pressure of the patient decreased to normal levels during the course of the

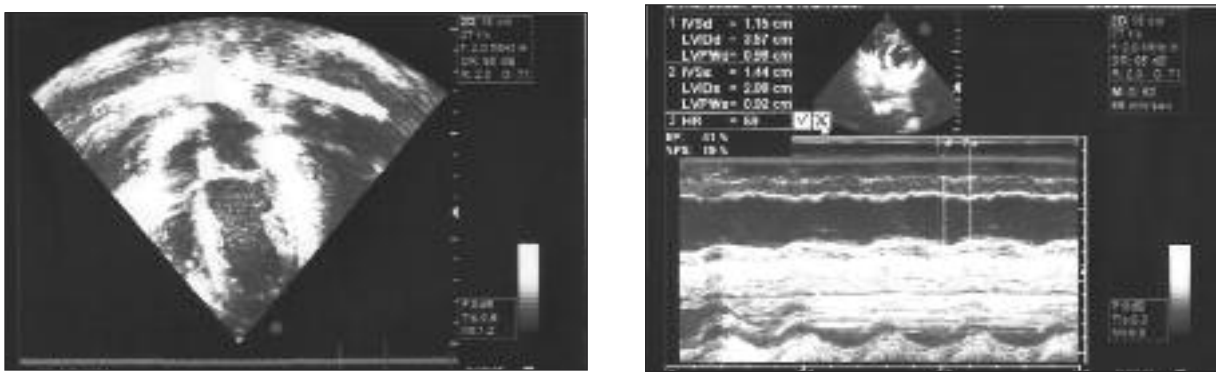

FIGURE 2: A) Spontaneous echo contrast appearance in the transthoracic echocardiographic examination. **B)** Systolic dysfunction in the M-Mode measurement.

examination. The electrocardiograpy showed that ischemic changes were reduced and echocardiography revealed that systolic dysfunction at the left ventricle was diminished.

In the serum examinations of the case conducted one week after the electric shock, glucose, urea, nitrogen, creatinine, and ions were detected normal whereas the following were found to have reduced to the values near to normal limits: aspartate transaminase (77 IU/L), alanin transaminase (91 IU/L), creatine phosphokinase-MB fraction (41 IU/L), lactic dehydrogenase (414 IU/L), and troponin I (0.42 ng/mL). The electrocardiographic examination of the case one week after the electric shock showed that the diffuse T wave inversion transformed into biphasic T wave form and then to normal (Figure 3). In the echocardiographic examination, left ventricular contractions and M-Mode measurements were found to be completely normal (Figure 4).

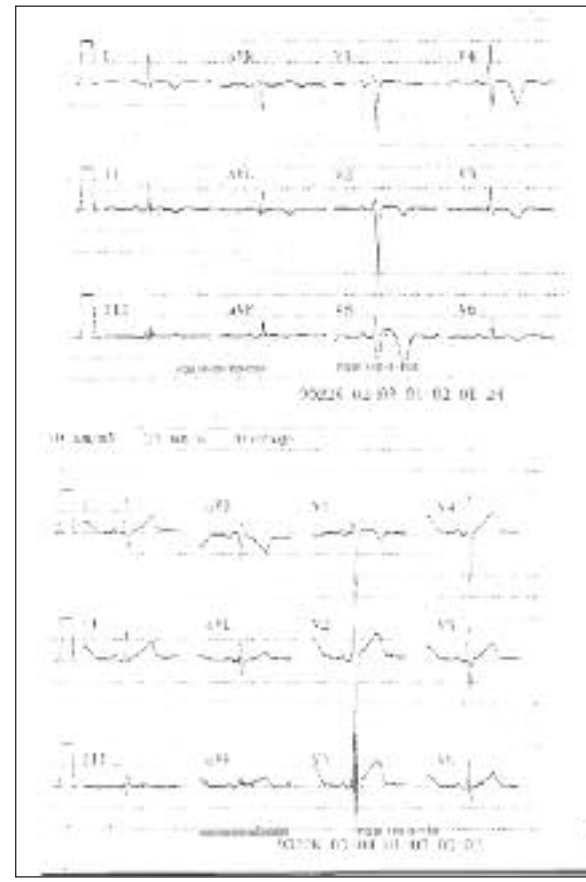

FIGURE 3: The electrocardiographic examination of the case whic is made one week after the electric shock.

DISCUSSION

Quantitation of cardiac damage is best evaluated by echocardiography or nuclear scintigraphy.[4] In order to identify whether cardiac damage due to electric shock has developed in the case and in order to monitor the course of cardiac damage, we have utilized serial electrocardiographic and echocardiographic examinations as well as serum cardiac troponin-I level measurements. Moreover, as the results in electrocardiography were more related to ischemia rather than infarct, we did not feel the necessity for scintigraphy. However, nuclear scintigraphy can be used to identify the ischemic tissue in myocardium and the viable tissue in those with infarct.[6]

Biochemical cardiac markers are useful tools in the diagnosis of both ischemic and nonischemic myocardial injury.[7] Troponin I has been found to

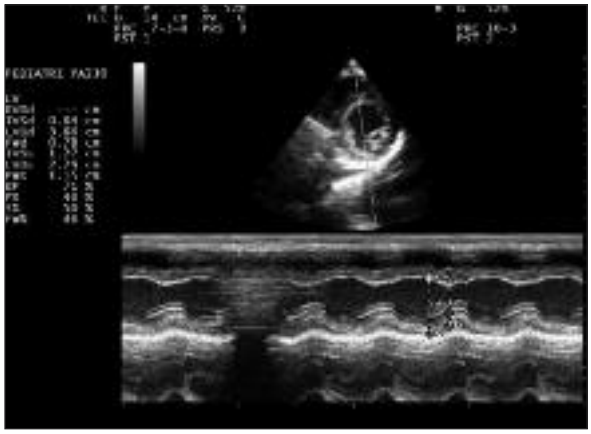

FIGURE 4: The echocardiographic examination whic is made one week after the electric shock.

be a highly sensitive marker for acute myocardial infarction and is equal or superior to the sensitivity of CK-MB. This sensitivity improves with time after injury, reaching its plateau about 12 hours after acute myocardial infarction.[8]

Hence, the serum troponin I level of the case measured 48 hours after the electric shock was found 160 times higher than normal and as systolic dysfunction and electrocardiographic findings recovered in the follow up, it has also come back to normal levels.

Walton et al. report in their study that despite broad electrocardiographic changes of transmural (Q-wave) infarction, left ventricular function was preserved well and that coronary angiography subsequently revealed normal coronary arteries.[9] Although our case did not have apparent findings of infarction, left ventricular dysfunction was present. On the other hand, Lewin et al. detected myocardial injury in the electrocardiography of the case, on which they conducted cardiopulmonary resuscitation following electric shock, in which ventricular fibrillation developed after resuscitation, and on which they ensured normal sinus rhythm after defibrillation, and they found that cardiac enzymes were high, as was the situation with our case. However, due to the diminishing of electrocardiographic abnormalities in the case, they claim that the myocardial damage was a "patchy" necrosis and assert that the electrocardiographic localization of the injury could be accidental.[1]

However, transient ST segment elevation after elective DC cardioversion not followed by concomitant rise in CK-MB or appropriate LDH isoenzymes or myocardial scintigraphy abnormalities have been reported.[10] In our case who did not have an apparent electrocardiographic finding suggesting myocardial infarction, the high level of serum cardiac troponin I, which is a good indicator of myocardial damage, along with systolic dysfunction may be due to a patchy necrosis as reported above.

The high mortality rates associated with high-voltage injuries are due primarily to ventricular fibrillation and asystole. These fatal arrhythmias can occur independent of the degree of myocardial injury and can cause transient cardiac dysfunction irrespective of cardiac muscle necrosis.[3] Ventricular fibrillation can cause left ventricular dysfunction by hypoperfusion. This can result in cardiac necrosis that would be another mechanism by which cardiac injury may have occurred.[4] Although the patient did not stop breathing develop immediately after the electric shock, his respiration got insufficient within 10 minutes and cardiopulmonary resuscitation was required. Ventricular fibrillation developing subsequent to resuscitation was recovered by defibrillation.

Management of ventricular failure requires aggressive support because substantial recovery of ventricular function may ensue over several days to months. Serial evaluation of cardiac function is essential to guide therapy and prevent complications. While no direct cellular data is available, this case suggests that electrical injury may have caused significant myocardial stunning that requires either prolonged recovery before mechanical function is restored or the development of compensatory hypertrophy of the remaining normal myocytes.[4] Clinical trials using ACE inhibitors and betablockers have been shown to blunt these adverse hemodynamic and neurohumoral effects and improve survival. Moreover sotalol has been used in the treatment of arrhythmia.[4] We have given acetyl salicylic acid, enalapril, and spiranolakton at antiaggregant doses orally to the patient. Acetyl salicylic acid was given for the spontaneo-

us echo contrast in the left ventricle. We considered the possibility of a general vascular injury along with coronary artery spasm or coronary artery ischemia. Therefore ACE inhibitor and spironolactone was administered to reduce any possibility of remodeling. Antifibrotic effect of spiranolactone has been reported particularly in studies conducted on adults. We did not start beta blocker for arrhythmia prophylaxis in our case, in whom ischemia rather than myocardial infarct was thought to have developed, and in whom arrhythmia was not detected neither in the follow up nor in the 24-hour Holter ECG monitorization.

Recovery of cardiac function following extensive highvoltage injury is variable. Case reports have, on rare occasions, documented complete recovery of systolic function over several days to weeks.[1,4] However, both left and right ventricular dysfunction have been reported.[3,4] We consider that the reason of permanent damage may occur because of the fact that myocardial damage is in the form of infarct rather than ischemia or infarct may have affected a higher number of cells in some cases. Hence, we saw in our case that systolic functions completely returned to normal in one week after the electric shock. The reason of this may be the fact that ischemia was prevailing in our case rather than infarct findings. On the other hand, the treatment given in order to reduce remodeling might have contributed to this.

Consequently, in cases who have suffered electric shock, we believe that in addition to a timely and good cardiopulmonary resuscitation, follow up with serial electrocardiography, echocardiography, and cardiac enzymes and delivery of appropriate treatment are important both in the prevention of certain complications that can develop and of the undesired outcomes of myocardial damage such as permanent ventricular dysfunction.

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