

## Delayed ST-Segment Elevation after Electrical Injury Mimicking Acute Myocardial Infarction: A Case Study

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**Abstracts:** 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 delayed transient ECG changes have also been described after electrical injury, such as ST segment elevation. 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. Here, we report a man with delayed ST Segment Elevation due to electric shock. Although he had frankly normal coronary arteries by coronary angiography, myocardial infarction was objectively evident by cardiac enzymes & electrocardiography. He was discharged in good health with non-specific electrocardiographic changes. [Dalvani V NJIRM 2015; 6(1):107-110]

**Key Words:** Electrical injury, ST segment elevation, electrocardiographic changes.

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**Introduction:** Electrical injury can cause cardiac problems such as dysrhythmias, myocardial or 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.<sup>1</sup> The heart in particular is liable to be damaged by electrical injury. Less serious transient ECG changes have also been described after electrical injury.<sup>2</sup> In our report, a case of delayed ST elevation in ECG after electrical injury is presented.

**Case Report:** A 30 year-old patient was referred to our hospital with a history of exposure to 240 volt alternative electric current while working. He was immediately taken to nearby hospital where he had arrhythmias. He was intubated, resuscitated from outside private hospital and was referred with ambulatory care for further management to our hospital. He was immediately hospitalised in ICU for further management. On examination, he was drowsy, disoriented, irritable but responding to deep painful stimuli with a Glassgow coma scale (GCS) of 10. His blood pressure was 120/70 mm Hg with a pulse of 120/min regular in rate & rhythm and respiratory rate of 22 cycles/min. His oxygen saturation was 98% on mechanical ventilation with

Fio2 80%, body temperature was 36.5°C. His lungs had equal and good air entry without any pathological sounds. He had tachycardia 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. On his right arm & hand there were burn marks. CNS examination revealed pupils of normal size & bilaterally reacting to light. Other systemic examinations were normal. His complete blood count, serum electrolytes, serum calcium, coagulation profile and cardiac enzymes were normal at the time of admission. ECG revealed sinus tachycardia with no significant ST-T changes. (Fig. 1) Further the patient was under constant cardiac monitoring. Beta-blocker and angiotensin converting enzyme inhibitor therapies were started. However, his general condition improved gradually after 3 days and he was weaned off from the ventilatory support. His GCS improved to 15. Two days later, ECG changes appeared in the form of ST segment elevation (2mm) in lead V1-V5. (Fig.2)

Cardiac enzymes were tested again. CPK-MB showed a considerable rise from 17 U/L (at the time of admission) to 115 U/L (reference range 7-25 U/L). Serum CPK total was also markedly raised from 680 U/L (at the time of admission) to 1522

U/L (reference range 35–232 U/L). Troponin I levels too were increased from 1.4 ng/ml (at the time of admission) to 10 ng/ml (reference range <1.5 ng/ml). However, there was no haemodynamic instability. Echocardiographic assessment revealed mild distal septal hypokinesia, normal left ventricular size and function (EF > 50%), no valvular pathology and no regional wall motion abnormality. Coronary angiogram was suggestive of normal coronary arteries with no occlusion. The patient's symptoms resolved one day later with ECG suggestive of ST segment depression and T wave inversion in leads V1-V5. (Fig. 3) The patient was discharged from the hospital with complete recovery.

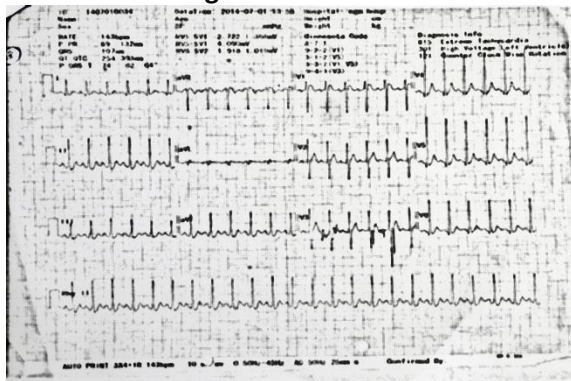
**Discussion:** Electrically injured patients typically are young and male<sup>3</sup>, and electrical injury to adults mostly occurs in an occupational setting<sup>4</sup>. Widespread and conflicting data is available in terms of myocardial injury after an electric shock. Despite the controversy, some mechanisms have been proposed to account for myocardial injury after electrical shock. These are: coronary artery spasm<sup>5</sup>; direct thrombogenic effect on coronary arteries<sup>6</sup>; direct thermal effect on myocardium<sup>5</sup>; ischemia secondary to arrhythmia-induced hypotension<sup>7</sup>; coronary artery ischemia as part of a generalized vascular injury<sup>8</sup>; and direct contusion during cardiopulmonary resuscitation with subsequent coronary artery injury<sup>9</sup>. Also, hypoxic condition after respiratory arrest might possibly contribute to myocardial injury<sup>10</sup>. In one report, abnormal ECG was detected in approximately 31% of patients following an electric shock<sup>11</sup>. Non-specific ST segment changes and sinus tachycardia are the most commonly reported ECG findings<sup>12</sup>. Other ECG findings include QT prolongation, bundle branch block, atrial and ventricular fibrillation, atrial and ventricular premature contractions<sup>11,12</sup>. Given the non-transmural nature of necrosis, ST segment elevation rarely occurs due to electrical shock<sup>13</sup>. These notable ECG changes can normalize and tend to be totally reversible in long-term survivors<sup>11</sup>. Both CPK total and CK-MB can markedly increase, owing to concomitant skeletal muscle injury and cardiopulmonary resuscitation as in this case. 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<sup>14</sup>. The above-mentioned 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. 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 myocardium, as well as regional hypokinesia.

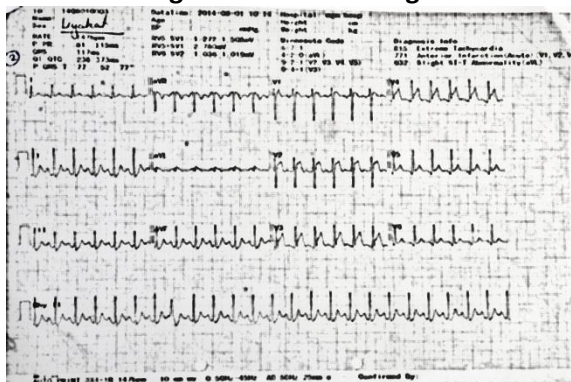
Echocardiographic findings may markedly improve in the follow-up<sup>15</sup>. Additionally, echocardiography may provide confirmation of the induced myocardial injury, as happened in the present case. In essence, coronary angiography is the first choice for the detection of the underlying mechanism of myocardial injury after electrical shock. Lesions are categorized as obstructive or non-obstructive. Demonstration of normal coronary arteries evokes non-obstructive mechanism. Coronary artery spasm<sup>5</sup>, direct thermal effect on myocardium<sup>5</sup>, ischemia secondary to arrhythmia-induced hypotension<sup>7</sup>, direct contusion during cardiopulmonary resuscitation with subsequent coronary artery injury<sup>9</sup> and hypoxic condition after respiratory arrest<sup>10</sup>, could all have contributed to myocardial injury in the current case. 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. On the other hand, myocardial injury might occur due to a non-occlusive mechanism, as in the present case. So 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<sup>15</sup>. Angiotensin converting enzyme inhibitors are as reliably effective as angiotensin II receptor blockers at protecting against remodeling<sup>15</sup>. Cardiac complications are managed similarly to other myocardial infarction causes and require follow-up evaluation<sup>16</sup>.

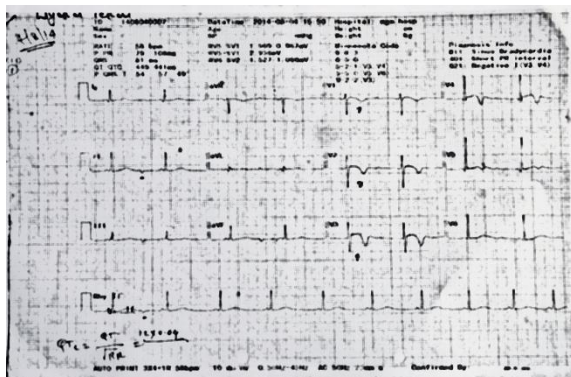
**Figure 1: Normal ECG**



**Figure 2: ECG Showing AMI**



**Figure 3: ECG showing ST segment depression and t wave inversion in leads V1-V5.**



**Conclusion:** 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 towards a more specific treatment. Considering the high incidence of cardiac complications, patients should ideally be observed closely during hospitalization and after discharge.

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

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