Case Report

ST-segment elevation after direct current shock mimicking acute myocardial infarction: a case report and review of the literature

Abstract

External direct current (DC) shocks are and have long been commonly used for electrical cardioversion/defibrillation of atrial or ventricular arrhythmias. ST-segment elevation after cardioversion with DC is an easily ignored phenomenon, occurring acutely and resolving during the first few minutes postshock. Here, we describe electrocardiographic findings of widespread ST-segment elevation lasting at least 1 hour after DC cardioversion for ventricular defibrillation due to Brugada syndrome and mimicking acute myocardial infarction (AMI). This case of ST-segment elevation without a dynamic and evolving AMI underscores the need to consider other causes of ST-segment elevation.

A 39-year-old man smoker lost consciousness suddenly and was immediately transferred to the nearest local community hospital while receiving continuous chest compressions by family members. Half an hour later at the emergency department, electrocardiogram (ECG) showed ventricular fibrillation, which converted to sinus rhythm after the patient was given a 360 J monophasic DC shock. Postresuscitation ECG 1 hour after defibrillation showed marked ST-segment elevation in leads I, aVL, and V1 to V6 (Fig. 1A). This was considered suggestive of an electrocardiographic finding of widespread ST-segment elevation due to Brugada syndrome and mimicking acute myocardial infarction (AMI). This case of ST-segment elevation without a dynamic and evolving AMI underscores the need to consider other causes of ST-segment elevation.

Coronary angiography (Fig. 2A and B) showed patent left coronary artery system and right coronary artery.

What caused the widespread ST-segment elevation? Was it induced by electrical cardioversion? And what caused the ventricular fibrillation? Continuous ECG monitoring during the next few days revealed 3 types of repolarization patterns in the right precordial leads identical to those of Brugada syndrome (Fig. 1D-F). Therefore, the electrocardiographic findings of widespread ST-segment elevation were caused by DC shock for ventricular defibrillation induced by Brugada syndrome. The patient underwent implantation of an implantable cardioverter defibrillator (ICD) and was discharged uneventfully; he has been event free during 1-year follow-up.

ECG is the most accessible and widely used diagnostic tool for patients with acute myocardial infarction (AMI), providing good correlation between ST-segment elevation and culprit coronary artery [1]. However, other conditions may mimic electrocardiographic signs of AMI [2]. Electrical cardioversion is one of the causes of ST-segment elevation.

The prevalence of ST-segment deviations after transthoracic and epicardial shocks varies between 15.4% and 48%, depending on recording methods, including number of leads and recording timing; whether transthoracic, epicardial, or endocardial ICD shocks were applied; whether DC shocks were for atrial or ventricular arrhythmias; and model and energy applied in DC shocks [3-5]. Gurevitz et al [3] studied ST-segment changes in 28 patients undergoing 125 shock episodes during ICD implantation. ST deviations were observed after 49 (39%) of all shock episodes, including ST elevation in 30 (24%) of all shock episodes and ST depression in 31 (25%). ST depressions occurred more frequently after higher energy shocks, whereas ST-elevation prevalence was independent of energy applied [3]. Rumeau et al [4] prospectively studied the presence of ST-segment changes on 12 lead ECG immediately after the first DC shock in 91 consecutive patients referred for external cardioversion of atrial fibrillation. ST-segment changes were documented in 44 (48%) of all cardioversions, with ST depression in 12 (13%) and ST elevation in 32 (35%). Although ST-segment changes were not related to energy, ST elevation was significantly more often induced by monophasic and depression by biphasic DC shocks [4]. Kok et al [5] found ST-segment elevation in 15.4% of patients with ventricular tachyarrhythmias treated with transthoracic cardioversion, more commonly among those with lower left ventricular systolic function.

The recording leads of ST changes are in accordance with an anterolateral location near the defibrillation patches. Rumeau et al [4] found that ST changes were only observed in precordial leads except V6, being more prominent in V1 in 5%, V2 in 7%, V4 in 15%, V5 in 5%, and V6 in 5%, without significant difference between ST depression or elevation.

Although several theories have been proposed for postshock ST deviation, the exact mechanism has not been clearly established. Coronary spasm induced by DC shocks has been implicated as a cause for postcardioversion ST deviation. However, the fact that it takes at least 45 seconds after coronary spasm onset for ST-segment elevation to occur is inconsistent with the acute phenomenon of ST deviation after cardioversion, which occurs immediately and therefore more likely to reflect direct effects of the electrical currents on the myocardium than coronary spasm. Myocardial damage/subepicardial necrosis also has been suggested as an explanation for postcardioversion ST deviation. However, in patients who underwent DC cardioversion for atrial fibrillation or flutter, serum

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CK level became significantly elevated without changes in cardiac troponin level, indicating that the CK is derived from skeletal muscle and that cardiac damage does not occur as a result of cardioversion [4,6-8]. The theory of myocardial necrosis was further undermined by the demonstration by myocardial scintigraphy that transient ST-segment elevation after DC cardioversion does not necessarily indicate myocardial injury [9]. The theory proposing sustained depolarization of a critical mass of myocardium closest to the origin of the electrical current is best favored to account for postcardioversion ST deviation. In the myocardial cell layers closest to the origin of the countershock current, electrical currents produce transient sarcolemmal microlesions, which short circuit the membrane causing immediate depolarization and significant ionic exchange across the cell membrane, a phenomenon called “electroporation.” The depolarization may persist for many seconds to minutes [10], and a difference in electrical potential between the depolarized and normal

![Fig. 1. Dynamic ECG changes in the patient. (A) ECG 1 hour after defibrillation showed marked anterolateral ST-segment elevation, whereas ECG 3 hours after defibrillation (B) and admission ECG at our hospital (C) showed complete ST-segment resolution without T-wave inversion or Q-wave formation. Brugada type 1 (D), type 2 (E), and type 3 (F) ECG patterns were found during the next few days.](image)

![Fig. 2. Emergency angiography revealed patent left coronary system (A) and right coronary artery (B).](image)
tissue could be reflected as ST-segment deviations in the recording leads of the surface ECG.

ST-segment elevation post-DC elective cardioversion is a transient short-lived phenomenon, being maximal just after the DC shock with mean duration of 60 seconds, and usually resolving 5 minutes postshock [4]. However, in our case, the anterolateral ST elevation lasted at least 1 hour, which could be explained by the longer duration of resuscitation (30 minutes) as well as by the underlying Brugada syndrome. The higher prevalence of ST-segment elevation after transthoracic cardioversion in patients with out-of-hospital cardiac arrest (42%) than in patients with hemodynamically unstable ventricular tachyarrhythmia induced during invasive electrophysiologic study (15.4%), may be related with the longer median time interval between cardiac arrest and restoration of spontaneous circulation (25 minutes vs 1 minute) [5,11]. Propofol, known as a blocker for several ion channels, facilitates ST elevation after external cardioversion with DC shock [4]. It is speculated that various electrophysiologic and metabolic properties of the drug may interfere with phenomena such as electroproporation. It is therefore not surprising that the Brugada syndrome, an electrophysiologic disease produced by dysfunction of a cardiac ion channel involved in the generation of the action potential, might exaggerate ST elevation after DC shock [12].

Serial ECG monitoring is a powerful method to differentiate AMI from other conditions causing ST-segment elevation, and it may reveal the underlying electrophysiologic phenomena. For instance, in pericarditis, ST-segment elevation rarely exceeds 5 mm, and the segment usually retains its concavity, whereas early repolarization pattern is characterized by concave ST-segment elevation and asymmetrical tall upright T wave [13]. Acute myocardial infarction is a dynamic and evolving process with a predictable progression of changes: ST-segment elevation gradually resolves followed by T-wave inversion, R-wave regression, and Q-wave appearance. ST-segment elevation due to DC shock, which is a diagnosis of exclusion after coronary angiography has ruled out ischemic causes, should be considered if transient ST-segment elevation occurs immediately after DC shocks without highly specific changes for an AMI as exemplified in this case.