Return of Spontaneous Circulation after Correction of Acidosis in Prolonged Cardiac Arrest: A Case Report

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A 47-year-old man presented to the emergency department with out-of-hospital cardiac arrest. The patient had a previous history of heart transplantation with implantable cardioverter defibrillator (ICD). Ventricular fibrillation persisted despite 18 minutes of standard advanced cardiac life support at the emergency department. He received intravenous administration of 68 meq sodium bicarbonate and subsequent return of spontaneous circulation was noted after correction of acidosis with sodium bicarbonate in prolonged cardiac arrest.

Key words: prolonged cardiac arrest, return of spontaneous circulation (ROSC), sodium bicarbonate, ventricular fibrillation

Introduction

The likelihood of successful defibrillation and resuscitation decreased as duration of cardiac arrest increases. Prolonged cardiac arrest is also associated with the occurrence of acidosis (1). Administration of sodium bicarbonate was associated with increased rates of restoration of spontaneous circulation in dogs with prolonged cardiac arrest (1,2). We report a case of return of spontaneous circulation (ROSC) after correction of acidosis at late stage of resuscitation in a patient who had a previous history of heart transplantation with implantable cardioverter defibrillator (ICD) 15 years ago.

Case Report

A 47-year-old male patient presented to our emergency department (ED) with dyspnea, nausea, and followed by fainting and cardiac arrest at the bathroom and resuscitation was initiated by emergency medicine technicians (EMTs). He had a previous history of heart transplantation with implantable cardioverter defibrillator (ICD) 15 years ago. Automated external defibrillator (AED) showed no indication for defibrillation. On arrival to the ED, cardiopulmonary resuscitation (CPR) and advanced cardiovascular life support (ACLS) were performed including endotracheal intubation with ventilated, cardiac massage, and intravenous administration of 1mg epinephrine every 3 minutes. Six minutes after resuscitation, an electrocardiograph (ECG) monitor showed ventricular fibrillation (VF). Biphasic defibrillation with 200 joule was given with consequence CPR. A single dose of 40 units vasopressin also given. The patient received intravenous administration of amiodarone...
300mg and Lidocaine 100mg followed by defibrillation was performed respectively. But the patient had persistent VF despite aggressive treatment according to ACLS algorithm (Fig. 1). Therefore, intravenous administration of 68 meq sodium bicarbonate was given for palliative treatment in the late stage of CPR. Dramatically, the recovery of spontaneous circulation occurred 20 minutes after resuscitation. The vital signs measured as blood pressure 90/30mmHg, pulse rate 70/min, and respiratory rate 20/min with ventilator support, and fluid resuscitation and intravenous dopamine and norepinephrine infusion were given.

Abnormal laboratory data revealed serum creatinine 3.5mg/dl, glucose 226mg/dl, potassium 3.2meq/L, blood urea nitrogen 31mg/dl. Arterial blood gas analysis showed pH 7.031, PaCO₂ 65.7mmHg, PaO₂ 36mmHg, HCO₃⁻ 17meq/L, BE −13.8, O₂ saturation 45.3%. The patient also treated by intravenous administration of 68meq sodium bicarbonate shortly after to correct metabolic acidosis. The family decided transfer to the previous hospital in which the surgery was performed. A 12 Leads ECG revealed pacemaker rhythm at the rate of 70 beats/min (Fig. 2). Before transfer, the vital signs measured as pulse rate 73/min, and blood pressure 111/52mmHg with ventilator and inotropic support.

**Discussion**

According to the standard advanced cardiac life support (ACLS), administration of sodium bicarbonate (SB) in cardiac arrest was controversial (3). Buffer administration during CPR promoted cerebral cortical reperfusion and mitigated subsequent post-resuscitation cerebral acidosis during lower blood pressure and flow in the reperfusion phase (4). Metabolic acidosis occurred during CPR, to have negative effect on cellular metabolism, to alter the propensity for induction of ventricular fibrillation (VF), and to...
diminish the cardiac and peripheral response to catecholamines. According to that reason, patients with cardiac arrest had a poor prognosis when untreated should be treated with bicarbonate to buffer the acidosis. This approach was substantiated in one experimental study in which the combination of catecholamines and bicarbonate seemed to improve the ability to resuscitate animals and to have them survive intact\(^5\). Although buffer agents alone have failed to improved the success of resuscitation, we now examine the widely held concept that is the combination effect of epinephrine (EPI) and sodium bicarbonate (SB) that significantly impact both immediate post-resuscitation hemodynamic and metabolic parameters as well as long term outcome\(^5,6\).

Bicarbonate therapy during CPR may have a beneficial or detrimental effect of its own, enhance the effect of EPI during CPR by improved receptor binding and correction of base deficit or counteract the side effects of EPI in the post-resuscitation period by correcting acidosis\(^7\). In previous study, the author revealed the rate of SB use decreased with increasing patient age-primarily reflecting shorter CPR attempts. They suggest that guidelines for SB use during CPR should emphasize the importance of pre-ACLS hypoxia time in contributing to metabolic acidosis and should be more specific in defining the duration of “protracted CPR or long resuscitation efforts”, the most frequent indication for SB administration\(^8\). The empirical early use of SB (1mEq/kg) has no effect on overall outcome in prehospital cardiac arrest. However, a trend toward improvement in prolonged (>15 minutes) arrest outcome was described\(^9\). In our case, the patient received resuscitation according to ACLS algorithm, but still had refractory VF 18 minutes from starting CPR. Finally, intravenous administration of 68meq sodium bicarbonate was given for correction of acidosis as palliative treatment in the late stage of CPR. We found ROSC shortly after. In one study, buffer therapy promotes successful resuscitation after prolonged cardiac arrest, regardless of coronary perfusion pressure\(^10\). Sodium bicarbonate, and to a lesser degree, carbicarb, are beneficial in promoting early ROSC\(^10\). In one study with animal model, bicarbicarb and sodium bicarbonate increased bicarbonate levels and corrected pH in the arterial and mixed venous blood\(^11\). In this model of cardiac arrest, carbicarb was not superior to sodium bicarbonate in correction of metabolic acidosis during CPR\(^11\).

Extramyocardial acidosis below pH 7.1 decreased cardiac performance and resuscitability after VF. This result indicates that progressive acidemia during cardiac arrest is one of the most important determinants of cardiac resuscitability\(^12\). In another study, buffer agents administered during CPR are evaluated. Although buffer agents may not improve the success of resuscitation when administered during CPR, they may ameliorate postresuscitation myocardial dysfunction and thereby improve postresuscitation survival\(^13\). Although administration of SB in cardiac arrest was controversial, we suggested that correction of acidosis by SB with adequate ventilation in the prolonged CPR was the choice of clinical practice. Electrolyte and metabolic abnormalities affect the threshold for myocardial stimulation in patients with cardiac pacemakers\(^14\). Correction of acidosis and hypoxia may improve the pacemaker malfunction during CPR. We believe that all the efforts must not be given up when such a critical condition.

In conclusion, correction of possible acidosis and electrolyte imbalance with adequate ventilation may be an important factor of ROSC in such a critical condition. The initial laboratory data in-time may be helpful during resuscitation. To our knowledge, our case is a rare finding of ROSC after correction of acidosis at the late stage of CPR in a patient of previous heart transplantation with ICD.
References


持久心跳停止患者矯正酸中毒之後恢復自發性脈搏：

病例報告

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我們報告一位47歲心臟移植之後再植入心臟去脈器的病患在醫院外心跳停止後到本院急診室急救。雖然給予基本救命術以及高級心臟救命術十八分鐘，仍然呈現心室纖維顫動，病患在急診室經救治沒有改善，最後給予六十八毫當量碳酸氨銨靜脈注射，隨後發現恢復自發性脈搏。

關鍵詞：持久心跳停止，恢復自發性脈搏，碳酸氨銨錠，心室纖維顫動