CLINICAL CASE REPORT

Complete Recovery With the Chain of Survival After a Prolonged (120 Minutes) Out-of-Hospital Cardiac Arrest Due to Brugada Syndrome

A Case Report

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Abstract: Out-of-hospital cardiac arrest (OHCA) is a crucial public health problem. To improve outcomes of patients after cardiac arrest, the American Heart Association promotes the concept of the chain of survival.

We report a case of a 19-year-old man with no markedly past medical history who suffered from OHCA, and he was resuscitated with cardiopulmonary resuscitation, without interruption, during the rescue process for 120 minutes until return of spontaneous circulation (ROSC). Electrocardiogram on admission showed right bundle branch block and ST segment elevation in leads V1–V2, and the patient’s uncle had experienced the same event and had received implantable cardioverter defibrillator (ICD) treatment. Therefore, the patient was diagnosed with Brugada syndrome. Postcardiac arrest care was performed after ROSC, including mild therapeutic hypothermia, hemodynamic monitoring and management, and ICD implantation, and then the patient completely recovered without any noticeable neurological or intellectual deficits in the follow-up examinations.

Our case demonstrates that even after an OHCA with prolonged time (120 minutes) until ROSC, survival with a favorable neurological outcome is possible, provided implementation of an extremely effective rescue chain.

INTRODUCTION

Brugada syndrome is an autosomal dominant genetic heart disease.1 It is thought to be responsible for fatal ventricular tachyarrhythmia that may lead to syncope or out-of-hospital cardiac arrest (OHCA) in young people without structural heart disease.2,3 Because arrhythmic events are more common at night and during sleep, it is difficult to diagnose and treat patients with Brugada syndrome.4 Here, we report a case of OHCA caused by Brugada syndrome, which had been successfully treated with “chain of survival” interventions.

CASE REPORT

A 19-year-old man with no markedly past medical history was found lying on the floor, unresponsive, by his colleagues at 09:40 AM of October 28, 2014. One colleague immediately called the emergency medical services (EMSs) by mobile phone, while another performed bystander cardiopulmonary resuscitation (CPR). The ambulance with 2 EMS providers arrived within 5 minutes. They found the patient pulseless, with no signs of breathing and nonreacting pupils. The first electrocardiogram detected pulseless electrical activity (PEA). The patient was transported to the emergency department under continuous manual chest compression. The patient did not receive any medications before admission.

The patient arrived at the Emergency Department of Nanjing Drum Tower Hospital 15 minutes after cardiac arrest. He had poor general conditions with Glasgow Coma Scale score 3; he had no spontaneous respirations and heart rhythm, no response to verbal stimuli, and had fixed and dilated pupils. The advanced life support protocol was immediately started by emergency physicians in our department. The patient was intubated and mechanically ventilated, while CPR was continuously performed using a gas-driven mechanical chest compression (Grand Rapids, Michigan Instruments; Hirtz, Koln, Germany) with compression rate 100/min and deepness of chest compression 5 cm). Ventricular fibrillation was first detected 35 minutes after cardiac arrest by the defibrillator, and 4 defibrillations (ventricular fibrillation or pulseless ventricular tachycardia) were performed in all, following injections of adrenaline (5 mg in all), as well as amiodarone and dopamine infusions. One hundred and twenty minutes after cardiac arrest, he presented successful return of spontaneous circulation (ROSC) with blood pressure 85/42 mm Hg and heart rate 103 bpm. The patient showed hypoxaemia (pulse oxygen saturation <90%; positive end-expiratory pressure 12 mm Hg, FiO2 100%, 1 hour after ROSC), although invasive mechanical ventilation was given. The arterial blood gas was pH 7.309, partial arterial CO2 pressure 40.3 mm Hg, PO2 52.8 mm Hg, FiO2 100%, and P–O2/FiO2 52.8, and the chest x-ray showed pulmonary edema (Figure 1).
Then the patient was moved to the emergency intensive care unit 3.5 hours after the onset, and his vital signs were as follows: temperature 39.8°C, heart rate 110 bpm, blood pressure 83/55 mm Hg (dopamine infusion 12 mcg/kg/min), and oxygen saturation 95% (mechanical ventilation, FiO2 50%), and the serum neuron-specific enolase (NSE) was significantly elevated (75.05 ng/mL, reference 0–16.3 ng/mL). Neurological examination was notable for the following: disappeared spontaneous respiration, fixed and dilated pupils, absent corneal reflexes, and absent motor responses to painful stimuli in all extremities. The repeat electrocardiogram showed a right bundle branch block and ST segment elevation in leads V1–V2 (Figure 2). The patient’s uncle had experienced the same event and had received implantable cardioverter defibrillator (ICD) treatment 1 year earlier. Therefore, the patient was diagnosed with Brugada syndrome.

A mild therapeutic hypothermia (MTH) was started (~5 hours after cardiac arrest) by infusing a cold (4°C) saline solution (1.8 L in all). The body temperature decreased to 35.9°C (24 hours after cardiac arrest), and the core temperature was maintained between 34°C and 36°C for 24 hours in combination with an external cooling mattress (HICO-Hypotherm 680 of Hirtz and HICO-Polyurethan water mat; Hirtz, Koln, Germany). Shivering appeared in MTH; the shivering was suppressed with lytic cocktail therapy (chlorpromazine, promethazine, and pethidine) and high doses of sedatives (midazolam, fentanyl, and propofol). No other complications occurred during the cooling procedure. Bedside echocardiography showed left heart insufficiency and an ejection fraction of 33%. Because of heart dysfunction and unstable circulation (systolic blood pressure 76–83 mm Hg), the patient received dopamine and an intraaortic balloon pump (IABP), and pulse index continuous cardiac output (PiCCO) monitoring system was introduced (Table 1). Active rewarming was initiated at 0.2°C/hour 48 hours after cardiac arrest. The rewarming goal temperature of 37.0°C was reached within 6 hours. Dopamine infusion and IABP were then discontinued because circulatory condition and heart function had improved.

The patient’s consciousness improved 58 hours after cardiac arrest; he was able to open his eyes to command and respond to painful stimuli. He was awake and intermittently moved his extremities 71 hours after cardiac arrest. The patient was extubated after a successful spontaneous breathing trial on day 4 after cardiac arrest. Then he spoke in full sentences, was fully aware of person, place, and time, and accurately exhibited full strength in all his extremities. The repeat NSE was 13.77 ng/mL. A non-contrast head computed tomography scan was performed, which showed no evidence of intracranial hemorrhage, mass infarction, or cerebral edema. He was able to ambulate with a walker and independently perform activities of daily life on day 5 after the cardiac arrest. Magnetic resonance imaging and an electroencephalogram were performed, showing nothing abnormal. He completely recovered with 1 point based on the criteria of cerebral performance category on day 8 after cardiac arrest and was then transferred to the cardiology department. He was implanted an ICD on day 10 after the cardiac arrest. The patient was followed up at 1 and 4 months after discharge and he had no noticeable neurological or intellectual deficits (Figure 3).

**DISCUSSION**

OHCA is a crucial public health problem. The estimated incidence of EMS-treated OHCA in the United States is about 40.3 to 86.7/100,000 persons/year, and the overall survival rate is only 3.0% to 16.3%. To improve outcomes of patients after cardiac arrest, the American Heart Association promotes the concept of the chain of survival. Following the new principle, patients with OHCA have a higher survival rate and better neurological outcomes.

Basic life support (BLS) is the foundation of chain of survival for a higher survival rate. Continuous chest
Compression is a critical component of BLS because cardiac output and coronary perfusion depend on compression during CPR. Therefore, chest compressions should be the highest priority and the initial action for the patients with OHCA. In our case, high-quality CPR that contributed to the successful resuscitation and favorable neurological outcome was performed, without interruption, during the rescue process until ROSC. Although the treatment of mechanical chest compression–decompression system in OHCA is controversial, at least during prolonged resuscitation, as in our case, it may avoid reducing the quality of CPR. Zimmermann et al also found that mechanical CPR improved the outcome of a patient with OHCA.

Early defibrillation is critical to survival from OHCA because the most frequent initial heart rhythm in OHCA is ventricular fibrillation, which should be immediately terminating the rhythm and allowing effective chest compressions.

Table 1: Time Course and Data

| Time After Cardiac Arrest, h/d | Day 1 | Day 2 | Day 3 |
|--------------------------------|-------|-------|-------|
|                                 | 1 h   | 3 h   | 5 h   | 7 h   | 8.5 h | 11 h | 19 h | 21 h | 24 h | 29 h | 30.5 h | 36 h | 47 h | 54 h | 56 h |
| Arterial blood gas              |       |       |       |       |       |      |      |      |      |      |        |      |      |      |
| pH                             | 7.22  | 7.309 | 7.321 | NA    | 7.32  | NA   | 7.332| NA   | 7.307| 7.356| NA     | 7.353| NA   | 7.391| NA   |
| PacO2, mm Hg                   | 50.3  | 40.3  | 38.1  | NA    | 38.8  | NA   | 40.7 | NA   | 46.8 | 38.5 | NA     | 45.1 | NA   | 42.6 | NA   |
| PaO2, mm Hg                    | 64.2  | 52.8  | 69.3  | NA    | 80.5  | NA   | 87.4 | NA   | 113.1| 179.6| NA     | 215.6| NA   | 187.1| NA   |
| PO2/FIO₂                       | 64.2  | 52.8  | 138.6 | NA    | 161   | NA   | 174.8| NA   | 251.3| 449  | NA     | 716.7| NA   | 623.7| NA   |
| Lactate, mmol/L                | 5.7   | 3.5   | 1.4   | NA    | 1.1   | NA   | 0.9  | NA   | 0.8  | 0.9  | NA     | 0.7  | NA   | 0.7  | NA   |
| HCO₃⁻, mmol/L                  | 20.8  | 19.9  | 19.9  | NA    | 20.2  | NA   | 21.7 | NA   | 23.6 | 21.7 | NA     | 25.3 | NA   | 26.1 | NA   |
| K⁺, mmol/L                     | 4.37  | 3.50  | 3.52  | NA    | 4.47  | NA   | 4.55 | NA   | 4.22 | 4.03 | NA     | 4.18 | NA   | 4.14 | NA   |
| PEEP, cm H₂O                   | 6     | 12    | 12    | 12    | 12    | 12   | 8    | 8    | 8    | 6    | 6      | 6    | 6    | 6    | 6    |
| PiCCO system                    |       |       |       |       |       |      |      |      |      |      |        |      |      |      |
| CVP, mm Hg                     | NA    | NA    | 6     | 10    | 8     | 13   | 11   | NA   | 9    | NA   | 13     | NA   | 17   |
| GEDVI, mL/m²                   | NA    | NA    | 506   | 586   | 554   | 590  | 557  | NA   | 557  | NA   | 520    | NA   | 560  | 619  |
| EVLWI, mL/kg                   | NA    | NA    | 15    | 11.7  | 10.6  | 11.4 | 10.5 | NA   | 7.8  | NA   | 6.7    | NA   | 6.6  |
| PVPI                           | NA    | NA    | 4.8   | 3.0   | 2.9   | 2.9  | 2.9  | NA   | 2.3  | NA   | 1.8    | NA   | 1.6  |
| MAP, mm Hg                     | NA    | NA    | 57    | 67    | 70    | 75   | 77   | NA   | 77   | NA   | 68     | NA   | 87   |
| Heart rate, bpm                | NA    | NA    | 120   | 106   | 105   | 82   | 85   | NA   | 81   | NA   | 61     | NA   | 88   |
| CI, L/min/m²                   | NA    | NA    | 2.53  | 2.46  | 2.59  | 2.19 | 2.49 | NA   | 2.95 | NA   | 2.64   | NA   | 3.17 |
| Vasoactive agent               |       |       |       |       |       |      |      |      |      |      |        |      |      |      |
| Dopamine, mcg/kg-min           | NA    | 20    | 20    | 16    | 14    | 14   | 13   | 10   | 8    | 6    | 4      | 4    | 3    | Stop |

CI = cardiac index, CVP = central venous pressure, EVLWI = extravascular lung water index, GEDVI = global end-diastolic volume index, MAP = mean arterial pressure, NA = not available, PacO₂ = partial arterial CO₂ pressure, PaO₂ = partial arterial O₂ pressure, PEEP = positive end-expiratory pressure, PiCCO = pulse index continuous cardiac output, PVPI = pulmonary vascular permeability index.

FIGURE 3. Course of the patient after OHCA caused by Brugada syndrome. CPR = cardiopulmonary resuscitation, ED = emergency department, EICU = emergency intensive care unit, IABP = intraaortic balloon pump, ICD = implantable cardioverter defibrillator, MTH = mild therapeutic hypothermia, OHCA = out-of-hospital cardiac arrest, PiCCO = pulse index continuous cardiac output, ROSC = return of spontaneous circulation.
defibrillated. However, no defibrillation was performed out of hospital in our case because the initial monitored heart rhythm of the patient was PEA, and bystander-performed CPR is more important than defibrillation for patients with a nonshockable rhythm.

Postcardiac arrest care has significant potential to reduce early mortality caused by hemodynamic instability, and later mortality and morbidity from multorgan failure and neurologic function damage. MTH, as a critical component of postcardiac arrest care, is an important resuscitation treatment that improves neurologic outcomes and gives a better chance of survival to discharge in survivors with cardiac arrest. Our case also showed that MTH after ROSC greatly improved the neurological outcome in the patient. A difference between our case and earlier studies is that our patient was maintained with a temperature between 34°C and 36°C. Although therapeutic hypothermia (targeted temperature from 32°C to 34°C) is now recommended in international resuscitation guidelines, a recent international multicenter trial demonstrates that a targeted temperature of 36°C has comparable survival rate and neurologic function of OHCA patients, compared with the temperature of 33°C.

Hyperthermia should be avoided following OHCA because it is associated with worse outcome. However, shivering is common and may raise body temperature when inducing MTH. Failure to control shivering is a common reason for delays in achieving target temperatures. Our patient was persistently shivering after ROSC and early phrase of MTH; it took us long time with sedation to control and achieve the target temperature.

The complex hemodynamic instability, including hypovolemia, myocardial stunning, excessive vasodilation, and pulmonary edema, is common after ROSC, which is associated with increased mortality. Therefore, it is important to monitor and evaluate hemodynamic variables in patients after ROSC under normothermic and hypothermic conditions. In our case, hypotension, heart dysfunction, and pulmonary edema occurred soon after ROSC and during the cooling procedure. We monitored cardiopulmonary function and hemodynamic parameters using the PiCCO monitoring system (Table 1), and improved the cardiopulmonary function using IABP and diuretics, all of these measures are very important in goal-directed fluid management patients after OHCA.

The diagnosis and treatment of the cause of underlying disease is fundamental to the management of OHCA. Brugada syndrome is diagnosed in our case with typical electrocardiogram findings (coved ST elevation in leads V1–V2) and family history. An operation of ICD implantation was successfully performed for the patient, and he showed complete recovery in the follow-up examinations.

In conclusion, our case demonstrates that even after an OHCA with prolonged time until ROSC, survival with a favorable neurologic outcome is possible, provided implementation of an extremely effective rescue chain.

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