

Case Report

Intravascular heat exchange technology successfully treats a patient after ultra-long cardiopulmonary resuscitation: a case report

Xiuli Zou, Tiejun Wu, Lina Zhang, Xihong Zhang, Hui Tian

Intensive Care Unit, Liaocheng People's Hospital, Shandong 252000, China

Received December 11, 2014; Accepted February 6, 2015; Epub February 15, 2015; Published February 28, 2015

Abstract: Objective: To investigate the effect of intravascular heat exchange in saving the lives of patients after cardiopulmonary resuscitation. Methods: The clinical data of a female patient who was given cardiopulmonary resuscitation for 1.67 h in July 2014 in our hospital was retrospectively analyzed. An intravascular heat exchange procedure was performed on the patient to control her body temperature. Comprehensive treatment measures also included mechanical ventilation, vasoactive agents, and osmotic dehydrating agents to reduce the elevated intracranial pressure, improving cerebral metabolism and anti-infective therapy. Results: After providing an intravascular heat exchange for 6 h, the patient's eyes automatically opened but the patient was unconscious. After 28 h, the patient became conscious. The breathing machine was withdrawn after 2 d and the vasoactive agents were stopped after 5 d. The patient was discharged after 17 d. Conclusion: Intravascular heat exchange could be an important complementary treatment to cardiopulmonary resuscitation.

Keywords: Vascular heat exchange technology, hypothermia, cardiopulmonary resuscitation

Introduction

During cardiac arrest, cardiac contraction and pump functions of the patient suddenly stop, interrupting the body's normal cycle and causing severe systemic ischemia and hypoxia. Studies have shown that 14-40% of patients with cardiac arrest return to spontaneous circulation after cardiopulmonary resuscitation (CPR) for continued treatment in the hospital, but most can suffer from brain dysfunction resulting from severe ischemia and hypoxia of the brain tissue after cardiac arrest [1] and might also have sequelae because of damage to the nervous system, even after they are discharged in good condition. In July 2014, a delivery woman was successfully resuscitated through endovascular cooling using heat exchange catheters. After CPR had been performed for 1.67 h. The incident is reported here:

Clinical data

The patient was a 36-year-old female who was given a lower uterine-segment cesarean sec-

tion in the Shen County People's Hospital because of fetal distress at 37 weeks of gestation. Four hours after the surgery, the patient complained of dizziness, followed by loss of consciousness, cardiac and respiratory arrest, and urinary incontinence. CPR was performed immediately. Thrombolytic therapy and electrical defibrillation were also performed because the doctors suspected that a massive pulmonary embolism might occur. During treatment, the sinus rhythm was restored but difficult to maintain until after 1.67 h of CPR, when the autonomous heartbeat could be maintained. The patient was then transferred to the Intensive Care Unit of Shandong Liaocheng People's Hospital with an oral trachea cannula, transport ventilator-assisted breathing, and continuous intravenous infusion of norepinephrine to maintain blood pressure.

Physical examination on admission

On admission, the patient was given a physical examination. The results were as follows: temperature, 35.9°C; pulse rate, 120 beats/min; respiration, 20 beats/min (mechanical ventila-

tion); and blood pressure, 89/45 mmHg. The patient was unconscious with a Glasgow Coma Score (GCS) of 3 and was unresponsive to examination. In addition, the size and shape of the patient's pupils were 5 mm in diameter, pupillary reaction to light was slow, and slight die blausucht occurred. She presented with abnormal breathing, redness of the sternum skin, irregular heartbeat, wet rales in both lungs, low and blunt heart sounds, and maintained by intravenous infusion of norepinephrine. There was abdominal distension; the dressing on the midsection of the lower abdomen was dry, indicating no bleeding; and there was no swelling of the lower limbs. Bilateral Babinski sign of the lower limbs was not elicited.

Rapid bedside detection in ICU

The measure of arterial blood gas revealed the following: pH, 7.127; partial pressure of carbon dioxide, 37.8 mmHg; partial pressure of oxygen, 97.3 mmHg; hemoglobin, 9.1 g/dL; hematocrit, 0.28; lactate, 7.1 mmol/L; bicarbonate, 12.0 mmol/L; actual base excess, -16.0 mmol/L; glucose, 15.5 mmol/L; procalcitonin, (PCT) 3.5 ng/mL; troponin, > 2000 ng/L; and pro-brain natriuretic peptide 656.7 ng/L. The biochemical tests showed the following: prothrombin time, 17.5 s; activated partial thromboplastin time, 44.0%; international normalized ratio, 1.61; fibrinogen, 0.51 g/L; DD dimer, 89.9 mg/L; fibrinogen degradation products, 182.1 mg/L; MI three CK-MB, 89.40 ng/mL; troponin I, 10.32 ng/mL; myoglobin, 2615.6 ng/mL; ALT, 518 IU/L; AST, 557 IU/L; blood urea nitrogen, 13.50 mmol/L; and creatinine, 354 μ mol/L. The pulmonary angiography and chest computed tomography (CT) routine scan demonstrated that the patient had a pulmonary embolism and a sternal fracture.

After CPR, cesarean section, the sternum fracture and the thrombolytic therapy for pulmonary embolism, the patient was diagnosed with multiple organ dysfunction syndrome (MODS).

Treatment in the ICU

After being transferred to the ICU, mechanical ventilation was used to assist the patient's breathing, and norepinephrine was intravenously infused to maintain her blood pressure. Meanwhile, mannitol, glycerol, sodium chloride,

methyl prednisolone, and albumin were given for dehydration and to reduce intracranial pressure. Ganglioside and xinnaojing were used to improve brain metabolism. Cefoxitin was given to resist infection. Myocardiac drugs (such as phosphocreatine) and proton pump inhibitors were also given as symptomatic and supportive treatment. On the second day after admission, the patient had a GCS score of 3 with assisted mechanical ventilation. She was given 5.0 mg/h norepinephrine by intravenous infusion. Her temperature reached a high of 40.1°C, which might have been related to central factors such as central high fever and infection, and the PCT was 67 ng/mL during the review. Because of the high fever and possibility of infection, the antibiotic was changed to imipenem and teicoplanin. To reduce brain metabolism and protect brain cells, the patient underwent mild hypothermia therapy using vascular cooling-heat exchange technology. An intravascular cooling system (Thermogard XP 3000, ZOLL Circulation, Inc., San Jose, CA, USA) and intravascular temperature control catheter were used with the consent of her family and a signature from an authorized person. The temperature-control catheter (five lumens) with an indwelling depth of 32 cm was placed in the right femoral vein and into the inferior vena cava; placement was confirmed by abdominal X-ray. Body temperature, monitored using a probe in the bladder, was 39.8°C. The target temperature was then set to 35.0°C, the cooling system set to "fever" mode, and the cooling rate set to 1.0°C/h. After 5.0 h, the body temperature decreased to the target temperature. The cooling process went smoothly with no obvious chills, and the circulatory system showed no obvious fluctuations. After 6.0 h of vascular heat exchange, the patient opened her eyes, became restless, and was uncooperative. At that point, the target temperature was set to 34°C, and the patient was given appropriate sedation. After 28 h of vascular heat exchange, the patient appeared to be in a clear state of mind and was able to follow instructions. To reduce the complications of hypothermia therapy, the temperature was slowly increased. The target temperature was set to 36°C, and the rewarming rate was 0.5°C/h. During the rewarming period, patient awareness, blood pressure, and blood clotting function did not change remarkably. On July 27, the vascular heat exchange hypothermia therapy

was stopped. The patient's body temperature again rose to 37.9°C and intermittent intravascular cooling therapy was administered. Norepinephrine was reduced to 3.0 mg/h, and the ventilator parameters were decreased. At 9:00 a.m. on July 28, the bedside ultrasound examination of the patient revealed no deep vein thrombosis of the right lower limb. The temperature-control catheter was removed from the femoral vein of the right lower limb and mechanical ventilation was stopped, followed by removal of the orotracheal intubation tube. Non-invasive mechanical ventilation was initiated and the norepinephrine dosage was further reduced until stopped on July 28. On July 30, the examination showed that the brain edema was not aggravated by stopping vascular cooling, and there was no ischemia-reperfusion injury. The dosage of drugs for dehydration was reduced. Two consecutive blood cultures were negative, indicating no bacteria obtained from the culture. The white blood count was $11.29 \times 10^9/L$, and the neutrophil ratio was 92.8%. The antibiotic was changed to cefoperazone-sulbactam in combination with teicoplanin. On August 1, non-invasive mechanical ventilation was replaced with oxygen inhalation through a nasal cannula. On August 5, the patient was moved to a general ward. The blood, kidney function, and blood coagulation indices were normal. On August 12, the patient was discharged. After 2 weeks, the follow-up study showed no discomfort. The blood analysis and biochemical tests were conducted again and the results were normal.

Discussion

Cardiopulmonary arrest is threatening to human life. The data from CPR database of the United States showed that 47% of patients return to spontaneous circulation after CPR, among whom only 18% survive, 19% quit the treatment and up to 63% die of MODs [2]. The majority of patients after resuscitation is in a coma or has different degrees of neurological dysfunction. Only 20-30% of patients are discharged alive without any neurological dysfunction [3]; therefore, whether cerebral resuscitation is successful is the key to the survival and the patient's quality of life after resuscitation. It took 1.67 h for our patient to recover after CPR. The ischemic cell dysfunction can lead to release of excitotoxicity factors, such as an

increase in the release of acid toxic factors. An electrolyte imbalance could result in apoptosis, perfusion, and recanalization and can cause rapid production of free radicals, leading to damages to proteins, lipids, and DNA and the destruction of mitochondria, causing death and apoptosis of reperfused cells [4]. Endovascular hypothermia therapy can decrease a patient's temperature to the target temperature of 32-34°C in a short period of time [5], which can inhibit the release of toxic factors during ischemia, reduce the production of free radicals during reperfusion, and decrease brain oxygen consumption, protecting cells, restoring the activity of cells undergoing apoptosis gradually, and reducing the production of free radicals thereby reducing ischemia-reperfusion injury [6].

Vascular cooling heat exchange is a new cooling method developed in recent years that has been used widely in medical institutions throughout Europe and many other places. The cooling system includes an extracorporeal device with a cooling effect, and a catheter that can be inserted through the femoral vein and into the inferior vena cava below the heart for heat exchange. After a target temperature is set, cold saline solution is pumped into the inflow conduit using the Thermogard XP system and into the three lumens attached to the catheter, in full contact with the blood in the inferior vena cava for heat exchange, and returned back to the Thermogard XP system through the outflow conduit in the catheter. The core temperature is measured using a probe placed in the bladder and transmitted to the Thermogard XP system for software analysis. Based on the results, the temperature is adjusted and the temperature of the saltwater used in the heat exchange cycle is controlled. The Thermogard XP temperature control system and the catheter constitute a closed-circulation system; therefore, the cold saltwater cannot flow into the circulating blood of the patient. Vascular heat exchange is a revolutionary cooling technology characterized by a simple operation, accurate measurement, fast cooling, ease of controlling the rewarming rate, and good performance for maintaining the default temperature with small fluctuations, all of which are better characteristics than those of other traditional cooling methods [7]. Because the catheter can be programmed to automatically

execute the cooling process according to protocols, medical staff do not need to constantly monitor the procedure, which greatly reduces its workload. No serious complications have been reported with this procedure. It can accurately control the body's core temperature to the arbitrarily set target temperature in many models, such as hypothermia (32-35°C), antipyretic therapy (36.5°C), and rewarming, thus the technique for cerebral resuscitation is important in the treatment of critically ill patients with conditions such as cardiac arrest, acute stroke, traumatic brain injury, malignant hyperthermia, heat stroke, status epilepticus, and acute myocardial infarction [8], and has a high clinical value in saving the lives of these patients.

Disclosure of conflict of interest

None.

Address correspondence to: Dr. Tiejun Wu, Intensive Care Unit, Liaocheng People's Hospital, Shandong 252000, China. Tel: +86-13869530330; Fax: +86-021-64085875; E-mail: tiejunwu@hotmail.com

References

- [1] Chin JY, Koh Y, Kim MJ, Kim HS, Kim WS, Kim DS, Kim WD and Lim CM. The effects of hypothermia on endotoxin-primed lung. *Anesth Analg* 2007; 104: 1171-1178.
- [2] Nolan JP, Laver SR, Welch CA, Harrison DA, Gupta V and Rowan K. Outcome following admission to UK intensive care units after cardiac arrest: a secondary analysis of the ICNARC Case Mix Programme Database. *Anaesthesia* 2007; 62: 1207-1216.
- [3] Nadkarni VM, Larkin GL, Peberdy MA, Carey SM, Kaye W, Mancini ME, Nichol G, Lane-Truitt T, Potts J, Ornato JP, Berg RA; National Registry of Cardiopulmonary Resuscitation Investigators. First documented rhythm and clinical outcome from in-hospital cardiac arrest among children and adults. *JAMA* 2006; 295: 50-57.
- [4] Nielsen N, Wetterslev J, Cronberg T, Erlinge D, Gasche Y, Hassager C, Horn J, Hovdenes J, Kjaergaard J, Kuiper M, Pellis T, Stammedt P, Wanscher M, Wise MP, Åneman A, Al-Subaie N, Boesgaard S, Bro-Jeppesen J, Brunetti I, Bugge JF, Hingston CD, Juffermans NP, Koopmans M, Køber L, Langørgen J, Lilja G, Møller JE, Rundgren M, Rylander C, Smid O, Werer C, Winkel P, Friberg H; TTM Trial Investigators. Targeted temperature management at 33 degrees C versus 36 degrees C after cardiac arrest. *N Engl J Med* 2013; 369: 2197-2206.
- [5] Nolan JP and Soar J. Mild therapeutic hypothermia after cardiac arrest: Keep on chilling. *Crit Care Med* 2011; 39: 206-207.
- [6] Choi HA, Badjatia N and Mayer SA. Hypothermia for acute brain injury—mechanisms and practical aspects. *Nat Rev Neurol* 2012; 8: 214-222.
- [7] Keller E, Imhof HG, Gasser S, Terzic A and Yonekawa Y. Endovascular cooling with heat exchange catheters: a new method to induce and maintain hypothermia. *Intensive Care Med* 2003; 29: 939-943.
- [8] Schmutzhard E, Engelhardt K, Beer R, Brossner G, Pfausler B, Spiss H, Unterberger I and Kampfl A. Safety and efficacy of a novel intravascular cooling device to control body temperature in neurologic intensive care patients: a prospective pilot study. *Crit Care Med* 2002; 30: 2481-2488.