

Successful treatment of a young woman with acute complicated myocardial infarction

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Abstract

Therapeutic hypothermia is method used to improve the neurological status of patients who are at risk of ischaemia after myocardial infarction. We report a case of a 28-year-old woman who suffered acute myocardial infarction complicated by ventricular fibrillation. The patient was successfully resuscitated. Invasive and non-invasive medical treatment was applied including therapeutic hypothermia. Success was achieved due to adequate public reaction, fast transportation, blood vessel revascularization and application of therapeutic hypothermia. The patient was successfully discharged after one week of treatment, and just minor changes in heart function were present.

Key words: cardiac arrest, ventricular fibrillation, therapeutic hypothermia.

Introduction

Cardiac arrest (CA) is one of the most common causes of death in developed countries [1]. Complications related to myocardial infarction (MI) (such as acute heart failure, pericarditis, ventricular aneurysm, and ventricular rupture with tamponade), cardiac rhythm and conduction disorders are considered to be among the most common ones. Ventricular fibrillation (VF) is a dangerous complication of MI that causes sudden cardiac arrest and clinical death [2].

Cardiac arrest is the leading cause of death in Europe and the USA, affecting about 750 000 people annually. Due to improved public training of cardiopulmonary resuscitation (CPR) and advances in professional emergency medical response, the rate of restoration of spontaneous circulation (ROSC) has risen in the past decades [3]. However, being subject to a variety of recovery measures, the survival rate after cardiac arrest remains low (6–2% in the USA) [4]. The post-cardiac arrest syndrome, once systemic ischaemia develops, is one of the leading causes of death in resuscitated patients [1, 5, 6]. After

cardiac arrest, there is a high probability that the resuscitated patient will have some neurological deficits; therefore, the usual resuscitation techniques are not enough. There is evidence that a better neurological outcome is achieved with therapeutic hypothermia [3–5, 7, 8].

According to 2013 ACCF/AHA and recent ST-elevation myocardial infarction (STEMI) European guidelines for the management of STEMI, therapeutic hypothermia is class I level of evidence B indication and should be started as soon as possible in comatose patients with STEMI and out-of-hospital cardiac arrest caused by VF or pulseless ventricular tachycardia (VT), including patients who undergo primary percutaneous coronary intervention (PCI) [3, 5, 9–11].

Aim

With this report, we want to show that mild therapeutic hypothermia induction and maintenance are possible with a device which is not dedicated for daily clinical use in such a situation. In such cases, induction of mild therapeutic hypothermia has demonstrated a huge impact on the final patient state.

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Case report

A 28-year-old woman was found unconscious at the airport. Cardiopulmonary resuscitation was started by witnesses and took approximately 15 min. Duration from sudden cardiac arrest (SCA) to CPR is not exactly known (supposedly ~15 min because she was already found unconscious). Then specialized resuscitation (including 3 defibrillation impulses due to ventricular fibrillation) was applied until ROSC was attempted. In total, the CPR took approximately 30 min and SCD-ROSC took about 45 min. For the following investigations and treatment the patient was emergently admitted to our university hospital with suspicion of acute coronary syndrome. Full medical history of the patient was obtained from her mother. She revealed that during childhood ductus arteriosus was diagnosed and soon after, it was closed. The mother denied pregnancy of her daughter, usage of any kind of medication, alcohol, nicotine or narcotic agents. Recently, increased blood pressure was noticed – 170/100 mm Hg. Before the flight, the patient was anxious, because it was the first flight in her life. The family history revealed that her grandfather (mother's side) died at 39 years of age due to an acute myocardial infarction. Other family relatives have no clinical evidence of any cardiovascular disease.

Physical examination at the intensive care unit showed that the patient was of normal constitutional type – body mass index (BMI): 23 kg/m², unconscious – Glasgow coma scale (GCS) with a score of 3. She was intubated and ventilated, with stable hemodynamic: blood pressure 143/86 mm Hg, pulse 68 beats per minute. An electrocardiogram (ECG) showed sinus rhythm, and ST elevation in the frontal and lateral walls (Figure 1). Due to the diagnosis of acute coronary syndrome (ACS) the

patient was immediately transferred to the heart catheterization laboratory. Percutaneous coronary intervention was performed after 160 min since her spontaneous circulation was restored at the airport after resuscitation. Coronary angiography showed occlusion of the left anterior descending (LAD) proximal segment which was successfully treated with bare metal stent implantation with the diameter of 3.0 mm × 14 mm (Figure 3). After the procedure the patient was transferred back to the intensive care medicine (ICU) for further medical treatment and continuous artificial lung ventilation (ALV).

Heart ultrasound demonstrated severe reduction of left ventricular EF (20–25%) with suspected left ventricular aneurysm in the apex. Due to the bad neurologic status, an urgent clinical neurological investigation and head computed tomography (CT) scan was scheduled and performed. There were no clinical pathological symptoms, and the CT scan did not show any acute focal brain pathology. In this case the condition with a low score of GCS was determined by hypoperfusion of the brain. Laboratory workup revealed the following results: glucose, troponin I, BNP and D-dimer were elevated; blood gas was in the normal range; and no deviation of protein C and S homocysteine concentration was found (Table 1).

Five hours after admission to the ICU, it was decided to perform mild therapeutic hypothermia cannulating the femoral vein and right internal jugular vein, in order to start cooling with a cardiopulmonary bypass (CPB) machine, because the special dedicated machine was not present at that time. The target temperature was set to 33°C. All of the procedures were performed according to the local therapeutical hypothermia (MTH) protocol: the required temperature was achieved within 2 h – decreasing 2°C per hour, which remained stable during the cooling time

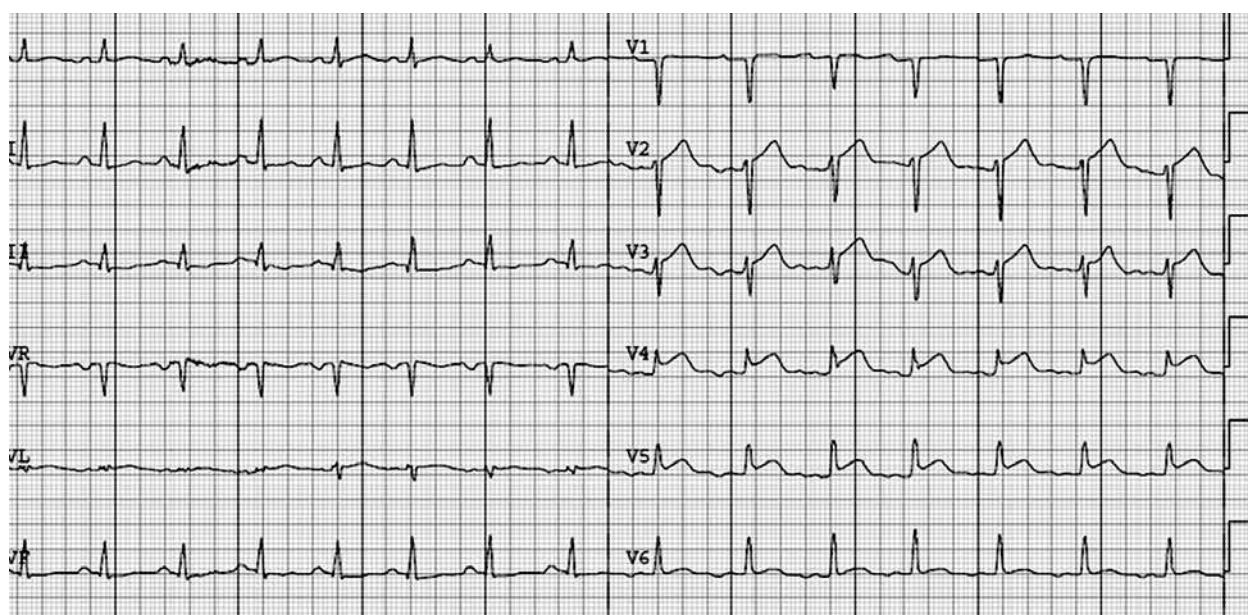


Fig. 1. Patient ECG upon arrival at the hospital: sinus rhythm, ST elevation in V2–V5 derivations

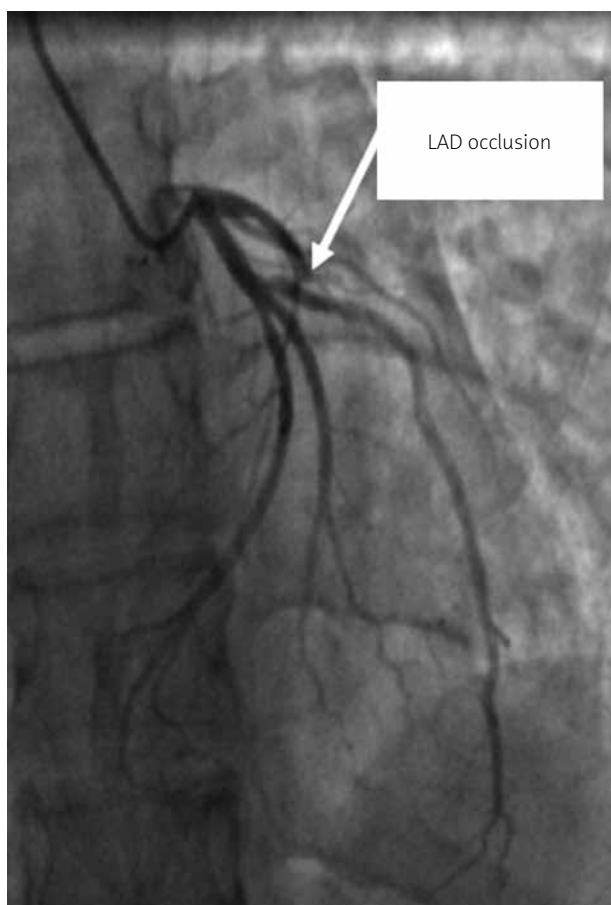


Fig. 2. Coronarography: white arrow shows occlusion of the left anterior descending coronary artery



Fig. 3. Coronary angiograms after PCI: successful revascularization, no residual stenosis

of 24 h. The patient's consciousness was suppressed by midazolam and propofol continuous infusion. Shivering prevention was achieved by continuous rocuronium infusion. After 2 h, when the target temperature of 32.6°C was reached, hemodynamic instability was present followed by ventricular fibrillation complicated by cardiogenic shock. This situation was successfully treated according to the VT treatment algorithm [11]; additionally vasopressors – adrenalin ~ 0.17 µg/kg/min, noradrenalin 0.28 µg/kg/min – and intra-aortic balloon counter pulsation (IABP) were introduced. Also body temperature was increased from 32.6 to 33.5°C.

After 24 h of MTH delivery, it was decided gradually to warm up the patient to the normal body temperature of 36°C, applying 0.5°C per hour, and still ALV and IABP were retained. There was an anaemic state of the patient, which was caused by the mechanical effect of the cooling device to red blood cells. Moreover, there were various procedures carried out, such as PCI, intra-aortic balloon counter pulsation introduction, etc., due to which the patient was able to lose blood. This condition was corrected with red blood cell transfusion. After 2 days of artificial ventilation, the patient started to breathe normally, and the lactate concentration decreased from 6 mmol/l to

Table 1. Laboratory workup

Laboratory data	Normal value	Results
Haemoglobin	120–150 g/l	130 g/l
Glucose	3.3–5.5 mmol/l	19.37 mmol/l
Troponin I	< 0.05 µg/l	41.918 µg/l
Creatinine	23–97 µmol/l	68 µmol/l
C-reactive protein (CRP)	< 0.5 mg/l	58.2 mg/l
BNP	< 400 ng/l	790.0 ng/l
SPA (INR)	0.9–1.12	1.11
D-dimer	< 250 µg/l	880 µg/l
Potassium	3.8–5 mmol/l	3.5 mmol/l
AST (GOT)	0–35 U/l	168 U/l
ALT (GPT)	0–35 U/l	173 U/l
Cholesterol	2.6–5.2 mmol/l	2.47 mmol/l
Triglycerides	0–1.95 mmol/l	0.89 mmol/l
HDL-cholesterol	1–2.2 mmol/l	0.84 mmol/l
LDL-cholesterol	0–2.6 mmol/l	1.22 mmol/l

1.4 mmol/l within a day. Kidney function was regular, with sufficient diuresis. When the hemodynamic state was stable, IABP was removed, and β-blockers were launched. The next morning, the patient's condition was stable:

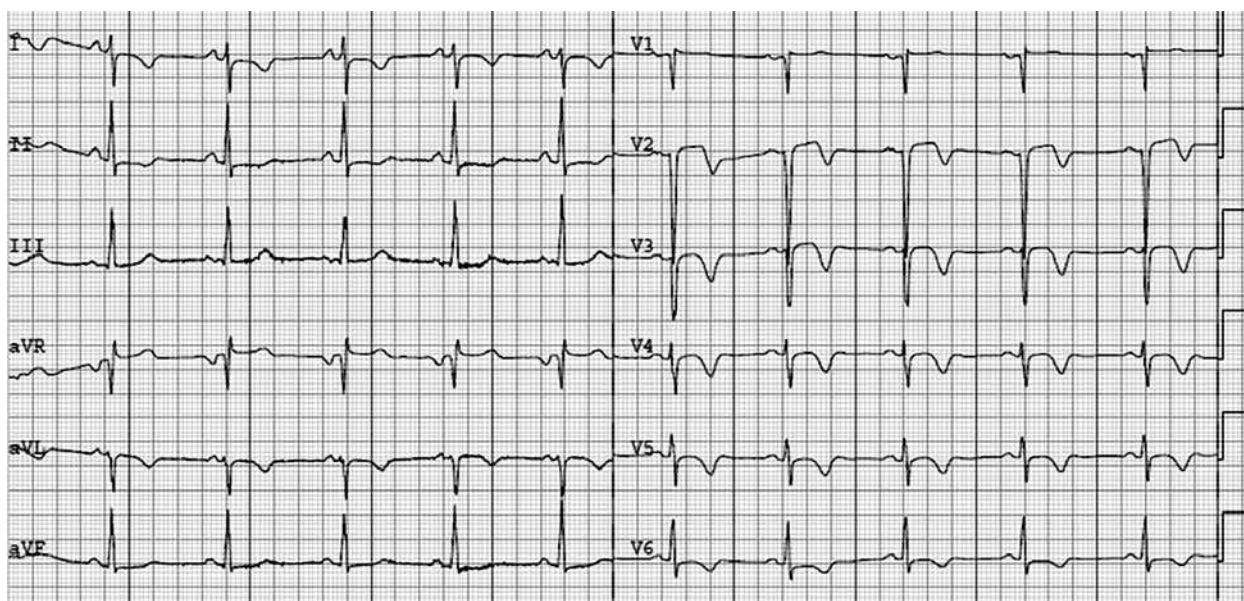


Fig. 4. Patient's ECG after discharge from hospital: sinus rhythm, QS V1–V2, T(–) I, aVL, V2–V6

conscious, easily disoriented. Hemodynamic parameters were within the normal ranges, without the need for adrenomimetics. Lung congestion was treated with small doses of loop diuretics.

After eight days of such a complicated condition, the patient was responsive to the surroundings. Blood pressure 118/67 mm Hg, heart rate – 86 beats per minute. ECG: sinus rhythm, QS V1–2, T(–) I, aVL, V2–V6 (Figure 4). Heart ultrasound demonstrated left ventricular contractility with the left ventricular ejection fraction (EF) of > 55% with residual slightly hypokinetic front wall middle-apical segments. Conservative treatment was continued and after 3 days the patient was transferred to another hospital for further treatment. According to the cerebral performance category (CPC), the patient was discharged with the score of 1. She was conscious, alert, and able to work and lead a normal life, without any psychological or neurological deficits. After the rehabilitation period, she returned to her previous work, to the same position – a social worker.

Four months after the discharge heart MRI showed slightly dilated left ventricle with hypokinetic septum and anterior wall. Late contrast gadodiamide enhancement sequence showed worse contrast accumulation in anterior wall and septum, with transmural scar at 17th segment, with preserved left ventricular ejection fraction – 57%.

Discussion

Cardiac arrest disrupted the whole body blood circulation, which develops as a result of ischemic processes [7]. Despite the pharmacological and invasive treatment in order to provide optimal neurological function, it is recommended to provide therapeutic hypothermia for at least 24 h [5, 8, 12, 13].

Therapeutic hypothermia is an effective treatment method not only to improve the neurological status of

patients with ischemic risk [1, 5–10, 12, 14, 15], but also to reduce mortality after myocardial infarction causing life-threatening complications (such as VF or VT). The principle of therapeutic hypothermia is to cool down the body. Within 12–24 h, the body is cooled to 32–34°C [1, 3–5, 7, 10, 12, 13, 16], which is normally the perfect balance between the positive clinical effects and complications [4]. On the cooling protocol the average temperature decrease of conventional cooling has to be 0.3°C to 2.5°C per hour, depending on the method; however, cooling should not be prolonged and must be as fast as possible to reach the target temperature [3]. After 24 h, the patient should be slowly and accurately warmed up [4, 16]. The temperature is increased from 0.25°C to 1°C each hour [4, 6]. The process should take about 8 [4] or 12 h [12]. The warming process can be done in various ways. It can either use warm blankets, blowing warm air, or patients may be left to warm up by themselves upon termination of MTH [4, 6]. In this clinical case, the patient was thawed gradually with CPB increasing the temperature to 36°C by 0.5°C. Most patients die in the heating phase due to lack of diligence. Common complications include arrhythmias, electrolyte imbalance, increased clotting, increased intracranial pressure, hyperglycaemia, hypoglycaemia, pneumonia or other opportunistic infections [2, 4–7, 10, 12, 16], which become unpredictable for 72 h after the procedure [5, 12]. Such complications were avoided in this case.

Knowing the principle of MTH it is important to remember when to use it. According to American Heart Association (AHA) guidelines, therapeutic hypothermia should be carried out as soon as possible after recovery and after the opening of blood vessels for spontaneous circulation recovery [1, 3, 5, 7, 8, 13, 15]. The optimal therapeutic window is 6 h [4, 7, 8, 16]. There are studies

suggesting that earlier hypothermia is more effective; it is proposed to begin during the resuscitation [1]. There are protocols that can be used to help accurately gather information and assess the patient's condition. For example the National Polish Mild Hypothermia Registry run by the Polish Society of Cardiology provides a validated protocol on intravascular and external hypothermia [17]. In this case, the MTH protocol which is approved by our university hospital has the following inclusion criteria: cardiac arrest with return of spontaneous circulation (ROSC) in or out of hospital, any gender, initial rhythm of ventricular fibrillation or pulseless ventricular tachycardia, age ≥ 18 , unresponsive after ROSC, head CT scan without acute intracranial process, systolic blood pressure can be maintained ≥ 90 mm Hg with stability (including with pressures, fluids, meant to exclude patients in shock). Exclusion criteria: pregnancy, all women of age ≤ 55 need a negative urine or serum β -HCG, another reason is being comatose (comatose at baseline, drug overdose, head trauma, stroke, status epilepticus) – consider head CT where appropriate, hypothermia (temperature $< 30^\circ\text{C}$) before cooling efforts begin, known coagulopathy or bleeding problem, IVC filter – excluded from long cooling catheter (Icy) must use shorter cooling catheter (Cool-Line) via IJ or subclavian approach.

Therapeutic hypothermia techniques and methodology:

1. Internal – invasive:

- a) cooling catheter – the catheter is introduced into the femoral vein. Cold saline solution (at 4°C 2 l 0.9% NaCl, Ringers) is injected, which circulates the tubing and cools the blood. The temperature is reduced from 1.5°C to 2°C per hour or 4°C every 30 min (different sources use different techniques). Optionally, the body can be cooled directly by injecting cold fluids intravenously. This level of accuracy allows doctors to avoid many pitfalls associated with excessively deep levels of hypothermia. Furthermore, catheters can raise the temperature at a steady rate, which helps avoid harmful rises in intracranial pressure. Catheter-based temperature management has been shown to provide faster, more precise and more efficient cooling compared to all external methods. However, one disadvantage remains – this technique requires a well-trained professional. The potential complications include bleeding, pulmonary oedema, infection, deep vein thrombosis, and vascular punctation – a blocked femoral artery is necessary for the other procedures, such as angiography [1, 4–8, 10, 13, 15, 16, 18];
- b) trans-nasal cooling – or RhinoChill method – is easier to perform, is effective at cooling a patient's brain (at about 2.6°C per hour) and body (1.6°C per hour), and provides the means of continuously cooling the patient throughout the early stages of therapeutic hypothermia and while continuing

the treatment in hospital. The device is a novel technology that uses two small cannula, inserted into a patient's nasal cavity, to deliver a spray of coolant mist that evaporates directly underneath the brain and the base of the skull. This cooling method can be started even before the return of blood circulation, and very shortly after the point of cardiac arrest [10, 19].

2. External – non-invasive:

- a) cooling blankets, torso vest, leg wraps, wet towels – this is a generalized way of lowering the whole body temperature. Such a technique can cover up to 70% of the patient's surface area. Onlays are in direct contact with the patient's skin. Two litres of cold liquid cools the body to 33°C for 65 min. The advantage of this method is that it does not require any special preparation of the invasive procedure. The equipment is easily available and can be started on the way to the hospital. The downside is that it is difficult to control the temperature, which can lead to excessive cooling of $< 32^\circ\text{C}$. The skin should be monitored every 2–6 h. There is a possibility that the external cooling blankets can cause significant burns to the patient's skin, and have a slower induction time versus internal cooling [1, 4–7, 10, 13, 15, 20];
- a) ice packs – this method is inexpensive and represents an appropriate way to initiate cooling. However, it can be messy and is less than optimal in the rate of cooling and target temperature maintenance. Practically, ice packs are placed in anatomic areas that have a large heat-exchange capability (the head, neck, armpit, and groin) and are replaced when they have substantially melted. In addition to ice packs, evaporative cooling with fans has been used. The average temperature drop using ice packs is moderately slow and highly variable and is reported to be 0.03 – 0.98°C per hour [1, 4, 6, 7, 10, 13, 16];
- a) chilled caps – it is a local method that only cools the head. A particular hat is filled with coolant agent such as ice or gel (-25°C to -30°C). Their most notable uses are in preventing or reducing alopecia in chemotherapy, and for preventing cerebral palsy in babies born with hypoxic ischemic encephalopathy. Since the hat quickly warms up, it must be replaced every 20–30 min [1, 4];
- a) cooling collars – the most up-to-date method, which is excellent in urgent situations. It is simple and easy to transport in a patient. A cooling fluid resides in the collar. This technique requires only a single use of the coolant and is non-hazardous. The brain gets cooled down by 1.7°C in less than 26 min [10].

More than five reviewed articles mention two clinical studies proving positive effects of TH in people revived

after cardiac arrest that occurred due to ventricular fibrillation. Patient's neurological symptoms improved and the survival rate increased by six months. The first clinical study was conducted in Europe. Patients were divided into two groups. One group consisted of patients who received TH and in the other group – the control group – TH was not used. All patients received resuscitation after cardiac arrest, and they were restored to spontaneous circulation. Patients were cooled outside the method – by cooling blankets and ice packs if necessary (33°C within 24 h). Significant differences between groups of complications were not seen. The second clinical study was conducted in Australia. Chilling involved ice packs and the desired therapeutic temperature was achieved within 12 h [4, 6–8, 13, 21].

Other studies confirm the aforementioned clinical results. Christian Storm and colleagues conducted a clinical study where the subjects were revived after the cardiac arrest. Intravenous therapeutic hypothermia was applied immediately after the external cooling [15]. Another clinical study was performed in Europe. A particular cooling method was chosen individually by each specialist. One group underwent invasive hypothermia with cooling catheters and another one underwent external therapeutic hypothermia using ice packs and cooling blankets. Efficacy was unambiguously positive, regardless of the method chosen by a doctor. This study confirms the safety of therapeutic hypothermia and efficiency, enabling the application of this therapy more often than it is mentioned here [8]. Dr. Ranka *et al.* conducted a clinical study where one group was assigned a rapid infusion of Ringer's solution at 4°C followed by surface cooling using ice or cold packs. The other group was hooked up to the water circulating external cooling device named Hilotherm Clinic. The speed of the cooling by the water-circulating system was significantly higher compared to conventional cooling. Furthermore, the temperature deviation was significantly lower with Hilotherm compared to the conventional cooling. However, there were no significant differences in the mortality between the groups within the 30 days in the hospital [3].

Singapore doctors Chin and Wong described the application of another clinical study, which is slightly different from those mentioned before. The study consisted of patients who were revived after cardiac arrest, and held on asystole or electromechanical dissociation. Therapeutic hypothermia was performed using cooling helmets surrounding the head and neck. Therapeutic temperature of 34°C was achieved after 4 h [7]. The benefits of therapeutic hypothermia after electromechanical dissociation or asystole are not sufficiently proven [5].

Conclusions

Therapeutic hypothermia is a successful and important neuroprotective measure for patients who experienced pulseless arrhythmia, ventricular fibrillation and cerebral

hypoperfusion. This therapy reduces ischemic neurological damage and the mortality rate in patients after resuscitation. In most of the articles, based on the long-term and proven practices, there are references to special freezing catheters, a cooling blanket or ice packs. However, as in this clinical case, when there is no possibility to use a special, easily adjustable device, the option is to use a cardiopulmonary bypass machine that has provided the opportunity to successfully save the life of a young patient.

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