Case report

Prolonged mechanical CPR of a 48-year old male patient in severe hypothermia conducted in the emergency department – case report

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ABSTRACT

Introduction: Hypothermia is still one of the major problems of modern emergency medicine. It causes reduction in oxygen consumption by brain tissue, which has neuro- and cardio protective effect. Most of the time, severe hypothermia leads to prolonged resuscitation resulting in decreased quality of cardiopulmonary resuscitation (CPR) due to the rescuers fatigue.

Aim: The aim of this paper is to introduce the case of prolonged resuscitation with the use of mechanical device, conducted in hypothermic patient.

Case study: We report a case study of 48-year-old male in severe hypothermia (19°C) and active gastrointestinal bleeding. We have conducted prolonged CPR for 142 minutes together with noninvasive core warming techniques that resulted in conversion of pulseless electrical activity to ventricular fibrillation and achievement of return of spontaneous circulation. Despite proper treatment, patient died next day in Intensive Care Unit due to the multi-organ failure.

Results and discussion: Cardiac arrest in case of severe hypothermia can lead to survival with good neurologic outcome, however prolonged cardiac arrest results in hypoxic brain injury and severe neurological dysfunction. It is crucial to initiate effective chest compressions to maintain minimal cerebral blood flow. Mechanical devices can be implemented in such situations in order to provide efficient CPR.

Conclusions: Cardiac arrest due to hypothermia can lead to extension of resuscitation. To improve survival of patients in situations requiring prolonged resuscitation, mechanical devices performing chest compressions should be implemented. It is possible to successfully warm up hypothermic cardiac arrest patients through noninvasive methods.
1. INTRODUCTION

Identification and treatment of reversible causes of sudden cardiac arrest (SCA) are considered to be crucial interventions in the algorithm of advanced life support (ALS). Potentially reversible causes are divided into two groups: 4Hs and 4Ts (Table 1).

Table 1. Reversible causes of cardiac arrest.

<table>
<thead>
<tr>
<th>4Hs</th>
<th>4Ts</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypoxia</td>
<td>Tension pneumothorax</td>
</tr>
<tr>
<td>Hypo- or hyperkalemia and other</td>
<td>Tamponade</td>
</tr>
<tr>
<td>electrolyte disorders</td>
<td></td>
</tr>
<tr>
<td>Hypo- or hyperthermia</td>
<td>Thrombosis (coronary and pulmonary)</td>
</tr>
<tr>
<td>Hypovolemia</td>
<td>Toxins (poisoning)</td>
</tr>
</tbody>
</table>

Hypothermia is still one of the major problems of modern emergency medicine. It is estimated that approximately 1500 patients suffer and die from accidental hypothermia in United States every year. Poland still lacks exact data on this entity. Hypothermia causes reduction in oxygen consumption which has a neuro- and cardio protective effect. In all hypothermic patients with no fatal illness nor lethal injury, cardiopulmonary resuscitation (CPR) should be implemented and conducted until patient's body is rewarmed. Most of the time, severe hypothermia leads to prolonged resuscitation resulting in decrease in quality due to the rescuers' fatigue. Mechanical devices can be introduced to the standard ALS algorithm while long-lasting CPR, to maintain high quality chest compressions.

2. AIM

The aim of this paper is to introduce the case of prolonged resuscitation with the use of mechanical device, conducted in hypothermic patient.

3. CASE STUDY

Patient, 48-year-old male, was brought by emergency medicine team (EMT) to emergency department (ED) of the Regional Specialist Hospital in Olsztyn, Poland. His neighbor found him unconscious in a garden-plot. The outside temperature didn’t exceed 6°C. Patient’s body was chilled and his clothing was covered in clotted blood. Rescuers managed to speak with the neighbors, who reported that the day before patient vomited the contents reminding of the coffee grounds. Additionally, patient had history of cerebral palsy, hypertension, heart failure NYHA IV and alcoholic cardiomyopathy. He was admitted to the hospital at 10:38 a.m. in severe condition, with peripheral cyanosis. After opening the airway, there were no signs of normal breathing, only agonal gasping. Due to the lack of pulse in the major arteries and the impossibility of detection of blood pressure (BP), cardiac arrest was diagnosed and CPR started immediately. At the same time, core body temperature was measured and determined at 19°C (at the level of lower one third of the esophagus). Simultaneously basic neurological examination was executed. Patient’s pupils were wide and almost not responding to light. On the Glasgow Coma Scale patient received 3 points. While starting CPR, patient was immediately connected to a monitor which showed heart rate of 20–30 bpm. In conjunction with the lack of pulse, mechanism of cardiac arrest was defined as pulseless electrical activity (PEA). Leading cause of cardiac arrest in this case was hypothermia. However, due to the presence of acidosis and hypovolemia, this patient manifested 3 of 8 reversible causes contributing to SCA.

ALS procedures were implemented immediately after recognizing cardiac arrest, according to the hypothermia protocol. In this case, we have promptly introduced both passive and active core rewarming techniques, such as covering patient’s trunk with the thermal blanket, infusing warmed fluids and bladder lavage with warm saline solution. After intubation patient was mechanically ventilated with 100% oxygen. Chest compressions were performed by load distributing band (LDB) mechanical device. After about 30 minutes of resuscitation, conversion of the rhythm to VF occurred. Therefore, a single defibrillation was performed. After delivering shock with the energy of 200 J, no return of spontaneous circulation (ROSC) was achieved and

Table 2. Venous/Arterial blood test results in time.

<table>
<thead>
<tr>
<th>Time</th>
<th>pH</th>
<th>pCO₂, mm Hg</th>
<th>pO₂, mm Hg</th>
<th>BE, mEq/L</th>
<th>Creatinine, mg/dL</th>
<th>Urea, mg/dL</th>
<th>APTT, s</th>
<th>INR</th>
<th>WBC, 10³/mL</th>
<th>RBC, 10⁶/mL</th>
<th>Hgb, g/dL</th>
<th>Hct, %</th>
<th>PLT, 10³/mL</th>
</tr>
</thead>
<tbody>
<tr>
<td>10:35 a.m. v</td>
<td>7.247</td>
<td>64.9</td>
<td>38.2</td>
<td>-0.5</td>
<td>0.8</td>
<td>56</td>
<td>58.8</td>
<td>3.62</td>
<td>1.82</td>
<td>4.25</td>
<td>10.6</td>
<td>34.9</td>
<td>22</td>
</tr>
<tr>
<td>12:05 p.m. a</td>
<td>7.258</td>
<td>42.2</td>
<td>173.7</td>
<td>-8.1</td>
<td>0.8</td>
<td>110</td>
<td>73.3</td>
<td>5.34</td>
<td>1.0</td>
<td>1.33</td>
<td>3.2</td>
<td>11.3</td>
<td>22</td>
</tr>
<tr>
<td>1:20 p.m. a</td>
<td>7.209</td>
<td>53</td>
<td>225.5</td>
<td>-6.8</td>
<td>0.8</td>
<td>96</td>
<td>61</td>
<td>4.24</td>
<td>2.19</td>
<td>3.2</td>
<td>5</td>
<td>16.2</td>
<td>11</td>
</tr>
<tr>
<td>3:45 p.m. a</td>
<td>7.339</td>
<td>40.1</td>
<td>294</td>
<td>-4.3</td>
<td>0.9</td>
<td>76</td>
<td>40.3</td>
<td>1.81</td>
<td>2.19</td>
<td>2.89</td>
<td>5</td>
<td>25.1</td>
<td>15</td>
</tr>
<tr>
<td>9:10 p.m. v</td>
<td>7.162</td>
<td>61.2</td>
<td>48.7</td>
<td>-6.8</td>
<td>0.9</td>
<td>76</td>
<td>41.7</td>
<td></td>
<td>1.09</td>
<td>2.89</td>
<td>8.2</td>
<td>25.1</td>
<td>15</td>
</tr>
<tr>
<td>5:35 a.m. v</td>
<td>7.196</td>
<td>77.2</td>
<td>41.7</td>
<td>-0.8</td>
<td>1.1</td>
<td></td>
<td></td>
<td></td>
<td>2.31</td>
<td>4.69</td>
<td>13.7</td>
<td>40.6</td>
<td>39</td>
</tr>
</tbody>
</table>

CPR (still performed by LDB device) was continued till 1:00 p.m., when ROSC occurred. At that time, central body temperature increased to 22.6°C. Resuscitation lasted for 142 minutes. Results of blood tests pointed to the presence of acidosis with pH of 7.247. Hemoglobin levels maintaining 10.6 g/dL at 10:40 a.m., decreased to 3.2 g/dL in less than 4 hours. Coagulation parameters were also highly elevated. All these disorders combined with history of coffee-ground vomiting, pointed to the features of active upper gastrointestinal bleeding (Table 2).

Gastroenterologist performed emergency gastroscopy, which revealed duodenal ulcer with a diameter of approximately 30 mm (Forrest IIb). Due to the increasing anemia, blood transfusions were initiated (3 units of fresh frozen plasma, 4 units of RBC concentrate, 2 units of PLT concentrate) while resuscitation was still ongoing. We have also administered proton pump inhibitors (esomeprazole a total of 7 ampoules, 40 mg each in continuous infusion), antibiotics (metronidazole 500 mg, ceftriaxone 1.0 g) and fentanyl 100 mcg.

At 7:15 p.m. patient has been transferred to intensive care unit of this same hospital with the BP of 130/74, heart rate of 93 bpm and core body temperature of 29.5°C. Despite of the further treatment conducted in intensive care unit (pharmacotherapy, transfusions of 12 units of PLT concentrate and 4 units of RBC concentrate) multiple organ failure was progressing and at the time of re-arrest, CPR has not been undertaken. Patient was declared dead at 9:45 am of the next day.

4. RESULTS AND DISCUSSION

Accidental hypothermia is an unintentional decrease of core temperature to 35°C or below which is most of the time caused by environmental exposure.3 There are some conditions and diseases predisposing to excessive drop in body temperature, such as injury, alcohol or drug abuse, elderly or very young age and lowered mental status.1 We presume that in this case gastrointestinal bleeding led to patient’s collapse in garden-plot while the outside temperature was low. Influence of both ambient temperature and unconsciousness resulted in severe hypothermia.

According to the Swiss system,1 hypothermia is classified in five stages:
(1) mild hypothermia (patient is conscious, his body is shivering: 35°C–32°C)
(2) moderate hypothermia (patients’ consciousness is impaired, there is no shivering: 32°C–28°C)
(3) severe hypothermia (patient is unconscious: 28°C–24°C)
(4) profound hypothermia (apparent death: 24°C–13.7°C)
(5) death due to hypothermia (less than 13.7°C).

Temperature of 13.7°C is the lowest recorded temperature in hypothermic patient who was successfully resuscitated.4

Low core temperature causes decrease in basic metabolism and neurologic function,5 which in terms of cardiac arrest can have neuroprotective effect. Although SCA in case of severe hypothermia can lead to survival with good neurologic outcome,6,7 prolonged cardiac arrest results in hypoxic brain injury and severe neurological dysfunction.10 It is crucial to initiate effective chest compressions to maintain minimal cerebral blood flow. One way of potentially improving the quality of chest compression is with automatic mechanical devices, which can apply compression more consistently than manual massage. Also, the engineering of such devices may target additional physiological mechanisms to improve circulatory output. Nowadays there are two types of mechanical devices used worldwide: LDB (AutoPulse) and active chest decompression piston device (LU-CAS). So far there are no clear evidences on the advantage from the routine use of these mechanical devices.1 However, ERC guidelines 2015 emphasize the key role of mechanical chest compression in selected patients (e.g. when manual chest compressions are impractical or compromise provider safety). Randomized controlled trials in the field of emergency medicine, especially in the subject of resuscitation, are controversial and difficult to conduct,11 which makes research on mechanical chest compressions challenging.

The implementation of mechanical chest compression device in case of this patient, has provided good quality chest compressions which was of a great importance considering longstanding CPR. Furthermore, since we have used mechanical ventilation, mechanical chest compressions device and monitored patient’s heart rhythm with the use of self-adhesive pads instead of paddles, we’ve saved a lot of human resources. Two physicians and two paramedics were present on patient’s bedside on admission and at the beginning of treat-
ment. After setting everything up only one physician and one paramedic were taking care of the patient. This model of SCA management is, in our opinion, the most effective, especially in circumstances of prolonged resuscitation.

According to the latest guidelines, extracorporeal life support may be helpful in some hypothermic cardiac arrest. At the time of patient’s admission to our department we immediate access to that device. However, literature and presented case study, show that it is possible to warm up hypothermic cardiac arrest patients successfully through noninvasive methods, which are both easy to apply and feasible in any hospital.

There are some studies on prognostic factors likely to identify patients in hypothermic cardiac arrest who would probably survive. According to Mair et al. on plasma potassium levels (serum potassium more than 9 mmol/L), central venous pH (pH less than 6.50) and ACT (activated clotting time more than 400 s) on admission can be used to identify hypothermic arrest victims with predicted poor outcome. Mair’s paper was a small retrospective study (analysis of 22 patients), however more investigators report connection between high potassium level and death. Our patient presented hypokalemia and acidosis, however neither potassium level nor pH were extremely low. Regardless these, seemingly, favorable values, patient’s poor general condition, co-morbidities, gastrointestinal bleeding and long-lasting hypothermia prior to arrival of EMT have put him at risk of multi-organ failure which resulted in his death the next day.

5. CONCLUSIONS

Cardiac arrest due to hypothermia can lead to extension of resuscitation. To improve survival of patients in situations requiring prolonged resuscitation, mechanical devices performing chest compressions should be implemented. It is possible to successfully warm up hypothermic cardiac arrest patients through noninvasive methods.

Conflict of interest

None declared.

References