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REWARMING FROM SEVERE ACCIDENTAL HYPOTHERMIA WITH CIRCULATORY ARREST

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This case report demonstrates successful cardiopulmonary and cerebral resuscitation (CPCR) of a young male explored 15 hours following a suicide attempt (carbamazepine intoxication) in deep hypothermia (19°C) with circulatory arrest. An extracorporeal circuit was used to rewarm the patient's blood. Weaning from extracorporeal circulation (ECC) was successful and without complications as was recovery from multiorgan dysfunction, severe rhabdomyolysis and carbamazepine intoxication. An excellent outcome was achieved without any neurological deficit at the time of discharge from the hospital.

INTRODUCTION

Severe accidental hypothermia remains a challenging clinical problem mainly affecting victims of outdoor accidents who suffer from prolonged exposure to a cold environment or immersion/submersion in cold water[1]. Most reports from urban environments are associated with drug or alcohol abuse, serious illness in the elderly, suicide or accidents related to sports activity[2]. Only a few case reports present a larger patient population of severe accidental hypothermia[3–8]. Single or few cases are routinely analyzed. Deep accidental hypothermia (core temperature below 28 °C) causes circulatory and neurological disturbances. Despite immediate treatment, improved prehospital survival, rewarming techniques in hospital and new types of heart and lung assistance, it is still associated with high mortality ranging from 30–80 %. The wide variability of survival reported in the literature is due to such factors as differences in accident mechanism, patient populations, core temperature as well as pre-hospital handling and rewarming indications. As far as we know life function stabilization leading to good physical and mental recovery after 4 hours of resuscitation in a victim of severe deep hypothermia with a core temperature below 20 °C is rare.

It has been shown that within the range of 0 °C to 42 °C, O_2 consumption will fall 50% for each 10 °C decrease in temperature. As the rate of temperature falls, the rate of O_2 consumption in general decreases more rapidly than supply, and local stores are consumed more slowly. Hypothermia thereby exerts a protective effect on the cells from anoxia. In the patients suffering from severe hypothermia the most important point influencing the outcome is the level of hypothermia in the moment when the ventricular fibrillation occurs.

The prognosis for patients suffering from severe hypothermia could be influenced also by the intoxication. This case report demonstrates the combination of hypothermia and lethal dose of carbamazepine intoxication. This combination seems to be unique in literature reviews.

CASE REPORT

This case report demonstrates successful resuscitation in a young man aged 26 found 15 hours after a suicide attempt (carbamazepine intoxication) in a deep hypothermia of 19 °C with circulatory arrest.

Extracorporeal circulation (ECC) was used to rewarm the patient. Weaning from ECC was successful as was recovery from extensive metabolic disturbance due to severe rhabdomyolysis and carbamazepine intoxication, the patient showed no neurological deficit at time of discharge from the hospital.

A 26-year-old man with a 19 year history of secondary epilepsy and benign tumorous expansion localized on the left parietooccipital region with no other somatic comorbidity. The convulsion frequency was approximately 1–2 / year. Chronic medication consisted of a combination of valproate (Depakin Chrono 300 mg) and carbamazepine (Timonil 200 mg). There was also a 2–3 years history of psychostimulating drug abuse.

As a victim of suicide attempt he consumed a total dose of 15 grams of valproate (Depakin Chrono) combined with 15 grams of carbamazepine (Timonil) on January 6th as a consequence of an argument with his parents. The body was discovered 15 hours later in a forest. The patient's condition was: unconscious, deep hypothermia with circulatory arrest due to ventricular fibrillation. A snow base presented an outdoor temperature oscillating around 0 °C. Oral endotracheal intubation and 100 % oxygen ventilation started cardiopulmonary and cerebral resuscitation (CPCR). Repeated defibrillation failed due to profound hypothermia. Following this procedure by the

Table 1. Admission data

age	26
sex	Male
core temperature	19 °C
cause of hypothermia	exposure
cardiac rhythm	ventricular fibrillation

Table 2. Acid base parameters and s-potassium values after initialization of ECC

pH	7.09
PaO_2 (kPa)	46.2
$PaCO_2$ (kPa)	6.7
BE (mmol/l)	-12.8
potassium (mmol/l)	4.7

Table 3. Biochemical parameters in the early postoperative period

	1. day	2. day	3. day	4. day	5. day	8. day	Normal range
PH	7.19	7.43	7.44	7.37	7.46	7.38	**7.35–7.45**
PaO2 (kPa)	19.3	20.3	16.5	17.8	22.8	6.7	**9.99–13.33**
PaCO2 (kPa)	6.6	7.2	5.8	6.0	5.5	5.5	**4.66–5.98**
BE (mmol/l)	–9.5	–9.8	–4.3	–8.1	5.3	–0.6	**–2.5–2.5**
Potassium (mmol/l)	4.0	3.9	3.7	3.8	3.6	3.9	**3.5–5.3**
Lactate (mmol/l)	12.0	1.39	2.62	0.97	1.1	---	**0.63–2.44**
Myoglobin (ng/ml)	7371	8606	2760	546	253	---	**0–70**
Carbamazepine (µg/ml)	65.0	---	16.3	10.1	---	---	**4–12**
Alanine aminotrans-ferase (µkat/l)	1.1	1.89	2.4	2.41	---	---	**0.15–0.9**
Aspartate amino-transferase (µkat/l)	1.98	6.44	9.22	7.15	5.25	---	**0.15–0.57**

professional rescue team, the patient was transported to Ostrava University Hospital and taken immediately to the operating theatre of the Department of Cardiac Surgery. CPCR was ongoing. The esophagal temperature was 19 °C. Ventricular fibrillation appeared on the screen. Admission data are summarized in Table 1.

Rewarming using ECC was initiated immediately following insertion of cannulas using standard equipment for open-heart surgery (heparin-uncoated oxygenator and tubing set). Arterial and venous access for ECC was performed via femoral artery and vein. Acid base parameters and s-potassium values after initialization of ECC are presented in Table 2. Extracorporeal rewarming (gradient 5°C, heat exchanger vs. actual core temperature) was completed after 158 min. The weaning from ECC was performed without the need for inotropic support.

Antioedematous therapy continued after surgery with furosemide and manitol. Carbamazepine was eliminated using active charcoal inserted via nasogastric tube and forced urine output performed. Total intravenous nutrition, antibiotics, nootropics, antioxidants as well as volume expanding solutions were applied. Surgical care was taken of II. – III. grade chilblains localized on the back of the patient's body in approximate 3 % area of body surface at the same time.

Trends of blood samples in the early postoperative period are summarized in Table 3. Thus shows the shock status with elevation of specific cardiac marker levels as they responded to prolongated CPCR. Results of toxicological samples and biochemical urine samples also showed a lethal dose of carbamazepine as serum levels.

The regression of neurological symptoms continued subsequently as mydriasis converted to miosis 12 hours after beginning CPCR and papillary response became symmetrical. The serum level of myoglobin reached 8606 ng/ml on 2nd day after surgery due to extensive rhabdomyolysis. Forced urine output and urine alkalinization continued. Serum levels of carbamazepine declined gradually reaching the maximum therapeutic level of 16 µg/ml. The serum level of myoglobin also dropped. Weaning of artificial pulmonary ventilation terminated with extubation in full consciousness without neurological deficit the 5th day after surgery.

The patient was transferred on the 8th day after surgery to the ICU of the Department of Plastic Surgery of Ostrava University Hospital to continue in chilblains treatment. The patient was discharged from hospital in a stable condition, without neurological deficit 3 weeks later. Psychiatric examination established suicidal attempt as an action performed in a depressed state. The risk of recurrence of suicidal attempt was declared marginal.

DISCUSSION

Victims in deep hypothermia with circulatory arrest are generally in a critical condition. The most effective treatment technique apart from CPCR is to connect the victim to an extracorporeal circuit and rewarming. A literature review demonstrates only a few reports presenting a larger patient-population of severe accidental hypothermia. Walpoth[7] presented the results of 11 hypothermic patients in 1990. Mortality was 35 %. Mortality in Kornberg's[5] group of 24 patients was 87 %. Hauty[4] reported a similar mortality of 82 % whereas no patient with hypothermia below 20 °C survived. Farstad[1] described the results of group of 26 patients with 79 % mortality. Single or few cases are routinely analyzed.

The largest patient population found in literature reviews was reported by Vretenar[9]. This was a large retrospective analysis of all studies and case reports of hypothermic patients resuscitated using an extracorporeal circuit. This group consisted of 68 patients. Sixty-one had circulatory arrest. The overall mortality in this group was 40 %. 80 % of those who survive were without neurological deficit. Mortality was dependent on initial central temperature. The mortality of a group of patients with an initial temperature below 20 °C was 59 % (10 out of 17). Mortality of 100 % occurred in a group whose initial temperature was below 15 °C while the mortality in patients in a group with an initial temperature over 20 °C was 33 % (17 out of 51).

The results varied according to differences in composition of patient grouping. Patients with asphyxia, children (most often drowned) and severe hypothermia are poor prognosticators. Patients with hypothermia up to 25 °C have better prognosis while survival of patients with a central temperature below 20 °C is rare. Gilbert case[10] demonstrating successful resuscitation of a patient with a central temperature of 13.7 °C is unique.

Prognosis and the neurological sequellae in patients suffering from deep accidental hypothermia is uncertain as well as quality of their lives subsequently. Potassium serum levels over 10 mmol/l, pH under 6.5, low PaO_2 (arterial partial oxygen pressure) are poor prognosis predictors[1]. Prognosis worsens too for patients with asphyxia.

A combination of deep hypothermia and intoxication makes impact on prognosis more under the influence of the hypothermia itself. The case report described here was induced by a combination of hypothermia and lethal dose of carbamazepine. This combination has not been described before. Carbamazepine lowers cell membrane sensitivity. It's effect seems to be central nervous system protecting by blocking reuptake at the level of neurones. As explanation of survival of demonstrated patient without neurological disturbances, we presume ventricular fibrillation occurred with the onset of severe hypothermia in addition to the antiarythmic effects of carbamazepine.

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