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Out-of-hospital asystole caused by hanging treated with endovascular mild therapeutic hypothermia: a case report

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Hanging may result in death either from direct, immediate damage to vital organs (spinal cord injury, airway disruption, carotid artery laceration), or from indirect effects on the cardiovascular system (autonomic reflex through the carotid sinus body compression, airway occlusion), with cardiac arrest representing the worst clinical complication [1]. Here we report a case of out-of-hospital cardiac arrest caused by hanging, which has been treated with mild therapeutic hypothermia by an endovascular technique, with complete neurological recovery.

A 53-year-old man attempted suicide by hanging. The Mobile Pre-hospital Emergency Team was alerted by his wife, and arrived on the site 10 min after the event. At the scene, the medical team found the patient positioned on the floor in cardio-respiratory arrest (first cardiac rhythm detected was asystole). A cervical collar and an intravenous line were immediately put in place, and the Advanced Life Support protocol was started by the paramedics and the emergency physician on the scene. The patient was immediately intubated, artificially ventilated, and received epinephrine (2 mg) and atropine (1 mg). After 5 min of resuscitation, during which time the patient remained in asystole, the patient recovered a spontaneous cardiac sinus rhythm (heart rate 90 beats/min), with an arterial blood pressure of 120/70 mmHg. The Glasgow Coma Scale (GCS) was 4, and pupil assessment was normal. The patient was transferred to the

Trauma Center by Emergency Helicopter after medication with midazolam, morphine, atracurium.

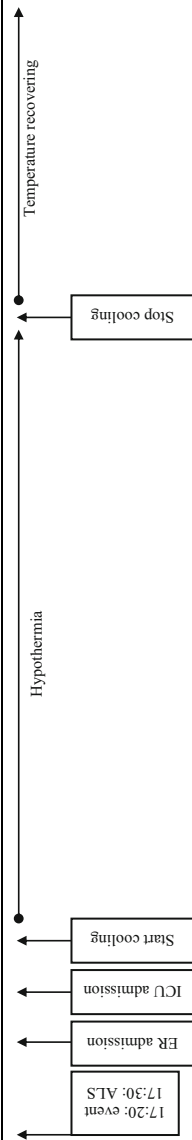
The patient was admitted to the ED 1 h after the event. An anesthesiologist from the Intensive Care Unit (ICU) was present at admission, and followed the patient during ED assessment, as provided in the internal protocol of the Trauma Center. A central venous oximetry catheter (Pre-Sep, Edwards Lifesciences LLC, Irvine, CA, USA) was inserted in the right internal jugular vein, under the guide of a bedside ultrasonography, and a catheter for invasive arterial pressure monitoring (Leadcath, Vygon, Ecouen, France) was placed in the left femoral artery. A mean arterial pressure (MAP) above 65 mmHg, and a central venous oxygen saturation (CvO₂ sat) above 70% was achieved with fluid [saline and 6% hydroxyethylstarch (HES 130/0.4)] infusions. After stabilization, the patient underwent a computerized tomography (CT)-scan of the head–neck–spinal vertebrae region, which demonstrated no encephalic alteration or traumatic dislocation of the cervical vertebrae, and was transferred to the ICU.

On admission to the ICU, the patient had a GCS of 7. He presented myoclonus limited to the upper limbs. Empiric therapy for post-anoxic seizures was started with valproic acid which was maintained until discharge. After sedative (propofol and fentanyl) wash-out, we performed an electroencephalographic (EEG) examination that showed signs of post-anoxic cerebral distress, but no seizure-like electrical activity. Somatosensory evoked potentials (SEPs) did not show cortical–subcortical signal alterations. A mild therapeutic endovascular hypothermia was started almost 5 h after the event and maintained for 24 h (tympanic temperature at admission: 35.9–C). The CoolGard Icy Catheter (Alsuis corp., Irvine, California, USA; 8,5 fr) was positioned in the right femoral vein. The patient's core temperature was maintained between 32 and 34°C. An

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Table 1 Time-course and parameters before ICU admission, during cooling and after temperature recovering

CVP (cm/H ₂ O)	63	80	85	7.23	7.38	7.37	7.42	7.42	7.50	7.38	7.35	7.40	7.36	7.43	7.37	7.35	7.43	7.40	7.33	7.43	7.37	7.45	7.46	7.37	
MAP (mmHg)	63	80	85	77	77	77	83	83	77	77	75	68	74	68	70	75	72	68	70	75	85	88	83	85	92
HR (beat/min)	90	85	88	72	65	60	65	65	60	50	54	55	62	46	50	47	44	46	50	44	47	55	60	62	95
pH	7.23	7.38	7.37	7.42	7.42	7.50	7.42	7.42	7.50	7.38	7.35	7.40	7.36	7.43	7.40	7.37	7.35	7.43	7.40	7.33	7.43	7.37	7.45	7.46	7.37
PaCO ₂ (mmHg)	61.9	36.9	38.5	30.2	31	21	34.2	40.2	31.8	34.2	40.2	31.8	31.1	30.6	40.5	34.5	39	30.6	42.3	42.3	33.6	33.5	32.3	31.7	41
pO ₂ /FiO ₂	392	238	454	412	400	488	460	434	454	460	434	454	460	483	471	517	426	483	471	449	463	457	434	426	205
Art O ₂ sat (%)	99.6	98.9	99.9	100	100	100	100	99.6	99.7	99.5	99.5	99.7	99.5	100	100	99.3	100	100	100	99.5	99.9	100	99.4	100	99.3
CvO ₂ sat (%)	78.2	82.6	87.7	91.1	92.3	92.0	91.8	93.6	90.5	92.0	91.8	93.6	90.5	90.7	91.1	89.7	91.0	90.7	91.1	85.6	84.0	81.1	78.4	77.6	76.4
Hb (g/dl)	15.6	13.9	14.5	15.8	15.5	15.8	14.8	15.3	16.4	14.8	15.3	16.4	14.2	16.5	16.8	15.8	16.8	16.5	16.8	16.9	16.2	16.4	16.4	16	15
K ⁺ (meq/l)	4.6	3.9	3.7	4.1	4	3.5	4	3.9	3.1	2.6	4	3.9	3.1	4.8	4.7	4.3	4.6	4.8	4.6	4.6	4.1	4	3.8	3.5	3.8
Na ⁺ (meq/l)	141	138	136	132	134	133	137	137	137	137	137	137	139	135	136	136	136	135	136	136	133	136	134	133	134
Ca ²⁺ (meq/l)	1.10	1.12	1.04	0.99	1	0.99	0.99	1.12	1.11	0.99	1.12	1.11	0.94	1.15	1.12	1.11	1.11	1.15	1.12	1.1	1.12	1.05	1.08	0.99	1.11
HCO ₃ ⁻ (meq/l)	21.9	21.9	22.5	21.9	22.2	22.2	21.3	21.4	20.8	21.3	21.4	20.8	19.1	22.4	22	21.6	22.4	22.4	22	21.6	23.6	24.1	24.4	24.8	23.3
Glucose (mg/l)	138	98	102	122	125	110	116	89	118	116	89	118	91	104	120	115	113	104	120	115	125	121	146	118	104
Lactate (mg/l)	3.5	1	1	0.9	1	1.7	1.8	3.3	3.9	3.1	3.3	3.9	3.1	3	3.4	3.2	3.4	3.4	3.5	3.5	2.4	2.1	1.8	1.7	1.1
T (°C)	36.1	35.9	35.4	33.6	33.2	33.1	33	33.1	32.8	33.1	33.1	32.8	33.2	33.8	34.1	33.4	33.6	33.8	34.1	34.5	35.1	35.4	35.9	36.2	36.3
Time	18:30	21:00	22:00	00:00	02:00	04:00	06:00	08:00	10:00	12:00	14:00	16:00	18:00	20:00	22:00	00:00	02:00	04:00	06:00	08:00	10:00	12:00	14:00	16:00	18:00



CVP Central venous pressure, MAP mean arterial pressure, HR heart rate, PaCO₂ partial arterial CO₂ pressure, PaO₂ partial arterial O₂ pressure, FiO₂ oxygen inspiratory fraction, Art O₂ arterial O₂ saturation, CvO₂ central venous O₂ saturation, Hb hemoglobin, T temperature

accurate glucose control was performed according to post-resuscitation support protocol. Clinical parameters were monitored continuously, and an arterial blood gas analysis was performed every 2 h (Table 1). At the end of the cooling period, the patient recovered normothermia passively (Table 1). Shivering was avoided with muscular paralysis (atracurium). Venous districts were monitored frequently by ultrasound examination for detection of deep venous thrombosis. No complications occurred during or after the cooling procedure.

At day 7, the patient was conscious and breathing spontaneously. The EEG showed a clear improvement with normal SEPs. At 26 days from the attempted suicide, the patient was transferred to a Physical Rehabilitation Center to continue physiotherapy and psychiatric assistance.

Mild therapeutic hypothermia (MTH) has been proposed to avoid cerebral damage after out-of-hospital cardiac arrest [2]. Previous ILCOR guidelines recommend MTH only if the initial rhythm was ventricular fibrillation. Indeed, evidence supporting the use of MTH for post-cardiac arrest syndrome, even due to out-of-hospital asystole, have been recently reconsidered, and several data indicate a possible beneficial role of MTH in numerous pathological conditions in which neurons need to be protected by hypoxic injury [3]. In the present report of a suicidal hanging, the patient did not have a fall from a high height greater than his body length (it was less than 2 m). In cases like this, cerebral damage is caused by carotid artery and jugular vein occlusion [1], thus, the use of MTH in these situations appears to be particularly appropriate in regard to obtain cerebral protection. In a similar case report, a combination of ice packs and cold air convection were applied, and the authors reported an excellent neurological recovery; however, no information on the type of rhythm present at the time of cardiac arrest was available [4]. In our report, parameters at hospital admission were not dramatic. Nevertheless, the presence of myoclonus, considered an early manifestation of brain damage, suggests that the use of MTH could have had a role in neurological improvement.

The goal of treatment should be the initiation of cooling starting in the field or immediately on ED admission. However, and despite ILCOR guidelines [2], it has been reported that MTH is still underemployed in many German hospitals because of supposed difficulties of the technique in the intensivists' opinion [5]. In alternative intravascular cooling, the external cooling methods are more diffuse, even if the endovascular cooling system results in the most reliable method to obtain and maintain a correct and steady temperature [6], with rapid achievement of MTH.

In conclusion, we report the use of MTH in helping to prevent cerebral hypoxic damage in a patient who sustained a cardiac arrest in asystole caused by hanging.

Conflict of interest statement The authors declare that they have no conflict of interest related to the publication of this manuscript.

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