DETERMINISM, COINCIDENTAL OR MULTIPLE CONCURRENT ISCHEMIC EVENTS IN SEVERE HYPOTHERMIA – CASE REPORT

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ABSTRACT

Introduction
Accidental hypothermia produces a multitude of complex issues in the evaluation plan, investigative strategy, tactics and choice of therapeutic resources and the development and prognosis of the patient, especially in a plurilessional context.

Case study
We examined the case of a 79 years old patient, assisted at ED Craiova in November 2012 with severe hypothermia, found down the street. On admission to the ED, he appeared confused, disoriented in space and time, GCS = 10, no focal signs with generalized rigidity, tremor. Killip III cardiogenic shock, peripheral pulses hardly perceptible. Cold extremities, mottled.

The patient with severe hypothermia (central T - 29.1 ° C) had multiple confounding aspects of cerebral ischemia, peripheral of the limb and myocardial, possible attributable to both hypothermia, cardiogenic shock (acute coronary syndrome in the comment), an evolving ischemic stroke, an acute peripheral left limb ischemia with severe rhabdomyolysis, acute bilateral pneumonia, or association in different degrees and their sequences, with the prognosis of extreme gravity.

The determinism, primary or secondary of the cardiac rhythm disorders in relation to hypothermia, give also rise to discussions and the possibility of little peripheral embolism can be neglected.

Conclusions
• Cardiogenic shock was the turning point in determining the evolution of this case and its double determinism conditioned most likely the poor prognosis of the patient.
• Pneumonia was the second severe impairment that influenced survival, limiting refilling and affecting oxygenation optimization
• The question of etiology of heart disrhythmia, its seniority and role in the induction of cerebral and peripheral ischemia
• The myocardial vulnerability maintained both during hypothermia and after reheating
• A number of clinical syndromes may have particular aggravating evolution in the context of hypothermia
• Therapeutic response was affected by hypothermia
• A series of subclinical suffering became manifests in these circumstances, such as renal suffering and peripheral arterial disease
• It is always difficult to distinguish the primary event in a clinical context complicated by the presence of hypothermia.

Keywords: Accidental hypothermia, stroke, acute coronary syndrome, acute peripheral ischemia.

Introduction

Hypothermia, in the emergency services activity, is not a exclusive hibernate phenomenon, so it must be suspected, certified, treated, or prevented if appropriate, in many other circumstances of age, pathologic or iatrogenic, which may expose some patients to altering the ratio between the generation and loss of heat. The elderly, children, traumatized, immobilized patients, patients under the influence of drugs or alcohol, submersion victims, patients for whom it was proceeded to cooling the burn injuries or induction of general anesthesia, hipovolemics actively infused in the extrahospital environment are at risk of hypothermia (9).

A number of symptoms of hypothermia are of great clinical interest because they interfere with the other clinical pictures that mask or mimic, diverting the attention of the medical team from the severity elements and altering the clinical examination findings. A number of primary lesions may be seen as secondary and thus amendable with reheating, thus delaying the appropriate treatment setting and some even require alternative means of therapy because hypothermia may increase the risk of adverse effects.

A series of laboratory investigations may require temperature correction for the results to be accurate and truly useful.

Finally, the gap between the rapid need for vigorous active reheating, the poverty of the reheating means at their disposal outside the hospital, require the rapid transfer of the critical patient to the hospital, even before satisfying all desiderata stabilization of the vital functions and risking enormous heart vulnerabilities.

Case study

The patient CC - male, 79 years old, from rural areas (93 229 ER FO) B2 brought by ambulance to the Emergency Department of the Clinical Emergency Hospital Craiova on 22.11. 2012, at 2:39, found in the street.
On admission at ED, this patient was confused, disoriented in space and time, Glasgow Coma Score = 10 without focal signs, with generalized rigidity, tremor. Central temperature 29.1 °C. BP 95/60 mm Hg. HR - 56/min. Disrhythmia. Peripheral pulses hardly perceptible. Cold extremities, mottled. Decreased breath sounds on the thoracic right side, disseminated bronchial rales, crepitation rales at both lung bases. No visible injury marks. Ingual right hernia, voluminous, reducible. No details about the event onset or the initial duration of exposure to cold were available. No medical history, current medication or allergies were available.

The stage diagnosis of the emergency team was: Severe hypothermia; Killip III degree cardiogenic shock; Atrial fibrillation with slow ventricular response; Confusional syndrome; Suspected stroke in evolution; Acute pneumonia.

In addition to the initiation of monitoring the vital signs and supportive management and reheating, the paraclinical investigation was launched, focused on the following objectives:

- Identification and prioritization of life threatening injuries
- Identifying associated pathological elements
- Establishing a temporal sequence of pathological events
- Shaping metabolic panel results in combination with other elements of pathological hypothermia
- Follow evolutionary dynamics

Initial ECG - Atrial fibrillation with slow ventricular rate, Osborne wave which persists until cT exceeding 33 ° C. Also, AF is maintained after returning CT above 36 ° C

Relevant elements of the laboratory results were:

- hypoglycemia (glycemic level 38 mg% increases to 228 mg% in 6 hours – during this time he was given 500 ml glucose of 10% and 500 ml glucose of 5%), Na 166.3 mmol / l, K 4.1 mmol / l, pH 7, 17, BE - 9.8, Urea - 78 mg% creatinine - 1.36 mg%, PO2 - 83.8 mmHg, PaCO2 - 50.5 mmHg
- Hb - 12.1 g / l, hematocrit - 38%, it decreases to 4 hours at 33.8%, no. WBC - 6800/mm3.
• Ck - Mb - 352microg / ml, troponin T – negative, dynamic at 4:00 hours
Troponine T - 0.021 microg / ml, and at 8:00hours Troponin T - 0.112 microg / ml,
myoglobin - 1000 microg / ml
• Presepsin 682 pg / ml.
• Tox - quality of saliva - negative
• CXR at bedside - increased the interstitial perihilar right lung and bilateral basal
with reticulomicronodular aspect suggestive of interstitial pneumonia. Cord with
aortic configuration.
• FAST - no free fluid in the peritoneal cavity or pericardium.
• Cardiac ultrasound has not noted areas of akinesia or dyskinesia.
• Ct native skull recent did not visualize focal lesions (only the bleedings were
excluded at that moment). Cortical atrophy.

Internal active heating was initiated- multiple vascular access (one internal jugular
vein and 2 peripheral veins) heated, humidify oxigen, gastric and bladder lavage and
active external heating (hot air) with reheat average rate of about 2 ° / 3hours (time 5.30 to
31.2 ° C, at 8.10 to 33 ° C, at 11.05 to 35.8 ° C).

The patient was intubated endotracheal and SIMV volume-cycled assisted, deep
sedation (diazepam and etomidate), broad-spectrum antibiotics.

During 8 hours of therapy with the administration of saline 1500 ml, 1000 ml of
Ringer's solution, 500 ml of 10% glucose and 500 ml of 5% glucose, the positive inotropic
support (dobutamine 7 micrograms / kg body weight / minute, increased to 10 mcg / per kg
body weight / minute, associated with dopamine 7.5 micrograms / kg body weight / 
minute), total urine output 600 ml, systolic BP showed decreasing trend to 65 mmHg (at
7hours, when core temperature was 35 ° C), and rales crepitation persistent tended to
expand to apical.

After 4 hours of tracking, central temperature of 33 ° C, at the level of the left
lower limb it was delimited an acute ischemia area with a level in third lower thigh, absent
distal pulse and no detectable blood flow (Doppler signal absent), while the pulse
controlateral was maintained distal palpable at all levels. After another 3 hours at this level
cyanosis improved, the skin heated slowly but persisted the color and temperature
differences compared to the contralateral limb for another 6 hours, during which – it was
kept the pulping muscle mass and the absence of distal arterial pulse.

The patient was admitted to the medical intensive care unit in a induced coma,
normotermic with SBP 70-80 mmHg maintained with inotropic and vasoactive support
with average AF- monitored. 72 hours later, he dies from multiple organ failure (renal,
respiratory) and progressive shock.

Anatomopathological examination revealed right ventricular myocardial infarction,
pneumonia in the right medium lobe, recent stroke in the tent of the cerebellum of 13 mm.
Stasis and pulmonary edema

Discussions

The specific features of the case which deserves special discussion were:
**Myocardial depression, cardiogenic shock.** The enzyme picture of myocardial and muscle breakdown - already important at admission in UPU and evolving - induced the supposition of cardiac etiology of degradation of the patient's condition - evolving acute coronary syndrome (event that led to the degradation of consciousness and to the onset of hypothermia) but not ruled out the contribution of severe hypothermia on depression ejection fraction (6) during and after reheating. Myocarditis was equally diagnostic suspicion, justified by the existence of pneumonia, hypothermia context, the evolution of enzymes, but once they grew, they advocated more for coronary etiology. However, it is not precluded the development of myocarditis during the 72 hours of evolution, that might have further complicated the ischemic and metabolic suffering of the heart. In this context it is to be discussed on the one hand the extent to which hypothermia may have protective effect by lowering basal metabolism and myocardial oxygen consumption (7) and secondly how it influences therapeutic decision of quick refill (10), reperfusion, including the choice of means (11). Not lastly, contractile cardiac dysfunction caused by reheating is another severe problem, whose contribution to overall mortality of accidental hypothermia is significant (6).

**Cardiac disrhythmia** - atrial fibrillation - can be both an effect of hypothermia, but proved to be resistant to heat, outstanding throughout the whole future development, which made us interpret it as a chronic disease, compounded hypothermia and hardly quantifiable risk of embolism in the context of disorder of the increase in blood viscosity. Hypothermia ruled out from the emergency phase the temptation for digitization as management attempt of AF due to low frequency, but especially for fear of the risk of inducing ventricular rhythms favored by myocardial hypersensitivity to endogenous and exogenous catecholamines.

**Acute peripheral ischemia.** Decompensation during reheating of peripheral circulatory insufficiency in the left limb may have several causes: the collapse of regional flow within the splanchnic and renal under maldistribution, coagulation disorders (13,14,15), hyperviscosity (12), but there is the possibility that the acute ischemic episode may have been the result of an arterial embolism caused by atrial fibrillation (arteriography was considered risky to impaired renal function). Rhabdomyolysis caused by this added extra gravity general metabolic affection and therapeutic controversy concerning the opportunity, the moment and the administration of the anticoagulation management protocol (8).

**Pulmonary injury** was suspected to have pre-existed, to be the cause of the infection, but may have been caused by hypothermia and complicated by acute heart damage. In addition, poor tolerance to the attempt of loading pulmonary fluid (7), generated by the deterioration of lung compliance and by the force of contraction of the heart werea limiting therapeutic options.

**Altered mental status** - as other clinical factors are claimed to be interpreted as an effect of severe hypothermia or secondary brain suffering from shock and hypothermia (3) or a stroke especially in a patient with atrial fibrillation and unknown treatment history. Also toxic etiology had to be excluded. From another perspective, the management of ischemic stroke may assume (by analogy with the treatment of brain trauma or cardiac
resuscitation) inducing hypothermia (4) but it is controlled hypothermia, moderate, limited to the cephalic extremity (1.16) for to benefit of the brain suffering.

Due to the circumstances of uncertain etiology (patient found down the street) posttraumatic symptoms had to be excluded - abdominal, brain, spine (2, 15) – and up to the exclusion, the treatment had to be adapted, which added a number of practical difficulties in achieving endotracheal intubation and central venous access.

**Kidney suffering** possible already preexisting to the ED arrival, possible background or foreground of cardiogenic shock, possibly induced and maintained also by hypothermia was certainly a key factor for worsening the prognosis in the context of severe rhabdomyolysis.

Severe hypothermia, causing extreme disruption of the defense mechanisms and repair tissue, favored bacterial invasion, created conditions for rapid and unfavorable of the pneumonic process, with the onset and progression of sepsis in the context of myocardial suffering and poor peripheral circulation.

Also disturbing the coagulation system - fibrinolysis (initial hypercoagulable - increased viscosity (11) / decrease in platelet activation, prolongation of PTT, complement activation, and priming intravascular disseminated coagulation - release massive thromboplastin followed by massive defibrinisation (12, 14) in 24-36 h from reheating resulted in worsening the ischemic suffering, initially subclinical, cerebral, myocardial and at the level of the lower limb over adding to the vascular component, rheological component. Nevertheless, the presence of atrial fibrillation of unspecified length brought into discussion the possibility of multiple emboli (lower limb and brain) that were the basis of ischemic events in these territories and obviously were aggravated by bleeding disorders and vasoconstriction. Nevertheless, it is possible that hypothermia to be a valuable cause of inducing rhythm disorder and suffering due to acute myocardial, which clearly influenced the ejection fraction of the heart.

Disruption of protein synthesis, including the synthesis of antibodies, extreme vasoconstriction, decreased cardiac ejection fraction (decrease in both force of contraction and heart rate, precipitation of arrhythmias) led to alterations in vital organs or regional flows involved in priming subsequent multiple organ failure as a territory within the splanchnic circulation already damaged as a result of activation of netting cardiogenic shock. It also can lead to a combination between secondary brain suffering to the primary one (the acute cerebral ischemic event was one of causes that can be criminalized in patient immobilization in external environment)

In addition to these initial carbohydrate metabolic imbalance (maximum utilization of glucose leads to muscular and hepatic glycogenolysis, and gluconeogenesis from amino acids, resulting in metabolic acidosis) which emphasizes the secondary brain suffering.

Rhabdomyolysis, initially by intense muscle activity due to the substrate, then secondary to the acidosis, electrolyte imbalances (especially hyper potassium levels), and reabsorption disorders completed a highly unfavorable metabolic context of cardiac, vascular motility and distribution of microcirculation, contributing to the generation cardiogenic shock.

Temperature correction of blood samples was performed for the determinations under 34 degrees. C (cT)
The clinical picture of hypothermia can cause confusion with other clinical syndromes at initial assessment (mild hypothermia generalized tremor which disappears as the temperature decreases, pulse bradycardia, irregular, filiform, low blood pressure, mydriasis, paraesthesia, decreased motility, altered progressive consciousness to coma) and altered mental status may predispose itself to effects of removal of symptoms and clinical signs characteristic of other injury.

It is also known that the low rate of basal metabolism in hypothermia makes that drugs administered accumulate to toxic levels sometimes, especially arrhythmogenic effects being enhanced in terms of acidosis and hyperkalemia, and the heart of the patient was expected to be extremely sensitive including endogenous catecolaminene being ischemic. Therefore, the intervals to re-administration were adjusted according to the level of hypothermia, but it was obvious that the initial inotropic support failed to optimize cardiac index (stasis rales persisted, cardiac tension were kept down). It is equally true that the combination of low pulmonary compliance, pulmonary infection was a negative pulmonary response element to the fast fluid refilling fast, which could not be compensated for vasopressor support, especially on a heart hardened by hypothermia and possible acute coronary syndrome.

If at this stage we add frostbite, tissue damage as metabolic impact (rhabdomyolysis, metabolic acidosis, hyperkalemia) it was particularly important on myocardial function (dysfunction resulting from precipitated arrhythmias and pump) on vasomotricity (generating opening arteriovenous shunts and emphasizing vascular collapse) and not least over the renal function.

Triggering endotoxin shock was a consequence of all these affections. These changes bring significant harm themselves in the development of primary lesions, with few resources compensations even in the presence of strong, early and multidirectional management.

Conclusions

- Cardiogenic shock was the turning point in determining the evolution of this case and its double determinism conditioned most likely the poor prognosis of the patient.
- Pneumonia was the second severe impairment that influenced survival, limiting refilling and affecting oxygenation optimization
- The question of etiology of heart rhythm disorders arised, its seniority and role in the induction of cerebral and peripheral ischemia
- The critical myocardial vulnerability maintained both during hypothermia and after reheating
- A number of clinical syndromes may have particular aggravating evolution in the context of hypothermia.
- Therapeutic response was affected by hypothermia.
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