Case Report

Hypothermia after Cooling Therapy in Exertional Heat Stroke: A Case Study

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Abstract

Exertional heat stroke is an emergency condition which can be found in occupational settings. Pre-hospital and medical management is essential for prevention of morbidity and mortality. A 33-year old male ranger was sent to a district hospital presenting with unconsciousness and generalized tonic-clonic seizure. His had an axillary temperature of 42ºC, blood pressure of 60/40 mmHg and heart rate of 160 beats per minute (bpm), His blood glucose level was 59 mg/dL. Exertional heat stroke was suspected. He was resuscitated, intubated, medicated and referred to a tertiary hospital. During an hour of transportation, his body was submerged in ice and water. At the tertiary hospital, his esophageal temperature was 29ºC with presentation of Osborn wave in electrocardiogram and electrolyte imbalances. After rewarming and correction of electrolytes in the intensive care unit, he gained consciousness the following day. A day after that, hyperthermia developed from Enterococcus faecalis septicemia which responded to medication. On admission day 6, physical examination showed only weakness of his right nasolabial fold. Although this patient had minor complications, lethal or serious outcomes could have occurred in this patient. This report emphasizes the need for early recognition and proper medical care in heat illness.

Keywords: heat stroke, heat injury, hypothermia, core temperature, core temperature afterdrop

Since the beginning of the 21st century, global temperatures have risen and are predicted to rise even higher.[1] Heat reduces worker's productivity and affects body thermoregulation and homeostasis. The hypothalamic thermoregulatory center and peripheral heat receptors are activated when body temperature reaches 37ºC.[2,3] Inadequate body response causes inflammation in heat-related illness which occurs as a spectrum from heat edema, heat cramp, heat syncope, heat exhaustion to heat stroke.[4-7] Clinical outcome depends on environmental conditions, physical activities and individual biological factors. The environmental component includes climate, geographic and socio-economic factors. Individuals who are motivated and perhaps fear losing their job might do strenuous physical activities beyond their heat tolerance.[3,8] Physical exertion produces more heat while protective clothing and equipment may worsen the condition.[9] Moreover, medical or mental conditions, youth, aging, pregnancy, taking drugs or some medications, history of previous heat illness and heat acclimatization may influence the variation of heat response.[7,10,11]

Heat illness usually starts with a mild outcome such as heat edema or heat cramp. If left untreated, it may in turn become more critical such as heat stroke and heat exhaustion. Hyperthermia directly damages cells and reduces oxidative phosphorylation in cellular production of energy.[2,3] Patients may present with multi-organ failure, disseminated intravascular coagulation or even death. Heat stroke is diagnosed when a patient presents with a core temperature higher than 40ºC and significant neurological deficit such as coma, delirium or convulsion.[3,12] Exposure to high temperatures in the environment may cause classic heat stroke (CHE) while strenuous exercise with pathologic hyperthermia may cause exertional heat stroke (EHS).[13] Absence of central nervous involvement and lower core temperature make heat exhaustion differ from heat stroke.[14] To reduce body temperature,

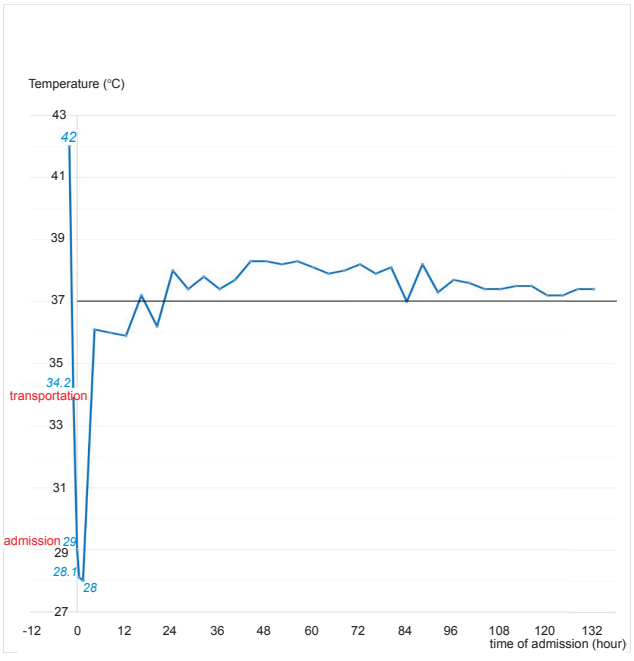
removing the patient out of the heat and rapid cooling should be managed.10 A target temperature near 38ºC for evaporative cooling and 39ºC for immersion cooling were seen in the majority of studies.15 Hypothermia from overcooling may lead to life-threatening cardiac arrhythmia.[15] When rewarming a patient with high-grade hypothermia, core temperature afterdrop can be one of its complications.[16] It is described as further drop of core body temperature after removing the patient from a cold environment.[17] This case demonstrated an EHS patient with iatrogenic hypothermia, which should be of concern in both emergency and delivery medical care. Furthermore, core temperature afterdrop was also observed during management of his hypothermia.

Case report

A 33-year-old male ranger was admitted to a 90-bed district hospital with unconsciousness and general tonic-clonic seizure. He had been fatigued and nauseated during his first few days of an advanced military training course. He felt too unwell to take food or drink during the strenuous training. At the district hospital, his medical record showed axillary temperature of 42ºC, blood pressure of 60/40 mmHg, and pulse rate at 160 beats per minute. Endotracheal intubation, volume resuscitation with 2 liters of room temperature normal saline, Foley's catheter and continuous inotrope (epinephrine) were provided. The seizure was controlled with medications (diazepam and phenytoin). Intravenous glucose was also given due to hypoglycemia of 59 mg/dL. He was then referred to a tertiary hospital. Documentation showed that his axillary temperature was 34.2ºC when leaving the district hospital. During 1-hour transportation, he was laid down on a plastic sheet, surrounded with ice cubes and water without core temperature monitoring.

At an emergency room of a tertiary hospital, his blood pressure was 110/60 mmHg. Glasgow Coma Score was E1VTM1. Ten minutes later, he was transferred to an intensive care unit (ICU) where his esophageal temperature measurement was 29ºC. Heat stroke with iatrogenic hypothermia and hypovolemic shock was diagnosed. Medical electric blanket was applied to keep temperature above 36ºC. His lowest temperature during rewarming was 28ºC. Figure 1 shows his body temperature. During the first two days, his core temperature and central blood pressure were measured. Initial electrocardiogram (EKG) showed junctional rhythm with J wave between QRS complex and ST segment (Osborn wave). Continuous monitoring of cardiac wave showed no adverse event and the wave became regular sinus rhythm after rewarming. No evidence of intracranial hemorrhage, recent infarction or herniation in brain tomography. Table 1 demonstrates his laboratory findings during treatment. On the first day, the investigations showed metabolic acidosis with respiratory alkalosis, hypokalemia, hypophosphatemia, rhabdomyolysis, pre-renal azotemia, and elevated liver transaminases. Intravenous fluid was administered and electrolyte imbalance was corrected. He gained consciousness with stable vital signs on the second day of admission. The inotrope was discontinued and extubation was performed.

Hyperthermia, thrombocytopenia with normal fibrinogen level developed on day 2. Empirical antibiotic (ceftriaxone) was given. Septic workup showed a positive hemoculture for *Enterococcus faecalis*. Neurological deficit was assessed on day 6. Only weakness of right nasolabial fold remained without swallowing difficulty. Cerebellar signs were intact. Thai Mental State Examination (MSE) was normal (score 25/30). He was sent to a community hospital in his hometown to complete the treatment for infection.



Time after admission (hr)	Temperature (°C)
0.0	29.0
0.5	28.1
1.5	28.0
4.5	36.1
8.5	36.0
12.5	35.9
16.5	37.2
20.5	36.2
24.5	38.0
28.5	37.4
32.5	37.8
36.5	37.4
40.5	37.7
44.5	38.3
48.5	38.3
52.5	38.2
56.5	38.3
60.5	38.1
64.5	37.9
68.5	38.0
72.5	38.2
76.5	37.9
80.5	38.1
84.5	37.0
88.5	38.2
92.5	37.3
96.5	37.7
100.5	37.6
104.5	37.4
108.5	37.4
112.5	37.5
116.5	37.5
120.5	37.2
124.5	37.2
128.5	37.4
132.5	37.4

Figure 1: Fluctuation of temperature in this case.

Table 1: Laboratory data of the patient

Day		1	2	3	4	5	6	7
Hb	(g/dL)	15.5	15	14	13.5		14.2	15.5
WBC	(x 10^3 /µL)	20.380	13.510	14.680	13.500		8.160	
platelets	(x 10^3 /µL)	116	79	64	81		175	116
CPK	(U/L)	7,015	6,108	5,090			478	
BUN	(mg/dL)	42.8	38.1		10.9	7.6		
Cr	(mg/dL)	1.93	1.51		1.16	1.30		
AST	(U/L)	210						
ALT	(U/L)	61						
Na	(mmol/L)	133	133	132	138	139	134	134
K	(mmol/L)	2.3	2.5	2.0	2.9	4.3	3.4	3.4
PT INR		0.9		1.37				
pH		7.433	7.408	7.331				
PCO2	(mmHg)	19.8	24.5	39.7				
PaO2	(mmHg)	601	183	139				
HCO_{3-}	(mmol/L)	17.7	18.5	20.6				
SaO_2	(%)	99.6	99.1	98.8				
FIO_2	(%)	40	40	21				
Fibrinogen	(mg%)				553.8			
Ca	(mg/dL)	8.4	7.6	7.8	8.8			
PO4	(mg/dL)	4	2.5	2.9	1.9	2.4		
Mg	(mg/dL)	2.66						
Urine amphetamine		negative						

Discussion

This young and healthy man with hyperthermia and loss of consciousness after extreme outdoor training is a typical presentation of EHS.[3] He was experiencing severe dehydration that led to hypovolemic shock and hyponatremia. Environmental and physical predisposing factors in the case were advanced military training, tropical ambient environment, thick grey outfits and inadequate rehydration. One to two liters of sweat per hour or 15 liters per day could be lost during strenuous training.[2,3] Dehydration burdens the heart, increases work of sodium-potassium adenosine pump in cellular metabolism and lowers the heat transport.[3,18] Heat distribution to capillary network of skin is served by splanchnic vasoconstriction and cutaneous vasodilatation.[2,3] Heat destroys megakaryocyte and decreases platelet levels. Splanchnic hypoperfusion and ischemia from heat injury usually present with nausea and vomiting.[18] Permeability of intestinal mucosa increases after heat damage.[2] Translocation of gastrointestinal bacteria and endotoxin to systemic circulation led to E. faecalis septicemia and systemic inflammatory response syndrome (SIR).[3] Elevation of hepatic aminotransferases demonstrates typical hepatic injury in heatstroke.[18] This impairs gluconeogenesis and induces hypoglycemia.[18] Abnormal CPK and creatinine from rhabdomyolysis and acute renal failure are mainly found in EHS rather than CHS.[18] Amphetamine did not involve the rhabdomyolysis in this case. Rhabdomyolysis usually presents with hyperkalemia and hyperphosphatemia. However, a report showed 8 out of 24 military heat injury cases with rhabdomyolysis had hypokalemia.[19] Hypophosphatemia can be associated with severe heat exhaustion.[20] Metabolic acidosis and respiratory alkalosis were found in heat injury.[19] Alkalinisation may increase activity of pH-sensitive phosphofructokinase which enhances cellular uptake of phosphorous.[21]

Temperature was not measured on arrival at the emergency department of the tertiary hospital because oral temperature is not accurately correlated to core temperature.[22] Instead, esophageal temperature probe was placed in the ICU. This patient encountered both hyperthermia and hypothermia. Most studies successfully used temperature below 39ºC for termination of ice immersion in heat injury to avoid hypothermia.[13,15,23] The patient's axillary temperature before leaving the first hospital was already under the usual target temperature. Cooling with 2ºC water cooling can reduce core body temperature by 0.35ºC per minute.[24] Using this method during long distance transportation caused hypothermia.

Hypothermia in this patient was moderately severe (mild = 32.2-35.1ºC , moderate = 28.1-32.1ºC, severe = less than 28.1ºC).[16] Osborn wave in the EKG is a diagnostic pattern when body temperature reaches below 32ºC.[25] Risk of atrial fibrillation and cardiac arrest is increased.[16,25] Rewarming is indicated in this case.25 His temperature shows core temperature afterdrop from 29 ºC to 28ºC in the first hour of rewarming. This phenomenon occurs from convection and conduction heat loss from increased blood flow form core to peripheral region during rewarming.[17] It disappears after the balancing of core and peripheral temperature.[25] A case has been reported with no complication after termination of cooling.[26]

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Neurological deficit after treatment was mild in this case. Recently, successful therapeutic hypothermia of 33ºC in non-response heat stroke patients has been reported.[27,28] Neurological benefit after therapeutic hypothermia has also been shown in cardiac arrest patients.[29] No exact mechanism may describe the weakness of right nasolabial area. A study showed that incidence of Bell's palsy in the American army was significantly higher in waterless regions.[30]

Concern with risk factors, early detection, cooling methods, management pitfalls and return to duty program are essential in primary, secondary and tertiary prevention of exertional heat injury (EHI).[24, 31] This patient should have 2 weeks of work restriction, at least 1 month of physical training and at least 1 month of gradual acclimatization.[31] However, return to the military training is not possible in this case. His work plan was uncertain. Brief advice was given for return to work plan. Evaluation tests on heat tolerance would be recommended if he continued his military service.[8,31,32]

Occupational EHS is preventable. Workplaces with heat sources should measure and assess occupational heat stress. Health and safety controls can reduce modifiable risk factors in metabolic heat production and heat exchange.[32] Air ventilation, insulation and air conditioning equipment can be used in engineering control.[32] Administrative control may include work/rest schedules and acclimatization.[32] Medical monitoring programs before and during employment should be established.[32] Health and safety training for workers should aim to teach effective self-preparedness such as proper rehydration and early recognition of symptoms.[32] Supervisors should be able to monitor weather warnings.[32] Workplace heat alert programs are recommended for emergency preparedness.[32]

Conclusion

This case report demonstrates the EHS patient with multiple organ dysfunction and overcorrection of core body temperature. Continuous application ice cubes and water while transfer for prolonged period of more than 1 hour has put the patient at risk of hypothermia. The typical Osborn wave in the EKG was also reported. To prevent the sequelae of hypothermia, we recommended that the patient' core temperature during cooling method should be monitored closely not only in the hospital but also in the transfer vehicle. It is of note that the transfer of EHS patients from one medical facility to another should become a national issue for emergency medical service, especially in the vicinity of military camps.

In conclusion, preparedness and training for emergency response, safety during transfer are necessary for both high risk workplaces and the medical settings in charge in order to prevent and control of heat stroke and its fatal consequences.

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