Management of Hypothermia with Warm Peritoneal Dialysis

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Introduction

Sir Jonathan Hutchinson first described hypothermia in 1875, defined as core body temperature below 35°C. The most common cause of hypothermia is immersion in cold water. Medical risk factors include, extremes of age (especially the elderly), homelessness, ethanol use, malnutrition, poverty, mental illness, use of neuroleptic medications, disorders of central nervous system, generalized skin disorders, hypothyroidism, hypoglycemia, diabetic ketoacidosis and chronic renal failure [1]. Predisposing factors in surgical patients include, operating room temperature, paralysed temperature control, exposure, metabolic shutdown, and cold IV fluids. Hypothermia has been reported after spinal cord injury [2,3] and it has also been used to treat spinal cord injury [4]. Here we report a case, who developed hypothermia following high cervical cord injury associated with acute oliguric renal failure following natural disaster at Bhuj (Gujarat, India). The case was diagnosed with high degree of clinical suspicion and treated successfully with passive and active rewarming.

Case Report

A 65 year old male was rescued after 4 hours of lying under the debris of a large mass of concrete after the Bhuj earthquake on 26th January 2001. He noticed that he was unable to move all limbs, and had lost sensations below the shoulder level. After first aid, he was transferred to Command Hospital, Southern Command, Pune by air. Initial examination revealed bradycardia (46/min), hypotension (BP 80/60 mmHg) and hypothermia (31°C). There was spastic quadriparesis possibly due to compressive myelopathy (cervical spine trauma) and acute oliguric renal failure. On electrocardiogram (ECG), there was sinus bradycardia, QRS widening (144 msec), QTc prolongation (531 msec) and Osborne waves (Fig. 1a). He had hyperkalemia (7meq/L) and azotemia (Blood urea 120 mg/dl, creatinine 4.3 mg/dl). Other laboratory parameters were within normal limits (Plasma glucose, Liver function test, Thyroid function test). There was no evidence of myonecrosis - normal LDH, creatinine kinase and no myoglobinuria. MRI of cervical

spine revealed fracture dislocation of C5 and C6 vertebrae with soft tissue injury (Fig. 2). He was diagnosed as a case of traumatic compressive cervical myelopathy, complicated by acute oliguric renal failure due to prolonged hypotension associated with hypothermia.

He was put on cervical traction and resuscitated with IV fluids. Hypothermia was managed with passive and active rewarming. Blankets and electric hot air blower were used for passive external rewarming. Warm peritoneal fluids (40-42°C) were used for active rewarming and management of acute renal failure. Two litres of crystalloid dialysate was instilled in the intraperitoneal cavity over 20 minutes and was drained out after 20 minutes for ten cycles. Additional

Fig. 1 : Electrocardiographic changes in hypothermia (a) showing sinus bradycardia, Osborne waves (arrow), prolonged QTc. (b) showing normalisation of changes following rewarming

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Fig.2 : MRI scan spinal cord showing fracture dislocation of cervical vertebrae 5 & 6.

twenty cycles of two hours each were administered with dialysate at 39°C. His temperature gradually returned to normal within 12 hours (Fig. 3) with normalization of ECG (Fig. 1b). His renal parameters and potassium levels improved over period of three days. He was subsequently managed in the spinal trauma care center with surgical decompression of the spine.

Discussion

Hypothermia is simply a lowering of the body's normal temperature. Significant hypothermia begins at body temperatures below 35°C (95°F), and severe hypothermia occurs at temperatures below 32°C (90°F). All body functions are slowed in severe hypothermia, including heart rate, respiratory rate, metabolism and mental activity. Patients with spinal cord transection above C8-T1 are poikilothermic. This patient had sustained injury above C8-T1 spinal segment and was also exposed to cold environment due to extreme weather conditions, which resulted in development of hypothermia. Hypothermia in these patients is due to loss of skin and core temperature efferents, loss of sympathetic tone, reduced muscle mass, inability to alter environment due to compromised mobility and shiver effectively [2,3].

Hypothermia can produce cardiovascular dysfunction leading to bradycardia, hypotension and arrhythmias [5]. ECG shows sinus bradycardia, prolongation of PR & QTc intervals and widening of QRS complex. ECG of this patient had all these changes except PR prolongation. First-degree and third degree heart blocks develop with increasing severity of hypothermia; at <30°C and 20°C respectively. Osborn JJ described J wave or elevated J-point in experimental hypothermia in 1953 [6]. It is a deflection that appears

Fig. 3 : Temperature following peritoneal dialysis

in the late part of the QRS complex as a late delta wave or a small secondary R wave (R'). This results from a marked accentuation of the spike and dome morphology, due to loss of the action potential dome of the epicardial and M cells (mediated by a transient outward current). This heterogeneous distribution of the action potential morphology gives rise to a transmural voltage gradient during ventricular activation that is responsible for the inscription of the J wave.

Management of hypothermia should be aggressive. Functional survival in adults has been reported even after 6½ hours of cardio pulmonary resuscitation. Mortality associated with hypothermia is about 12% in city dwellings but increases to around 50% if associated with significant comorbid conditions and increases by 1.8% for each degree fall in body temperature following admission [1].

Treatment should address initial first aid and transport, stabilizing cardiopulmonary status, gradual rewarming (active and passive), treating the cause of hypothermia and treating any complications. Initial care in spinal immobilization, airway, breathing and circulation need no elaboration. Transportation of the patient with utmost care to prevent deterioration of the spinal injury and body temperature is imperative. Widely held belief that sharing of sleeping bags with a normal individual with a view to use body heat to rewarm has been found to have no significant benefit. Stabilization of the cardiopulmonary status involves use of IV crystalloids and colloids to achieve adequate intravascular volume preferably with central venous pressure monitoring and use of central catheters for fluid infusion. Management of persistent hypotension may require pressor agents. Arrhythmias need to be managed emergently with special considerations in the setting of hypothermia [1].

Hypoglycaemia is a frequent accompaniment and may be missed in view of similar presentation and attributing the altered sensorium to hypothermia alone. Acute tubular necrosis has been reported in hypothermia but is infrequent and probably results from hypoxia and shock [1].

Rewarming may be passive external, active external and active central rewarming. Passive external rewarming involves shielding patient with blankets to allow the natural thermogenesis to raise the body temperature. Average increase in body temperature being 0.38°C/hour, it is adequate only if core temperature is >30°C. Active external rewarming involves use of hot blankets, electric heating pads, hot water bottles and circulating warm air. This is a controversial method but is effective even with core temperature as low as 17°C. Mortality with this method has been higher compared to other methods, possibly due to peripheral vasodilation leading to shock and increased incidence of acidosis due to abrupt return of blood to the core from relatively hypoperfused areas [1]. Active central rewarming is the fastest and most invasive method of rewarming. It involves use of warm IV fluids, gastric lavage and peritoneal dialysis by warm fluids. Peritoneal dialysis can be safely done with crystalloid dialysate at 40 to 42°C and it raises the body temperature by 4 to 6°C/hour. Direct rewarming of the liver further helps in correcting the metabolic dysfunctions. Associated complication of renal failure in our patient further justified the use of peritoneal dialysis. Use of haemodialysis machine to remove, heat and return blood back to circulation efficiently raises

temperature by 1 to 2°C/hour. Rewarming method selected must be appropriate to the patient's condition and the available infrastructure and injudicious and overenthusiastic rewarming may prove counterproductive.

In conclusion, hypothermia is a known complication of high spinal cord injuries and although infrequent must be watched for especially because of its eminently protean presentation and being an avoidable cause of mortality in these patients.

References

1. Curley FJ, Irwin RS. Disorders of temperature control. Hypothermia. In : Irwin RS, Rippler FS, editors. Intensive Care Medicine, Vol 1, 4th ed, Blackwell Science Ltd. London, 1997;828-44.
2. Hutchison J. Temperature and circulation after crushing the cervical cord. Lancet 1975;1:713-5.
3. Altus P, Hickman JW, Nord HJ. Accidental hypothermia in a quadriplegic patient. Neurology 1985;35:427-8.
4. Martinez-Arizala A, Green BA. Hypothermia in spinal cord injury. J Neurotrauma 1992;9(Suppl 2):S497-505.
5. Trevino A, Rasi B, Beller BM. The characteristic electrocardiogram of accidental hypothermia. Arch Intern Med 1971;127:470-3.
6. Osborn JJ. Experimental hypothermia : respiratory and blood pressure changes in relation to cardiac function. Am J Physiol 1953;12:389-98.

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President Bush is visiting Baghdad and Saddam Hussein is showing him how devastated the Iraqi economy is. Seeing all kinds of things, they arrive at a red button and Bush asks what it is. Hussein tells him "Press it!" so Bush presses the button and a hand comes out of the wall and hits him in the face. He gets real mad, and says "I'm going back to Washington", and returns to USA.

A couple of months later, Saddam is invited to Washington. They show him all kinds of things, and eventually they arrive at a red button. Hussein asks what it is, and Bush tells him to press it so Saddam presses it, but nothing happens. So he laughs and says "I'm going back to Baghdad!", to which Bush responds: "There is NO Baghdad!".

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